Canine Rehabilitation and Physical Therapy

SECOND EDITION

Canine Rehabilitation and Physical Therapy

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CANINE REHABILITATION AND PHYSICAL THERAPY ISBN: 978-1-4377-0309-2
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The Publisher

Library of Congress Cataloging-in-Publication Data

Canine rehabilitation & physical therapy.
Canine rehabilitation and physical therapy / [edited by] Darryl Millis, David Levine.—Second edition.
p. ; cm.
Preceded by: Canine rehabilitation & physical therapy / [edited by] Darryl L. Millis, David Levine, Robert A. Taylor. c2004.
Includes bibliographical references and index.
ISBN 978-1-4377-0309-2 (hardback : alk. paper)
1. Dogs—Diseases—Treatment. 2. Veterinary physical therapy. 3. Medical rehabilitation. I. Millis, Darryl L., editor. II. Levine, David (Veterinary physical therapist), editor. III. Title.
[DNLM: 1. Dog Diseases—therapy. 2. Physical Therapy Modalities—veterinary. SF 991]
SF991.C244 2014
636.7′089582—dc23

2013024902

Vice President and Publisher: Linda Duncan
Content Strategy Director: Penny Rudolph
Content Manager: Shelly Stringer
Publishing Services Manager: Gayle May and Hemamalini Rajendrababu
Senior Project Manager: Antony Prince
Design Direction: Karen Pauls
Cover Art: @ Grant Hindsley/AP/Corbis (cover center top row)

Printed in China

Last digit is the print number: 9 8 7 6 5 4 3 2 1

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Preface

Individuals involved in veterinary medicine and surgery have witnessed remarkable progress in their ability to diagnose and treat problems that, in the recent past, would have been untreatable. Total hip replacement in the dog, laparoscopic procedures, arthroscopic surgery and the widespread use of sophisticated imaging modalities such as MRI, CT, and real-time ultrasonic imaging are daily occurrences in veterinary medicine. With the availability of these sophisticated diagnostic techniques, the expectations for enhanced functional results have grown.

Human physical therapy is an internationally recognized discipline, and the positive efforts of post-surgical and post-injury rehabilitation have been documented and recognized in human health care. Relatively little attention has been given to veterinary patients afflicted with similar conditions. There is profound interest on the part of veterinary caregivers to learn about and provide rehabilitation and therapy following surgery, illness, or injury. Techniques used in human physical therapy are being adapted for use in small animal patients.

The purpose of this textbook is to provide a solid understanding of physical therapy techniques and interventions for dogs. Most professional programs in veterinary medicine do not provide training in physical therapy. Educational programs in physical therapy do not include evaluation and treatment of animals. These factors have resulted in the need for close collaboration between veterinarians, physical therapists, and veterinary technicians to provide optimal evaluation and treatment of animal patients. Currently, these collaborative efforts and relationships are being used to create treatment protocols for many types of cutaneous, neurologic and musculoskeletal injuries in animals. In this book, we hope to amalgamate the knowledge that physical therapists, veterinarians, and veterinary technicians possess to facilitate a faster and more complete recovery from debilitating conditions.

It is with these goals in mind that this edition was conceived and written. We have seen a tremendous interest, and growth, in veterinary rehabilitation since the first edition of this book was published in 2004. Canine rehabilitation has grown from a small area of interest to a recognized discipline during this time, and all animal patients, their owners, and veterinary caregivers are realizing the benefits of this progression. This book has expanded greatly from the first edition as knowledge within this field has rapidly grown. Many chapters have been added to disseminate this new knowledge, and all of the chapters have been updated to reflect advances in the respective topics. We hope this book is useful as both an in-depth study of the field, and as a quick reference on topics such as protocols for specific conditions that are seen clinically.



Acknowledgments

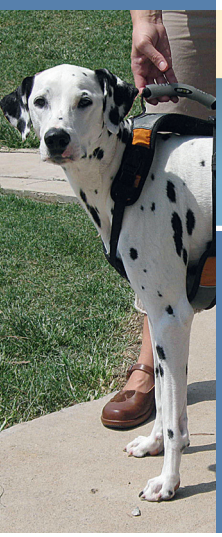
I am truly amazed at the continued interest and progress made in veterinary rehabilitation during the ten years since the first edition of this textbook was published. When writing a textbook in an area that incorporates two separate professions, there are many challenges to overcome and people to thank. I would like to especially thank Dave for his humor, expertise, and continual encouragement to keep things on track. Thanks are also in order to the many physical therapists, veterinarians, veterinary technicians, owners, and patients who have helped move the field forward and provided kind words and encouraged our efforts. The staff at Elsevier, especially Penny and Shelly, have done a tremendous job in keeping things on track and giving us nudges, pushes and shoves when needed. I would be remiss if I did not thank my colleagues at the University of Tennessee for providing help and support in the clinics and laboratory, especially Drs. Joe Weigel, Marti Drum, Dave Hicks, and Jason Headrick. I would especially like to thank my family for continuing to support and encourage me; my wife Linda who has always encouraged and supported my efforts, and put up with the hectic work and travel schedule; Chris and Nick, who are not only my sons, but my best friends, who always keep life interesting; and my mother Adeline and late father, George, who made my dream of becoming a veterinarian come true by encouraging and supporting me from the very beginning. Finally, I would like to thank my patients, past and present, for their wisdom in teaching me about rehabilitation.

All things bright and beautiful,
All creatures great and small,
All things wise and wonderful:
The Lord God made them all.
Cecil Frances Alexander, 1848

Darryl Millis

Many thanks are owed when a book five plus years in the making is finally completed. So many people have generously given their time and thoughts to help me, and all cannot be listed; however, a few special people need to be thanked by name for their support and encouragement. Thanks first to my co-editor and friend, Darryl, who is not only an exceptional surgeon but also an exceptional person. To the many contributors who have helped to keep this work spirited and always enjoyable. My colleagues, students, and patients, I continue to learn every day from all of you. Old friends, like Kevin, Ivan, Denis, and Randy who have kept me laughing for the last 25 plus years, let's keep the good times coming! To many family members across this country who remind me of what family means, and to the memory of my father, Jacob Levine (1913-2011). To my mother Marie, to whom I owe much more than I can ever repay. To my children Lauren Allyn, Sarah Marie, Hadley Christian, and Ava Katherine Ann who brighten every moment of every day. To my wife Allison—who somehow understands me completely and supports me wholly. And to the One who was with me before I was born.

David Levine



SECTION I

Introduction to Physical Rehabilitation

1 History of Canine Physical Rehabilitation

Lin McGonagle, Linda Blythe, and David Levine

History of Canine Physical Rehabilitation

The idea of applying rehabilitation principles and techniques to animals, although not new, has grown appreciably since the mid 1990s. More than 110 facilities providing physical therapy and rehabilitation are now operating in the United States and this number is growing rapidly as veterinarians and physical therapists realize the need and market for these services. Although many of the treatment protocols for humans were developed and continue to be developed using animal models,[1-7] a growing number of research studies are being conducted in universities and private practices that look specifically at the benefits of different methods of rehabilitation in animals, especially dogs. Higher owner expectations combined with increased sophistication and technical abilities of veterinary clinicians have resulted in greater interest in physical therapy and rehabilitation.

Interest in the practice of canine rehabilitation in the United States first gained momentum in the late 1980s and throughout the 1990s as a result of the influence of classic texts; sporadic journal articles; national presentations at the American Physical Therapy Association (APTA), American Veterinary Medical Association (AVMA), and the American College of Veterinary Surgeons (ACVS) meetings; and the formation of the Animal Physical Therapist Special Interest Group within the APTA. The International Racing Greyhound Symposium in conjunction with the Eastern States Veterinary Conference (currently the North American Veterinary Conference) in Orlando, Florida, was first started in 1986 and was expanded and renamed the International Canine Sports Medicine Symposium to include all sporting dogs. Rehabilitation was a frequent topic at these annual meetings and numerous articles have been printed in the proceedings of this symposium as well as others during the past 25 years.[8-19] Many veterinarians and physical therapists have lectured and presented continuing education and research findings at regional, national, and international human and veterinary conferences.

Many veterinarians have felt a need to improve postoperative patient care, because traditionally preoperative management, diagnostic procedures, and surgical treatment have been emphasized. The results seen with humans undergoing intensive postoperative rehabilitation have caused many veterinarians to rethink patient management strategies, so that postoperative rehabilitation, once overlooked, is now becoming more common in veterinary practice.

The APTA position statement and the AVMA "Guidelines for Alternative and Complementary Veterinary Medicine" have provided some initial guidelines for the field of animal physical therapy. Each professional organization has recognized the other and has published guidelines for collaborative working relationships. The APTA House of Delegates adopted a position statement in June 1993 regarding animal physical therapy, which states that the APTA "endorses the position that physical therapists may establish collaborative, collegial relationships with veterinarians for the purposes of providing physical therapy services or consultation."[20] The "Guidelines for Alternative and Complementary Veterinary Medicine" were adopted in July 1996 by the AVMA House of Delegates.[21] The document, which defined *veterinary physical therapy* as "the use of noninvasive techniques, excluding veterinary chiropractic, for the rehabilitation of injuries in non-human animals," established the following guidelines:

> Veterinary physical therapy should be performed by a licensed veterinarian or, where in accordance with state practice acts, by (1) a licensed, certified, or registered veterinary or animal health technician educated in veterinary physical therapy or (2) a licensed physical therapist educated in non-human animal anatomy and physiology.

The authors would like to acknowledge Robert Taylor for his work on the previous edition.

> Veterinary physical therapy performed by a non-veterinarian should be performed under the supervision of, or referral by, a licensed veterinarian who is providing concurrent care. Veterinary physical therapy performed by non-veterinarians should be limited to the use of stretching; massage; stimulation by use of low-level lasers, electrical sources, magnetic fields, and ultrasound; rehabilitative exercises; hydrotherapy; and applications of heat and cold.

New guidelines were adopted by the AVMA House of Delegates in 2001.[22] They evaluated several medical approaches described by the terms *complementary, alternative,* and *integrative* and collectively described them as *complementary and alternative veterinary medicine* (CAVM). Examples of CAVM include aromatherapy; Bach flower remedy therapy; energy therapy; low-energy photon therapy; magnetic field therapy; orthomolecular therapy; veterinary acupuncture, acutherapy, and acupressure; veterinary homeopathy; veterinary manual or manipulative therapy (similar to osteopathy, chiropractic, or physical medicine and therapy); veterinary nutraceutical therapy; and veterinary phytotherapy.

The basic concept behind these new guidelines was to emphasize that CAVM should be held to the same standards as traditional veterinary medicine, including validation of safety and efficacy by the scientific method. In addition, the guidelines state, "The AVMA believes veterinarians should ensure that they have the requisite skills and knowledge for any treatment modality they may consider using." Finally, another pertinent point is, "The quality of studies and reports pertaining to CAVM varies; therefore, it is incumbent on a veterinarian to critically evaluate the literature and other sources of information. Veterinarians and organizations providing or promoting CAVM are encouraged to join with the AVMA in advocating sound research necessary to establish proof of safety and efficacy." It is encouraging that a number of studies have already indicated its benefit in treating a wide number of conditions.[23-34] In addition, other studies have evaluated therapeutic modalities and the responses of tissues to rehabilitation following injury and during repair.[35-97]

Journals and books have provided information on animal physical rehabilitation for more than 30 years. However, an article titled "Postsurgical Physical Therapy: The Missing Link," by Taylor,[23] was one of the first to capture the interest of the veterinary community. Throughout the remainder of the 1990s and continuing to date, the number of publications increased. Topics included cranial cruciate ligament rupture and rehabilitation,[23-34] postoperative management of spinal surgery or spinal cord diseases in the dog,[35-48] orthopedic conditions and osteoarthritis,[49-61] and management considerations for trauma patients.[62,63] Collaboration of veterinarians and physical therapists has increased, resulting in publications regarding physical rehabilitation for the critically ill patient[64-66] and pain relief.[67-70] It is clear that the dissemination of information has strengthened the ties between veterinarians and physical therapists and increased support for the efficacy of rehabilitation approaches in animal care.

Initially, several books helped shape the field of animal physical rehabilitation. *Physical Therapy for Animals: Selected Techniques,* by Downer,[71] influenced professionals as early as 1978. *Canine Sports Medicine and Surgery*[72] added to the growing field of sports medicine and discussed the role of physical rehabilitation in treating injuries of dogs. Originally published in 1994 as *Care of the Racing Greyhound*[74] and revised in 2007,[73] *Care of the Racing and Retired Greyhound* outlines rehabilitation guidelines for musculoskeletal injuries sustained in racing and training that are applicable to any athletic dog.[74] The revised version was expanded to include retired greyhounds. Three recent texts written through the cooperation of veterinarians and physical therapists include *Canine Rehabilitation and Physical Therapy* (2004) by Millis, Levine, and Taylor[75]; *Essential Facts of Physiotherapy* (2004) by Bockstahler, Levine, and Millis[76]; and *Animal Physiotherapy—Assessment, Treatment and Rehabilitation of Animals* (2007) by McGowan, Goff, and Stubbs.[77] All of these recent texts provide details of examination procedures, intervention, and applications for specific conditions. Multiple articles describe the use of physical therapeutic modalities and orthotics.[78-97]

Physical rehabilitation is gaining greater acceptance in veterinary medicine and there are more options for training. Currently, rehabilitation rotations, electives, and instruction are available at a number of veterinary colleges, and rehabilitation lectures and even courses exist as a part of the professional curriculum. A university-based certificate program at the University of Tennessee has over 850 graduates worldwide as of 2012 (www.canineequinerehab.com). Graduates receive the Certified Canine Rehabilitation Practitioner designation. Other continuing education programs are available at conferences and meetings.

In August 1999 the First International Symposium for Physical Therapy and Rehabilitation in Veterinary Medicine was sponsored by and held at Oregon State University. More than 300 participants from 21 countries met during 4 days to present clinical and research findings and to share ideas. This meeting focused entirely on animal physical rehabilitation and brought the professions of veterinary medicine and physical therapy together to exchange information and share ideas. Subsequently, there have been additional symposia in the United States in Knoxville, Tennessee, in 2002; in Raleigh, North Carolina, in 2004; and in Minneapolis, Minnesota, in 2008. The fourth symposium was held in Arnhem, the Netherlands, in 2006 to expand and integrate rehabilitation knowledge, methods, and practices of other countries. In 2010, the sixth symposium was held at Auburn University in Alabama and the Seventh Symposium was in Vienna, Austria. Plans for a meeting in the United States in the summer of 2014 are ongoing.

Worldwide Animal Rehabilitation and Physical Therapy Associations

Veterinarians and physical therapists in many countries have been sharing information and working together for three decades. Physiotherapists have professional organizations for animal physical rehabilitation in at least 11 countries: Australia, Canada, Finland, Germany, the Netherlands, New Zealand, South Africa, Sweden, the United Kingdom, and the United States. In many of these countries, the groups are formally recognized by their respective national physical therapy associations. Box 1-1 provides a brief history of these organizations and contact information.

With the robust interest in the area of canine physical rehabilitation at local, national, and international meetings, there are now several formal veterinary associations. The International Association of Veterinary Rehabilitation and Physical Therapy (www.iavrpt.org) became an official association in July 2008. This diverse group of veterinarians, physical therapists, and other interested professionals grew out of the original symposium attendees and assists in guiding the organization of the biannual symposium. Their mission is to provide a forum for the presentation of

Box 1-1 Animal Physical Rehabilitation Organizations (Official Names)

International Associations

International Association of Veterinary Rehabilitation and Physical Therapy

Incorporated in 2008 for numerous professions working in animal rehabilitation

Website: www.iavrpt.org

Veterinary European Physical Therapy and Rehabilitation Association

First meeting held in 2010 for professions working in animal rehabilitation in Europe.

Website: www.vepra.eu

International Association of Physical Therapists in Animal Practice

Recognized in 2011 by the World Confederation for Physical Therapy

Website: www.WCPT.org

Australia

Animal Physiotherapy Group

Recognized by the Australian Physiotherapy Association.

Website: www.physiotherapy.asn.au

Canada

Animal Rehab Division, formerly known as Canadian Horse and Animal Physiotherapy Association (CHAP)

First organized in 1994. Recognized in 2004 by the Canadian Physiotherapy Association (www.physiotherapy.ca/)

Website: www.physiotherapy.ca/Division/Animal-Rehabilitation

Finland

Finnish Association of Animal Physiotherapists

Founded in 1997. Recognized as a subgroup of the Finnish Association of Physiotherapists

Website: www.fysioterapia.net

The Netherlands

Nederlandsee Verening voor Fysiotherapie bij Dieren (NVFD; Dutch Animal Physical Therapy Association)

Founded in 1989. Recognized by the Dutch Ministry of Agriculture since 1992.

Address: Hindelaan 56, 1216 CW Hilversum, Netherlands

South Africa

Animal Physiotherapy Group of South Africa, formerly known as the South African Association of Physiotherapists in Animal Therapy (SAAPAT)

First organized in 1988. Gained official recognition in 1998 by the South African Society of Physiotherapy.

Website: www.animalphysiogroup.co.za

Sweden

The Association of Registered Physiotherapists of Veterinary Medicine

Founded in 1995. Became a section member of the Swedish Association of Registered Physiotherapists in 1996.

Website: lsvet.se

United Kingdom

Association of Chartered Physiotherapists in Animal Therapy (ACPAT)

Recognized in 1988 by the Chartered Society of Physiotherapists

Website: www.acpat.org

Ireland

Chartered Physiotherapists in Veterinary Practice

Recognized by the Irish Society of Chartered Physiotherapists

Website: iscp.ie

Switzerland

Schweizerischer verband fur tierphysiotherapie

Established in 2007

Website: svtpt.ch, tierphysiotherapie.com

United States

Animal Physical Therapist Special Interest Group

Recognized in 1998 by the American Physical Therapy Association

Website: www.orthopt.org/sig_apt.php

American Association of Rehabilitation Veterinarians

Founded in 2008 for veterinarians.

Website: www.rehabvets.org

American Association of Rehabilitation Veterinary Technicians

Created for veterinary technicians who provide assistance in physical rehabilitation.

http://rehabvets.org/aarvt.lasso

American College of Veterinary Sports Medicine and Rehabilitation.

Provisional approval in 2010 by the American Veterinary Medical Association

Website: VSMR.org

clinical and research information and discussion of topics related to animal rehabilitation, to further scientific investigation, and to promote the continued development of this specialty area to provide improved quality of care based on sound evidence. Another new association, the Veterinary European Physical Therapy and Rehabilitation Association, held its first meeting in 2010 in Zagreb, Croatia. Another organization for veterinarians interested in rehabilitation in the United States is the American Association of Rehabilitation Veterinarians (www.rehabvets.org). This group has a goal of making veterinarians in the United States more aware of the benefits of rehabilitation and addressing practice concerns. A similar organization exists for veterinary technicians, the American Association of Rehabilitation Veterinary Technicians. Another formal association became established in April 2011 within the World Confederation of Physical Therapy (WCPT.org). The International Association of Physical Therapists in Animal Practice was created to foster collaboration among physical therapists worldwide and to share information relating to research, education, and practice.

Future Trends

Physical rehabilitation in small animal practice has become increasingly common and will continue to become more accepted as the scientific literature continues to evolve. In many orthopedic and neurologic conditions, physical rehabilitation is becoming commonplace as a means to enhance recovery, as it is in human medicine and surgery. Wellness and preventive medicine, such as physical rehabilitation for weight reduction and for maintenance of muscle strength and cardiorespiratory fitness, is also emerging as a trend among pet owners. In 2008, a national committee of veterinarians in the United States proposed that veterinary rehabilitation become a board-certified specialty under the American Veterinary Medical Association and the American Board of Veterinary Specialties (ABVS). This newly formed American College of Veterinary Sports Medicine and Rehabilitation has been given provisional approval by the AVMA to establish and maintain credentialing and certification standards for veterinary practitioners who excel in sports medicine and rehabilitation. The organization formed with a group of charter diplomats and administered its first certifying examination in 2012. With both academic and nonacademic pathways to certification, a veterinarian can become board certified in either canine or equine specialties under this college.

It is certain the field of animal rehabilitation will expand in future years both in the United States and worldwide. Our hope is that veterinary and physical therapy professionals will continue to collaborate to define practice models and establish effective clinical protocols, as well as provide education and training for the benefit of our animal clients and owners.

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2 Regulatory and Practice Issues for the Veterinary and Physical Therapy Professions

David Levine and Darryl Millis

Definition of Physical Therapy

The American Physical Therapy Association (APTA) has developed a model definition of *physical therapy.* In this model definition, physical therapy includes examining and evaluating patients with impairments, functional limitations, disability, and other health-related conditions to determine a diagnosis, prognosis, and intervention. Some examples of areas that may be examined include aerobic capacity, arousal, cognition, assistive and supportive devices, barriers, ergonomics, gait, balance, pain, posture, prosthetic requirements, and range of motion.[1]

Physical therapists also alleviate impairments and functional limitations by designing, implementing, and modifying therapeutic interventions. Some examples of these activities include therapeutic exercise, functional training, manual therapy techniques, electrotherapeutic modalities, and patient-related instruction. Physical therapists are also involved with helping to prevent injury, impairments, functional limitations, and disability, and with the promotion and maintenance of fitness, health, and quality of life in all age populations. Physical therapists accomplish these tasks in a variety of situations, including patient consultation, education, and research.

Definition of Veterinary Medicine

The practice of veterinary medicine is described in the directory of the American Veterinary Medical Association (AVMA) in a section titled The Model Practice Act (Act).[2] The Act has been periodically updated and revised, and in the current version (2012, Section 2.16), the practice of veterinary medicine means:

a. To diagnose, prognose, treat, correct, change, alleviate, or prevent animal disease, illness, pain, deformity, defect, injury, or other physical, dental, or mental conditions by any method or mode; including the:
   i. performance of any medical or surgical procedure, or
   ii. prescription, dispensing, administration, or application of any drug, medicine, biologic, apparatus, anesthetic, or other therapeutic or diagnostic substance, or
   iii. use of complementary, alternative, and integrative therapies, or
   iv. use of any procedure for reproductive management, including but not limited to the diagnosis or treatment of pregnancy, fertility, sterility, or infertility, or
   v. determination of the health, fitness, or soundness of an animal, or
   vi. rendering of advice or recommendation by any means including telephonic and other electronic communications with regard to any of the above.

b. To represent, directly or indirectly, publicly or privately, an ability and willingness to do an act described in subsection 16(a).

c. To use any title, words, abbreviation, or letters in a manner or under circumstances that induce the belief that the person using them is qualified to do any act described in subsection 16(a).

This updated Model Veterinary Practice Act includes the use of complementary, alternative, and integrative therapies. Until 2001 these had been listed under Guidelines for Alternative and Complementary Veterinary Medicine, which is now referred to as "Complementary, Alternative, and Integrative Therapies." These guidelines describe the potential use and application of these therapies for veterinary patients.[3] The Model Veterinary Practice Act has included these forms of treatment to help protect the public, by placing the definitions under the Practice of Veterinary Medicine. Approximately 20 states have followed the AVMA Model Practice Act and included complementary and alternative medicine in their state practice act definition of veterinary medicine, while another 20 have enacted exemptions for certain therapies, generally requiring veterinary supervision or referral.

Complementary, Alternative, and Integrative Therapies

The AVMA Guidelines for Complementary and Alternative Veterinary Medicine address topics such as veterinary acupuncture and acutherapy, and acupressure; low-energy photon therapy; magnetic field therapy; veterinary chiropractic; veterinary physical medicine and rehabilitation; veterinary homeopathy; and nutraceutical medicine.[3] The guidelines state:

The authors would like to acknowledge Nancy Murphy for her work on this chapter in the previous edition.

The AVMA believes that all veterinary medicine, including CAVM, should be held to the same standards. Claims for safety and effectiveness ultimately should be proven by the scientific method. Circumstances commonly require that veterinarians extrapolate information when formulating a course of therapy. Veterinarians should exercise caution in such circumstances. Practices and philosophies that are ineffective or unsafe should be discarded.

Furthermore,

"The AVMA believes veterinarians should ensure that they have the requisite skills and knowledge for any treatment modality they may consider using."

Recommendations for patient care state that:

> "The foremost objective in veterinary medicine is patient welfare. Ideally, sound veterinary medicine is effective, safe, proven, and holistic in that it considers all aspects of the animal patient in the context of its environment.
>
> Diagnosis should be based on sound, accepted principles of veterinary medicine. Proven treatment methods should be discussed with the owner or authorized agent when presenting the treatment options available. Owner consent should be obtained prior to initiating any treatment, including CAVM. Clients usually choose a medical course of action on the advice of their veterinarian. Recommendations for effective and safe care should be based on available scientific knowledge and the medical judgment of the veterinarian."

The veterinarian also undertakes responsibilities, including:

> "These guidelines support the requisite interaction described in the definition of the veterinarian-client-patient relationship.[1] Accordingly, a veterinarian should examine an animal and establish a preliminary diagnosis before any treatment is initiated.
>
> The quality of studies and reports pertaining to CAVM varies; therefore, it is incumbent on a veterinarian to critically evaluate the literature and other sources of information. Veterinarians and organizations providing or promoting CAVM are encouraged to join with the AVMA in advocating sound research necessary to establish proof of safety and efficacy.
>
> Medical records should meet statutory requirements. Information should be clear and complete. Records should contain documentation of client communications and owner consent.
>
> In general, veterinarians should not use treatments that conflict with state or federal regulations. Veterinarians should be aware that animal nutritional supplements and botanicals typically are not subject to premarketing evaluation by the FDA for purity, safety, or efficacy and may contain active pharmacologic agents or unknown substances. Manufacturers of veterinary devices may not be required to obtain premarketing approval by the FDA for assurance of safety or efficacy. Data establishing the efficacy and safety of such products and devices should ultimately be demonstrated. To assure the safety of the food supply, veterinarians should be judicious in the use of products or devices for the treatment of food-producing animals.
>
> If a human health hazard is anticipated in the course of a disease or as a result of therapy, it should be made known to the client."

Veterinary Physical Rehabilitation

Veterinary physical rehabilitation is the use of noninvasive techniques, excluding veterinary chiropractic, for the rehabilitation of injuries in nonhuman animals. Veterinary physical rehabilitation performed by nonveterinarians should be limited to the use of stretching; massage therapy; stimulation by use of low-level lasers, electrical sources, magnetic fields, and ultrasound; rehabilitative exercises; hydrotherapy; and applications of heat and cold. Veterinary physical rehabilitation should be performed by a licensed veterinarian or, where in accordance with state practice acts, by a licensed, certified, or registered veterinary or animal health technician educated in veterinary physical rehabilitation or a licensed physical therapist educated in nonhuman animal anatomy and physiology. Veterinary physical rehabilitation performed by a nonveterinarian should be performed under the supervision of, or referral by, a licensed veterinarian who is providing concurrent care.

Massage Therapy

Massage therapy is a technique in which the therapist uses only his or her hands and body to massage soft tissues. Massage therapy on nonhuman animals should be performed by a licensed veterinarian with education in massage therapy or, where in accordance with state veterinary practice acts, by a graduate of an accredited massage school who has been educated in nonhuman animal massage therapy. When performed by a nonveterinarian, massage therapy should be performed under the supervision of, or referral by, a licensed veterinarian who is providing concurrent care.[3]

The development of the AVMA Guidelines for Complementary and Alternative Veterinary Medicine is a recognition by the veterinary profession that other health care professionals have information and knowledge that may benefit veterinary patients. Veterinarians should not hesitate to seek the help, advice, and expertise of others to improve the care and management of their patients. When this expertise, assistance, and advice is sought, however, the responsibility for the care, diagnosis, treatment, and management of the patient remains with the attending veterinarian, and a veterinarian–client–patient relationship (VCPR) will be established.

The Veterinarian–Client–Patient Relationship

The concept of the VCPR was introduced by the AVMA to clearly describe the relationship of the veterinarian to clients (the owners) and patients served by the profession. This VCPR is the basis for professional interactions and has become part of a variety of official AVMA documents, including the Principles of Veterinary Medical Ethics, the Model Practice Act, and the Guidelines for Veterinary Prescription Drugs. The Guidelines for Complementary and Alternative Veterinary Medicine also refer to the VCPR, and, because these modalities are considered to be part of the practice of veterinary medicine, they should be offered only in the context of a valid VCPR.

As stated in the Principles of Veterinary Medical Ethics,[4] the VCPR is the basis for interaction among veterinarians, owners, and patients. A VCPR exists when all of the following conditions have been met:

1. The veterinarian has assumed responsibility for making clinical judgments regarding the health of the animal or animals and the need for medical treatment, and the client has agreed to follow the veterinarian's instructions.
2. The veterinarian has sufficient knowledge of the animals to initiate at least a general or preliminary diagnosis of the medical condition of the animals. This means that the veterinarian has recently seen and is personally acquainted with the keeping and care of the animals by virtue of an examination, or by medically appropriate and timely visits to the premises where the animals are kept.
3. The veterinarian is readily available, or has arranged for emergency coverage, for follow-up evaluation in the event of adverse reactions or the failure of the treatment regimen.

The Principles additionally state that when a VCPR exists, veterinarians must maintain medical records, which should contain information on the diagnosis, care, and treatment of patients.[4]

The Principles also discuss the termination of the VCPR, which applies when professional services are no longer assumed by the veterinarian or no longer needed by the client. Veterinarians may terminate a VCPR under certain conditions, and they have an ethical obligation to use courtesy and tact in discharging this responsibility. Guidelines for terminating the VCPR are as follows:

1. If there is no ongoing medical condition, veterinarians may terminate a VCPR by notifying the client that they no longer wish to serve that patient and client.
2. If there is an ongoing medical or surgical condition, the patient should be referred to another veterinarian for diagnosis, care, and treatment. The former attending veterinarian should continue to provide care, as needed, during the transition.

The Principles state that clients may terminate the VCPR at any time.[4]

Guidelines for Referrals

The expansion and growth of knowledge, the emergence of specialization, and the use of the services and expertise of other health care professionals have necessitated the increased use of referrals in veterinary medicine. Referrals are to be encouraged among veterinarians, and the Guidelines for Referrals[5] approved by the AVMA in 1990 and revised in 2009 should be followed when referrals are used. These referral guidelines are summarized and paraphrased as follows:

Definitions

The referring veterinarian is the veterinarian who was in charge of the patient at the time of the referral. The receiving veterinarian or the referral veterinarian is the veterinarian to whom a patient is sent either by referral or for consultation. A *consultation* is a deliberation between two or more veterinarians concerning the diagnosis of a disease and the proper management of the case. A *referral* is the transfer of responsibility of diagnosis, care, and treatment from the referring veterinarian to the receiving veterinarian, and a new VCPR is established with the receiving veterinarian.

In these descriptions and definitions, the assumption is made that referrals and consultations may occur only between veterinarians. The Guidelines for Referrals do not address a situation in which the individual who is referring or receiving the patient is not a veterinarian. If these Guidelines for Referrals apply only to veterinarians, then other health care professionals may not actually accept or recommend referrals without the involvement of a veterinarian and the establishment of a valid VCPR. However, when a treatment modality such as physical therapy is required, a referral directly to a therapist may be appropriate. In that instance, specific written instructions and orders must accompany the referral. Because of the necessity of a valid VCPR, other health care professionals who might be involved in the diagnosis, care, and treatment of veterinary patients must work closely with veterinarians so a valid VCPR will be established and maintained.

Method of Referral

When a referral is being considered, communication among veterinarians and other health care professionals is essential. Communications may occur by letter, telephone, direct contact, or other means, and the most appropriate method of communication should be determined by the individuals involved. The referring veterinarian should provide the receiving veterinarian with all the appropriate

information pertinent to the case before or at the time of the first contact with the patient or the owner. When the referred patient has been examined and a diagnosis has been established, the referring veterinarian should be promptly informed of those findings. Information provided should include diagnosis, proposed care, treatment plans, and other recommendations. If the patient undergoes a prolonged treatment or hospitalization, then immediately on discharge of the patient, the referring veterinarian should receive a detailed and complete report, preferably written, and should be advised as to continuing care of the patient or termination of the case. Each veterinarian involved in the case is entitled to collect fees for service, care, and treatments for professional services; fee splitting is not allowed.[4]

All licensed veterinarians may receive referrals, and referring veterinarians may refer to whomever they believe appropriate for the situation. Referrals should occur in a timely manner, and veterinarians who solicit or encourage referrals should abide by the AVMA Principles of Veterinary Medical Ethics. The receiving veterinarian should provide only services or treatments relative to the referred condition and should consult the referring veterinarian if other services or treatments are needed.

When referrals are made to other health care providers who may not be licensed veterinarians, the same standards and guidelines must apply. Other health care professionals (nonveterinarians) who are involved with the care and treatment of veterinary patients cannot assume a valid VCPR, and therefore the ultimate responsibility for diagnosis, care, and treatment of the patient remains with the referring veterinarian, as a VCPR must be in effect at all times.

State Practice Acts: Veterinary Medicine and Physical Rehabilitation

Although interest in and support for the unique practice of veterinary physical rehabilitation is growing nationally, each state individually regulates the standards for both veterinary medicine and physical therapy through the respective practice acts for each profession. Both the APTA and the AVMA have adopted positions and policies that support animal physical rehabilitation.[3,6]

These organizations are not the regulatory bodies for the disciplines of physical therapy or veterinary medicine. The practice of veterinary medicine and that of physical therapy are regulated by respective practice acts for each state, and it is the responsibility of individuals who practice veterinary physical therapy to understand the legal issues related to both the veterinary and physical therapy practice acts of their respective states before pursuing this specialized discipline. Individuals practicing veterinary physical rehabilitation who fail to comply with the rules and regulations of the respective state practice acts could be practicing both veterinary medicine and physical therapy without a license and might be subject to investigation, warning, disciplinary action, or criminal prosecution. A useful resource is this list entitled "State-by-State Assessment of Physical Therapy and Veterinary Practice Acts."

http://www.orthopt.org

This resource also lists people in the state to contact.

Veterinary Medicine

The veterinary practice acts are often vague when referring to the practice of veterinary physical rehabilitation. One exception is the Idaho Veterinary Practice Act, which states that "The requirement to have a license to practice veterinary medicine does not prohibit an allied health professional actively licensed and in good standing in any state from participating in a medical procedure involving an animal, provided that such participation is in his licensed field of medicine and under the indirect supervision of an actively licensed veterinarian."[7] "An allied health professional" means a person holding a current active license, in good standing, in any state to practice one of the healing arts including, but not limited to, medicine, dentistry, osteopathy, chiropractic, acupuncture and podiatry." Indirect supervision means "the supervising veterinarian is not on the premises but is available for immediate contact by telephone, radio or other means, has given either written or oral instructions for treatment of the animal patient, the animal has been examined by the supervising veterinarian as acceptable veterinary medical practice requires, and the animal, if previously anesthetized, has recovered to the point of being conscious and sternal."[7] The supervising veterinarian shall "Have examined the animal patient prior to the delegation of any animal health care task to a certified veterinary technician, temporary certification holder, or assistant. The examination of the animal patient shall be conducted at such times as acceptable veterinary medical practice dictates, consistent with the particular delegated animal health care task."

In the state of Pennsylvania, both the State Board of Veterinary Medicine and the State Board of Physical Therapy are regulated by the same administration.[8] This arrangement allows for a clear understanding of the regulatory issues affecting both veterinarians and physical therapists when members of either profession are interested in veterinary physical rehabilitation. However, individuals practicing their respective professions in that state must be licensed by the regulatory board of their respective profession. The State Board of Veterinary Medicine states that "Veterinarians may seek, through consultation, the assistance of other licensed professionals, including chiropractors, dentists, dental hygienists and physical therapists, when it appears that chiropractic, dental, dental hygiene or physical therapy procedures will enhance the quality of veterinary care. Chiropractic, dental, dental hygiene and

physical therapy procedures shall only be performed upon animals by chiropractors, dentists, dental hygienists and physical therapists in conjunction with the practice of veterinary medicine and under the direct supervision of a veterinarian, subject to a limitation provided by law or regulation."

Physical Therapy

The physical therapy practice acts of many states include variable wording for authorizing and sanctioning the application of physical therapy techniques and procedures to animals and to the direct access of owners to physical therapy services. Currently, many states use the words *human* or *human being,* some states use the word *person,* and others use the word *individual* in their physical therapy practice acts (Box 2-1). This wording may be a problem for physical therapists who wish to practice veterinary physical therapy in those states, because the state practice acts may actually prohibit physical therapists from providing care to animals. Obtaining continuing education units or credits for courses in animal rehabilitation may also be problematic if the state physical therapy board does not accept this area of practice as legal. Direct access to physical therapy by human patients is an intriguing issue, in that most states permit direct access to either "physical therapy evaluation" or "physical therapy evaluation and treatment" without a physician referral. In the states that allow direct access of human patients to physical therapy, a physical therapist does not have to act with a physician to initiate physical therapy modalities; however, such direct access is not allowed for veterinary patients because of the definitions of veterinary practice and the necessity for a valid VCPR.[2] The physical therapy state practice acts are readily available online.[9]

Because of the growing interest in animal rehabilitation and physical therapy, the Animal Physical Therapist Special Interest Group (APTSIG) within the Orthopedic Section of the APTA has collected information regarding the state practice acts and animal physical therapy. This information is intended to identify which state practice acts discuss animal rehabilitation and physical therapy, and which state practice acts include the words *humans, people,* or *persons,* which might limit the practice of animal rehabilitation and physical therapy in those states. The APTSIG is also collecting information regarding the veterinary practice acts in all states.

In many states the terms *physical therapy* and *physical therapist* are "protected terms," and therefore only licensed practitioners of physical therapy in those jurisdictions may lawfully represent themselves as physical therapists or state that they provide physical therapy services. Veterinarians or veterinary technicians practicing physical therapy on animals in these jurisdictions should state that they provide physical rehabilitation, or rehabilitation, to avoid potential conflicts, unless they employ a physical therapist or physical therapist assistant (PTA) in their practice.

The physical therapy state practice act of New Mexico is a physical therapy practice act that specifically addresses veterinary physical therapy, and in that act a doctor of veterinary medicine is described as a "primary health care provider."[10] This statement occurs in the section of the practice act pertaining to direct care (access) requirements for physical therapists. In this practice act a "primary health care provider" is defined as

> a health care professional who is licensed in the U.S. and provides the first level of basic or general health care for individual's health needs including diagnostic and treatment services and includes but is not limited to a physician (M.D., D.O., D.P.M.), doctor of veterinary medicine (D.V.M.), doctor of chiropractic (D.C.), doctor of dental surgery (D.D.S.), doctor of oriental medicine (D.O.M.), certified nurse practitioner (C.N.P.), certified nurse-midwife (C.N.M.), licensed midwife (L.M.), and physician assistant (P.A.) practicing under the auspices of one of the providers listed herein.

Box 2-1 Physical Therapy State Practice Acts

Human or Human Beings
Alabama
Alaska
Arkansas
Colorado
Florida
Georgia
Hawaii
Idaho
Iowa
Maine
Missouri
Montana
New York
Oregon
Rhode Island
South Carolina
Utah
Washington, DC
Wyoming

Persons
Arizona
California
Connecticut
Delaware
Illinois
Minnesota
Nebraska
Nevada
North Carolina
Ohio
Pennsylvania
Texas*
Vermont
Washington
West Virginia

Patients
Louisiana
Massachusetts
New Mexico
South Dakota

Individuals
Kansas
Kentucky
Maryland
Michigan†
Oklahoma
Tennessee
Virginia

No Specification
Indiana
Mississippi
New Hampshire
New Jersey
North Dakota
Wisconsin

*Part C states, "treatment, consultative, educational, or advisory services to reduce the incidence or severity of disability or pain to enable, train, or retrain a person to perform the independent skills and activities of daily living."
†Defines *individual* as "natural person."

The state physical therapy practice act of New Mexico further defines direct care requirements as follows:

> A physical therapist shall not accept a patient for treatment without an existing medical diagnosis for the specific medical or physical problem made by a licensed primary care provider, except for those children participating in special education programs … and for acute care within the scope of the practice of physical therapy.

The act continues to define direct care requirements with the following statement:

> When physical therapy services are commenced under the same diagnosis, such diagnosis and plan of treatment must be communicated to the patient's primary health-care provider at intervals of at least once every sixty (60) days, unless otherwise indicated by the primary care provider. Such communication will be deemed complete as noted in the patient's medical record by the physical therapist.[10]

Fundamentally, the New Mexico state physical therapy practice act allows a physical therapist to obtain referrals from a veterinarian and to have direct access to veterinary physical therapy patients who are in need of acute care physical therapy, as long as communication occurs between the veterinarian and the physical therapist within 60 days of initiating treatment and regularly every 60 days thereafter. Interestingly, the New Mexico veterinary practice act also states that because a physical therapist must work under direct supervision of a veterinarian, the fee for physical therapy services must be paid to the licensed veterinarian or licensed veterinary facility.

This is an example in which the practice acts for veterinary medicine and physical therapy are apparently in conflict. Although some state physical therapy acts allow the treatment of animals by physical therapists, the veterinary practice acts in those states may not allow the same degree of freedom; therefore, it is important for the physical therapist and the veterinarian to understand both the physical therapy and the veterinary practice acts in the states where they are working. Although the veterinary practice acts in most states do not address the subjects of who may perform veterinary physical rehabilitation and direct access to patients by physical therapists, the veterinary profession has been careful to monitor who may practice veterinary medicine. State veterinary boards have been active in restraining the practice of veterinary medicine without a license, and the AVMA, through various councils and committees, has promoted the importance of the VCPR. These activities ensure that a veterinarian is the primary health care provider who manages the diagnosis, care, and treatment of the veterinary patients. In addition, the association with other professions to enhance and improve the quality of veterinary care has been encouraged by the veterinary profession, and these relationships allow for a role of other health care professionals such as physical therapists in the improvement of such care.

Collaborative Relationships between the Professions

In 1993 the APTA House of Delegates approved and adopted a position statement concerning collaborative relationships between physical therapists and veterinarians. This position statement reads as follows:

> The American Physical Therapy Association endorses the position that physical therapists may establish collaborative, collegial relationships with veterinarians for purposes of providing physical therapy services or consultation. Physical therapists are the provider of choice for the provision of physical services regardless of the client. A collegial relationship is advantageous to the client.[6]

On the national level, collegial relationships have been established between physical therapists and veterinarians for several years with a national liaison between the AVMA and the APTA. International meetings, such as the International Symposium on Rehabilitation and Physical Therapy in Veterinary Medicine, which has been held every 2 years since 1999, is another example of the growing collaboration between the two professions. The International Association of Veterinary Rehabilitation and Physical Therapy (www.iavrpt.org) sponsors this meeting. Other international collaborative organizations have been formed to foster the growth and document the status of veterinary physical rehabilitation in other nations. In some countries such as the United Kingdom and the Netherlands, physical therapists have been performing veterinary physical therapy in collaboration with veterinarians for many years, and the experiences of practitioners in these countries are being shared and exchanged.

There are many ways for physical therapists and veterinarians to work together. Veterinary colleges could employ physical therapists to provide appropriate care and therapy to patients requiring rehabilitation. Faculty from universities with colleges of veterinary medicine and physical therapy departments could work together to provide clinical service, teaching, and research activities that would benefit students and faculty members from both disciplines. Veterinary hospitals and clinics could employ physical therapists with either full-time or part-time appointments, or these hospitals could consult with physical therapists as needed. In jurisdictions where permitted such as in Colorado, physical therapists could establish veterinary rehabilitation therapy practices and solicit referrals from veterinarians in accordance with the state practice acts. Other venues where physical therapists might work include zoos; wildlife rehabilitation centers and parks; and canine and equine performance centers, exhibitions, and rodeos.

When collaborative relationships are initiated between veterinarians and physical therapists, the veterinarian must first establish a VCPR and then develop a care and

treatment plan after a diagnosis is confirmed. This information must be communicated to the physical therapist, preferably in writing, before the physical rehabilitation is initiated. Such communications are analogous to writing "doctor's orders" in a medical record. The physical therapist may wish to perform an independent evaluation and confirm a course of treatment in conjunction with the veterinarian. If opinions differ on the appropriate diagnosis and course of treatment, these differences should be negotiated in a professional manner. After the treatment plan is initiated, contacts and communications should continue between the physical therapist and the veterinarian regarding the progress and response of the patient throughout the course of therapy. As in human medicine, in which the physician is ultimately responsible for the patient, the veterinarian must be ultimately responsible for the care and treatment of the patient, because a VCPR must be maintained.

Risk Management for Physical Therapy and Veterinary Professionals

As physical therapists are finding new opportunities for professional services, including veterinary medicine, participation in these situations opens new questions about conduct and professional practice. There is no doubt that physical therapists have knowledge and skills that can benefit veterinary patients, but the legal and ethical issues regarding these collaborative relationships sometimes are unclear. Both the AVMA and the APTA have been supportive of these collaborative relationships; however, many states do not recognize these activities as falling within the scope of physical therapy. For this reason, physical therapists who choose to practice with animals in any professional capacity should be certain that they are acting in compliance with the physical therapy and veterinary medical practice acts in the states where they are working.

As stated earlier, the APTSIG has established a liaison program to assist members with an interest in rehabilitation for animals. The goal of this program is to obtain copies of the various state veterinary medical and physical therapy practice acts and to produce a resource directory of individuals interested in veterinary rehabilitation. A national liaison coordinator for this program has been identified. While this liaison program is helpful, interested individuals should contact appropriate licensing boards for clarification of questions.

See Box 2-2 for guidelines that are recommended when physical therapy practitioners are interested in practicing veterinary rehabilitation.

Veterinary practice acts in most jurisdictions are quite clear on the issues of the definition of veterinary practice, access to veterinary patients, and who may practice veterinary medicine. In all cases, the establishment of a VCPR is mandatory, and this rule necessitates that a physical therapist who is involved with an animal patient work closely with the veterinarian who has established the VCPR with that owner and patient. Otherwise, a physical therapist practicing on that animal could be in violation of a state veterinary practice act. When a VCPR has been established and followed, any potential legal issues should be minimized.

See Box 2-3 for guidelines that are recommended when veterinary practitioners are interested in practicing veterinary physical rehabilitation.

Box 2-2 Guidelines for Physical Therapists on Practicing Veterinary Physical Rehabilitation

1. If the physical therapy practice act includes language that limits the practice to humans by using terminology such as *humans*, *people*, or *persons*, then practicing on animals may be in violation of that specific practice act. If the term *patient* is used, the language of that practice act may be permissive, and the physical therapist should check with the state licensing board about the exact wording and the current interpretation (see Box 2-1).
2. After reviewing the state physical therapy practice act, if the wording is still unclear, the physical therapy practitioner should contact the state licensing board for written clarification or consult with an attorney for a legal opinion.
3. If the activities of interest are clarified in the state practice act (and are within the scope of physical therapy practice as defined by the APTA), one should be able to obtain professional liability insurance for these activities. If activities are specifically excluded in the state practice act, it is unlikely that professional liability insurance will provide coverage or protection for potential litigation.
4. PTAs who are providing veterinary physical rehabilitation also need to be aware of their state practice act regarding their practicing under the supervision of a licensed physical therapist. This does not necessarily mean on-site supervision; however, it does mean that the physical therapist has seen the patient and decided on a plan of care before the PTA initiates treatment. If they are performing animal rehabilitation under the supervision of a veterinarian (but without supervision of aPT), but not calling it *physical therapy*, this may be permitted based on the veterinary practice act in that individual state.
5. If a state precludes either physical therapists or PTAs from practicing veterinary physical therapy based on the state's practice act, the physical therapists or PTAs still may be able to work under the direct supervision of a veterinarian, but should not call their practice *physical therapy;* rather they should term it *physical rehabilitation, animal rehabilitation, canine* or *equine rehabilitation,* or something similar. The choice to do this would still have the potential to be in violation of the state practice act and would need to be examined on an individual basis.

Box 2-3 Guidelines for Veterinary Practitioners on Practicing Veterinary Physical Rehabilitation

1. If the term *physical therapy* or *physical therapist* is a protected term in that state, the term *physical rehabilitation, rehabilitation,* or a similar term should be used.
2. If a veterinary technician, physical therapist, or PTA is to provide the veterinary physical rehabilitation, supervision by a veterinarian may be defined differently state by state, and the veterinarian should review this, especially if the individual is providing care off-site, such as in home visits.

Reimbursement and Remuneration for Services

The reimbursement for professional services is another issue that must be resolved when there are collaborative relationships between veterinarians and physical therapists. Both health care professionals are entitled to an appropriate fee, which should be based on the services rendered with consideration for education and training of the health care provider. If the physical therapist is an employee of a veterinarian, the fee for the animal rehabilitation may be included in the total charges for veterinary services. Because the physical therapist is an employee, that individual would be paid directly by the veterinary clinic where employment occurs and services are rendered.

If the physical therapist is self-employed or an independent contractor, a veterinarian can arrange a referral or consultation directly with a physical therapist; however, a VCPR must originate and be maintained with the attending veterinarian. In that instance, a separate or direct billing arrangement for veterinary physical rehabilitation services could be appropriate if all involved parties are in agreement.

If the physical therapist is an employee of another health care provider, then that provider could charge the veterinarian or the owner directly, depending on the financial arrangements negotiated at the onset of the treatment plan. The attending veterinarian is ultimately responsible for the diagnosis, care, and treatment of the patient and must write instructions or orders to the health care provider and physical therapist affiliated with that provider. The physical therapist is an employee of the health care provider and is paid by that entity.

The physical therapist is entitled to a reasonable fee for services rendered. If a veterinarian refers a patient to another veterinarian, physical therapist, or health care provider, the original veterinarian is not allowed to accept part of the professional fee for the referral of the patient and owner. Such an arrangement is called *fee splitting* or a *kickback;* it is unethical and is in violation of the Principles of Veterinary Medical Ethics.[4]

In conclusion, the affiliations and collaboration between physical therapists and veterinarians will continue to grow with benefits for patients, owners, veterinarians, and physical therapists. With evolution and additional collaborative experiences in the discipline of animal physical rehabilitation, the specific roles of physical therapists and veterinarians will be clarified as well. Communication and cooperation between the professions of veterinary medicine and physical therapy, the APTA and AVMA, and the respective licensing boards of each state are necessary to successfully integrate physical therapy into the practice of veterinary medicine.

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3 Conceptual Overview of Physical Therapy, Veterinary Medicine, and Canine Physical Rehabilitation

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The purpose of this chapter is to provide a conceptual framework outlining the general practice of canine physical rehabilitation and physical therapy. Background information regarding the professions of physical therapy and veterinary medicine is included to assist professionals from each discipline to understand the history, educational requirements, and current practice of each profession. Methods to effectively bridge the gap between the physical therapy and veterinary professions are addressed, with models for collaborative practice outlined.

Physical Therapy as a Profession

History of the Physical Therapy Profession in the United States

Physical therapy began in the United States in the early 1900s and focused on treatment of acute anterior poliomyelitis, which reached its peak during the first 2 decades of the twentieth century. At this time physical therapy was not a true occupation; however, the foundations for the profession were developed. Some of these early applications of physical therapy included exercise, massage, and certain physical agent modalities.[1] The need for physical rehabilitation during and immediately following World War I served to further enhance the emerging field of physical therapy. From its origins, physical therapy has focused on restoring maximal function to individuals with disabilities.

Formal training in physical therapy began around 1918 and was developed by cooperative efforts between the office of the Surgeon General and personnel in civilian institutions. Individuals who completed these training courses were given the title of *reconstruction aides,* the earliest title of physical therapists.[1] Many of these individuals worked in the military during this time. The first national organization was the American Women's Physical Therapeutic Association, which was founded in 1921. In 1922 this name was changed to the American Physiotherapy Association, and in 1947 to the American Physical Therapy Association (APTA).[1] Throughout the 1940s physical therapy continued to evolve and focused on treating patients who had contracted polio or those injured in World War II. This was a period of major growth in physical therapy, and because of the shortage of physical therapists, many more had to be trained during this time. From the 1940s to the present, physical therapy has gradually become a more autonomous and scientifically based profession. Physical therapy is an accepted medical intervention, and approximately 750,000 people are treated by physical therapists in the United States each day.[2]

The American Physical Therapy Association

The APTA is a national professional organization representing more than 71,000 physical therapists, physical therapist assistants, and students in the United States.[3] Membership in the APTA is not mandatory for physical therapists practicing in the United States, and currently more than 172,000 physical therapists are licensed in the United States.

Major responsibilities of the APTA are monitoring and improving physical therapy education, practice, and research and educating the general public about the role of physical therapy in health care. The APTA political action committee (PAC) is one of the largest health care PACs in the nation. It was formed to empower the physical therapist profession to be more involved in the determination of federal laws and policies.

Physical therapists practice in many settings, including hospitals, school systems, private practices, extended care facilities and nursing homes, home health agencies, academic institutions, research centers, and government agencies. The APTA has 19 specialty sections, which represent various areas in physical therapy. These include acute care aquatic physical therapy, cardiovascular and pulmonary, clinical electrophysiology and wound management, education, federal physical therapy, geriatrics, hand rehabilitation, health policy and administration, home health, neurology, oncology, orthopedics, pediatrics, private practice, research, sports physical therapy, and women's health. A physical therapist may become board certified by the American Board of Physical Therapy Specialties (Diplomate, ABPTS) in one of seven areas (cardiopulmonary, clinical electrophysiology, geriatrics, neurology, orthopedics, pediatrics, and sports physical therapy).

The orthopedic section of the APTA houses the animal physical therapist special interest group, which was formed in 1998 and has quickly grown to more than 400 members. The goals of this group are to:

- Promote physical therapy
- Share information
- Collaborate with other health professionals
- Develop educational programs
- Foster research
- Create guidelines for practice
- Encourage appropriate legislative changes
- Establish a national network
- Protect professional practice

Physical Therapist and Physical Therapist Assistant Education

The current entry-level physical therapy degree is a master's or doctoral degree. In the past, the entry-level degree was a bachelor's degree. The APTA supports changing the entry-level degree to the doctoral level by the year 2020. There are approximately 200 APTA-accredited physical therapy educational programs in the United States, which graduate roughly 5700 physical therapists each year. Every physical therapist must pass a national licensing examination. Additional requirements for practice vary from state to state.

Physical therapist assistants complete a 2-year associate's degree from an APTA-accredited program. This training prepares them to provide therapeutic interventions that have been delegated by their supervising physical therapist, but assistants cannot evaluate or prescribe treatment. Requirements for licensure vary from state to state, as do the continuing education requirements.

Educational backgrounds of both physical therapists and physical therapist assistants do not include any formal training in the rehabilitation of animals. Animal anatomy may be studied during undergraduate education, but most likely this will be anatomy of the cat. Application of physical therapy to animals is not included in standard curricula; however, a few physical therapy programs now offer elective coursework in this area.

Veterinary Medicine as a Profession

History of the Veterinary Profession

The first college of veterinary medicine was established at Lyon, France. The first college of veterinary medicine in the United States was established at Iowa State University in the 1800s. The initial emphasis in veterinary medicine was on agricultural production and livestock. Gradually the emphasis on the treatment of individual animals shifted to herd management. The emergence of companion animals as members of the family, combined with the shift in agricultural demand, has resulted in the development of small-animal and equine practice as the predominant emphasis in veterinary medicine. Furthermore, the explosion of knowledge in small-animal medicine and surgery, combined with the era of specialization, has resulted in improved health care for pets. One area that has been neglected until recently is the rehabilitation of animals with chronic ailments and following surgery. The goals of this new area are to increase function, improve the ultimate outcome of patients following major surgery, and enhance the quality of life.

The American Veterinary Medical Association

The national professional organization that represents the approximately 84,000 veterinarians in the United States is the American Veterinary Medical Association (AVMA).[4] The AVMA is responsible for evaluating and credentialing veterinary medical education, administering the national board examination for veterinarians, and overseeing the various specialty colleges (currently there are 21). The American College of Veterinary Sports Medicine and Rehabilitation, American College of Veterinary Surgeons; American College of Veterinary Internal Medicine, neurology subspecialty; American College of Veterinary Emergency and Critical Care; American Board of Veterinary Practitioners; American College of Veterinary Behaviorists; and American College of Veterinary Nutrition are specialty colleges or boards whose members are likely to treat patients that may benefit from rehabilitation. Examples of other related organizations include the American Academy of Veterinary Acupuncture and the American Veterinary Chiropractic Association.

Veterinarians undergo broad training that includes studying diseases of large, small, and exotic animals. Veterinarians undergo training in making medical diagnoses, with the aim to identify the pathophysiologic nature of the condition and its subsequent treatment. Currently, the American College of Veterinary Sports Medicine and Rehabilitation has received approval by the American Board of Veterinary of Specialties, which oversees specialties of the AVMA. Training in this area is relatively limited, although a number of colleges now offer courses or lectures in physical rehabilitation.

Veterinary and Veterinary Technician Education

Veterinarians usually have an average of 4 years of undergraduate education, followed by 4 years of professional curriculum at an AVMA-approved college. Currently there are 33 approved colleges of veterinary medicine in the United States and Canada graduating approximately 2100 veterinarians yearly.[4] In addition, there are now 13 non-Canadian foreign colleges of veterinary medicine that are approved by the AVMA. Veterinarians are required to pass national and, in some cases, state board examinations

to practice. Some graduate veterinarians pursue additional training in the form of general internships and residencies in specialties. Specialty certification requires an internship or equivalent training, completion of a formal 2- to 3-year residency program, publication and research requirements, and successful completion of a certifying examination. There are currently 21 specialties recognized by the AVMA.

Veterinary technicians complete at least 2 years at an AVMA-accredited program and receive at least an associate's degree. There are currently nine distance learning programs in veterinary technology accredited or in the process of accreditation by the AVMA Committee on Veterinary Technician Education and Activities. In approximately 40 states and provinces, veterinary technicians are certified, registered, or licensed.[5] Candidates are tested for competency through an examination that may include oral, written, and practical portions. A state board of veterinary examiners or the appropriate state agency regulates this process. A national examination is available; however, requirements vary by individual states. Veterinary technician specialty organizations recognized by the North American Veterinary Technician Association include the Academy of Veterinary Emergency Critical Care Technicians, Academy of Veterinary Technician Anesthetists, Academy of Veterinary Surgical Technicians, Academy of Veterinary Dental Technicians, Academy of Internal Medicine for Veterinary Technicians, Academy of Equine Veterinary Nursing Technicians, Academy of Veterinary Nutrition Technicians, and the Academy of Veterinary Behavior Technicians.[6]

Veterinary and veterinary technician programs do not typically include any course work in physical rehabilitation. Although most veterinarians and veterinary technicians have a basic understanding of the rehabilitation process, they generally do not receive any formal training in this area. Application of physical rehabilitation to animals is not included in the standard curricula; however, a few veterinary programs now offer lectures or even elective courses in this area.

Continuing Education

Continuing education is essential to gain the knowledge in the reciprocal field that practitioners need to effectively perform canine rehabilitation and physical therapy. Some ways of accomplishing this are attending continuing education courses; self-guided study; attending meetings of the other profession; volunteering with members of the other profession; and attending related courses such as animal training, handling, and behavior.

A number of courses are offered in this area throughout the world. The First International Symposium on Rehabilitation and Physical Therapy, which was held at Oregon State University in 1999, helped to bring the professions of physical therapy and veterinary medicine together in a professional meeting. Research has been identified as critical to bridge the gap between professions and to establish the efficacy and credibility of physical therapy protocols and treatment. There are educational programs in the United States, the Netherlands, Japan, mainland Europe, Australia, and the United Kingdom.

Physical Therapy Evaluation and Intervention

Physical therapy encompasses a spectrum of services for humans, including evaluation, intervention, assessment, consultation, education, and research.[2] Evaluation includes taking a history; evaluating body systems to be certain that physical therapy is the appropriate medical intervention; and administering a variety of tests and measurements to determine a diagnosis, prognosis, and intervention. Physical therapists assess aerobic capacity and endurance; joint motion and integrity; muscle strength; arousal and cognition; need for assistive and adaptive devices; cranial nerve integrity; environmental barriers; body mechanics; gait; locomotion; balance; skin integrity; motor function; neuromotor development and sensory integration; orthotic, protective, and prosthetic devices; pain; posture; reflexes; circulation; and edema.[2]

The physical therapy diagnosis is both a process and label in which physical therapists perform an evaluation to provide a diagnose that identifies the effect a condition has on function.[2,7] Physical therapists proceed from the functional limitations that the animal presents and investigate the structures responsible for the impaired function. Based on the diagnosis, the physical therapist is able to specifically design individual treatment plans and evaluate their interventions

Physical therapists alleviate impairment and functional limitation by designing, implementing, and modifying therapeutic interventions that include the following[2]:

- Therapeutic exercise (range of motion [ROM], aerobic conditioning, gait training, aquatic therapy, muscle strengthening, balance, coordination, posture, motor control)
- Manual therapy techniques (joint mobilization and manipulation, massage, remodeling scar tissue)
- Wound management (dressings, topical agents, debridement, modalities, oxygen therapy)
- Airway clearance techniques (postural drainage, percussion, vibration, shaking)
- Orthotic and prosthetic intervention
- Electrotherapeutic modalities (electrical stimulation, laser)
- Thermal modalities (superficial heat and cold, and deep heat including ultrasound and diathermy)

Some of the common goals of physical therapy treatment are to decrease pain, improve muscle strength, retard atrophy, decrease swelling, decrease muscle spasm, increase the rate of tissue healing, remodel scar tissue, and improve function and independence in activities of daily

living. Prevention of injury, impairment, functional limitation, and disability is also part of physical therapy. This includes promoting and maintaining fitness, health, and quality of life in all age populations.

Bridging the Gap between Physical Therapy and Veterinary Medicine

Traditional Physical Therapy and Veterinary Models of Practice

Physical therapists commonly practice by referral from a physician, although more than 30 states currently allow direct access to a physical therapist, meaning that a physical therapist practicing in these states may see a patient without physician referral. Additionally, some states allow direct access for evaluation only, meaning that after the evaluation is complete, the patient must still see a physician to allow treatment to begin. In this model, the physical therapist typically relates the findings to the physician to expedite the initiation of treatment. As discussed in Chapter 2, the veterinary practice acts in most states require the physical therapist to work under the direct supervision of a veterinarian (although there are some exceptions), thus precluding a physical therapist from direct access in the care of veterinary patients.

Veterinarians in general practice see a variety of patients with various conditions. In some cases, a referral to a specialist is made for treatment requiring advanced procedures and equipment. In most cases the referred patient is returned to the referring veterinarian for follow-up care and any general patient concerns. Veterinary technicians assist with the diagnostic and treatment procedures under the direction of the veterinarian.

Team Approach to Rehabilitation

The individuals listed in Box 3-1 may be involved in a practice offering animal rehabilitation. The attending veterinarian is often the primary person responsible for the medical diagnosis and decisions regarding appropriate rehabilitative care. Depending on the injury and repair, specific recommendations are given to the person responsible for the rehabilitation. Precautions to therapy, especially exercises, must be clearly communicated. When veterinarians initiate physical rehabilitation in their practices, the initial sessions should be performed with all team members present, as differences in terminology may cause confusion and possibly result in injury. For example, performing ROM exercises may have different meaning to veterinarians, owners, and physical therapists. Communication and documentation between the team members are critical, and forms such as those illustrated later in this chapter provide an easy way to facilitate this.

The team approach involves a group evaluation or an assessment of the same patient by two or more clinicians within a short period. A core group of clinicians—for example, a veterinarian, physical therapist, and veterinary technician—is identified as the initial team. The initial team documents their findings, meets, and decides together the most effective plan for intervention. The core group may request additional evaluations by other experts on the team on an individual case basis. The appropriate clinicians are chosen to initiate the intervention plan. The team reviews the progress of the client periodically. One member of the team is responsible for follow-up after discharge.

This team approach allows for a variety of perspectives on health care issues. The team offers the client a detailed interdisciplinary assessment as well as the most cost-efficient method of providing services to resolve or treat the problem.

Box 3-1 List of Individuals Contributing to Animal Rehabilitation

Referring veterinarian
Veterinary specialist (surgeon, neurologist, internist, emergency and critical care veterinarian)
Rehabilitation veterinarian
Physical therapist on staff or as a consultant
Animal behaviorist
Veterinary nutritionist
Veterinary anesthesiologist
Veterinary acupuncturist
Veterinary chiropractor
Veterinary technician
Physical therapist assistant
Support staff
Orthotist/Prosthetist
Biomedical engineer
Owner

Collaborative Model for Practice

A proposed model for canine physical rehabilitation is outlined in Figure 3-1 and is based on a similar model for humans.

Documentation

Documentation should provide clear communication between all of the parties involved in the animal's care, including the veterinarian, the referring veterinarian (if applicable), the person responsible for the physical rehabilitation, and in some cases the owner. Because documentation is a critical form of communication, all patient files should be updated and documented at each physical rehabilitation treatment. Notes should be written in a clearly understandable, legible manner. An important characteristic of documentation is that the information should be easily obtained in a short period. A physical rehabilitation chart should give easy access to pertinent information such as functional status, ROM, treatment given, and patient

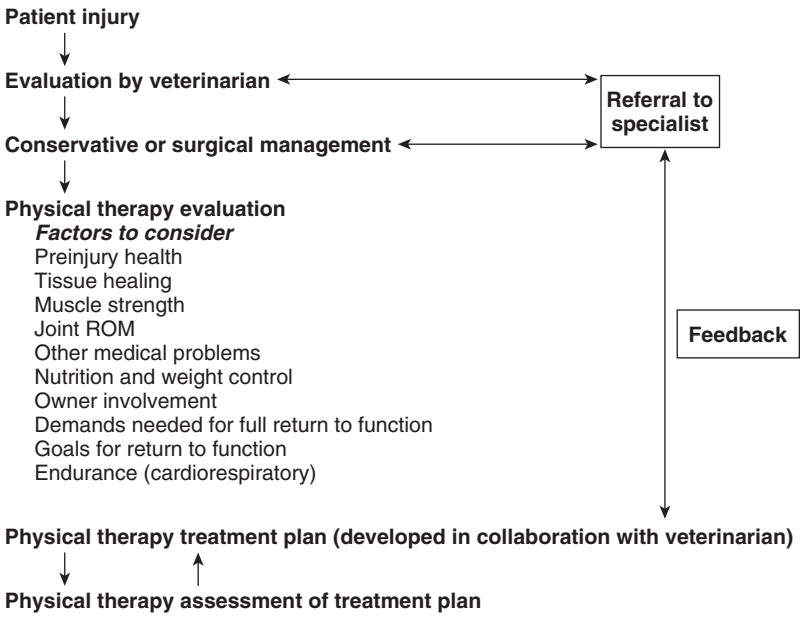


Figure 3-1 Proposed model for canine physical rehabilitation.

progress. Because terminology differs between professions, a glossary is included at the end of this book. An example of one difference is the directional term *anterior-posterior* used in human medicine versus *cranial-caudal* used in veterinary medicine. A treatment given once daily is written as *qd* in the human field, but *sid* in the veterinary profession (although qd is also used in veterinary medicine). Terms such as *closed kinetic chain exercises* (when the limb is in a weight-bearing position, such as in the weight-bearing or stance phase of walking) are not typically discussed in the veterinary field.

Documentation is also important from a legal perspective, as a record of the animal's condition and a clear documentation of the procedures performed. For example, consider an animal that has just completed a therapy session in which it demonstrated weight bearing on the affected extremity 75% of the time at a slow walk, and had a stifle ROM of 50 degrees flexion and 150 degrees extension, which was documented in the medical record. If this animal presents with non–weight-bearing lameness at the next treatment with a ROM of 60 degrees flexion and 130 degrees extension, that lends support to the idea that the animal had been injured not during therapy, but rather at home, in its cage, during a walk, or by some other means. Regardless of the mechanism of injury, the documentation may protect the therapist from accusations of fault, and also alert the therapist to contact the veterinarian. When an animal's status has changed, an evaluation by the veterinarian is necessary before therapy continues.

Another purpose of documentation is to verify the benefits of the treatment. The progress of the animal may be determined so that alterations can be made to the treatment protocol to further enhance recovery. Careful documentation and record keeping using objective assessment tools and standardized charts also provide a valid base for research. Conducting periodic chart audits can assist patient care in a variety of ways, including ensuring that all pertinent and necessary information is included, and that the treatment protocol is appropriate for the diagnosis.

The physical therapy chart should include the following key pieces of information:

1. A relevant medical history
2. The history of the present illness, including the attending veterinarian's referral information containing any precautions or contraindications for therapy, and treatment to be given
3. Any objective information such as ROM, function, limb girth measurements, and lameness scores
4. Primary problems to be addressed and goals for the treatment
5. The treatment itself in as much detail as possible
6. The response to treatment

Examples of documentation forms in this chapter include the following:

1. Client information form (Figure 3-2)
2. Referral form (Figure 3-3)
3. Physical rehabilitation evaluation form (Figure 3-4)
4. Orthopedic evaluation form (Figure 3-5)
5. Neurologic evaluation form (Figure 3-6)
6. Daily flowsheet form (Figure 3-7)
7. Referral letter (Figure 3-8)

Text continued on p. 30

NEW CLIENT INFORMATION

Owner's name	
Address	
Home phone	
Work phone	
Veterinarian/surgeon	
Date of surgery/injury	

PATIENT INFORMATION:

Name	
Breed	
Age	
Sex	
Color	
Spayed/neutered	

PATIENT HISTORY:

Rabies vaccination	
Past medical history	
Previous surgery	
Allergies	
Special diet/medication	
Previous activity level	
History of present illness	
Treatment since injury/surgery	
Owner's goals	

Figure 3-2 Client information form.

CANINE REHABILITATION CLINIC

Referral Form

Client_____ Patient_____ Date_____

Breed_____ Sex_____ Age_____ Weight_____

Referring veterinarian/clinic:_____

Clinical condition:_____ Onset/Sx date:_____

Special instructions/precautions_____

Frequency and duration: _____Times per day for _____days

☐Board until _____ Drop off: ☐ MWF ☐ Other_____

☐ T/Th

Plan: ☐ Evaluate and treat

☐ Hot pack
☐ Cryotherapy
☐ Ultrasound
☐ Electrical stimulation
☐ Therapeutic exercise
☐ Hydrotherapy
☐ Gait training
☐ Massage
☐ Joint mobilizations
☐ Weight-bearing/weight shifts
☐ Passive range of motion
☐ Neuromuscular reeducation
☐ OTHER:_____

DVM Signature_____

Figure 3-3 Referral form.

PHYSICAL THERAPY INITIAL EVALUATION

Patient's name:	
Date:	

PHYSICAL EXAMINATION:

Skin/incisions:		**Color/temp:**	
Heart rate:		**Respirations:**	

POSTURE/GAIT:

General observation:				
Preop/injury lameness:	**Walk:**		**Trot:**	
Postop/injury lameness:	**Walk:**		**Trot:**	
Standing limb position:		**Sitting limb position:**		
Circumference (cm):	**70% femur**	**80% humerus**	**Joint line**	**Other**
Affected:				
Unaffected:				
Other:				

RANGE OF MOTION:

Joint(s): Aff/Unaff	**Flexion**	**Extension**	**AB/adduction**	**Varus/Valgus**	**Other**
Hip:					
Stifle:					
Hock:					
Shoulder:					
Elbow:					
Carpus:					
Other:					

PALPATION:

Forelimb	
Hind limb	
Spine	
Other	

SPECIAL TESTS:

Neurologic:	
Orthopedic:	
Functional:	
Other:	

Figure 3-4 Physical rehabilitation evaluation form.

Continued

TREATMENT:

Modalities:	Manual:	Therex:
Interferential current	Massage	Gait training
Neuro-muscular electrical stimulation	Joint mobilization	Aquatic
	Passive range of motion	Functional
		Swiss ball
Other stim		Foam roll
Ultrasound	Other:	Owner education
Ice		Protocol review
Heat		Other:
Other		

ASSESSMENT/GOALS:

Decrease pain	
Decrease edema	
Increase weight-bearing	
Independent home exercise program	
Return to previous function	
Other	

PLAN:

Return visit	
Call for follow-up	
Call DVM	
Other	

DVM Signature _____

Figure 3-4, cont'd

CANINE ORTHOPEDIC REHABILITATION
EVALUATION FORM

Patient_____ Client_____ Date_____

Breed_____ Age_____ Sex_____ Date of Sx/onset_____

Referring vet/clinic:_____ Diagnosis_____

History:_____
_____
_____
_____

Medications:_____
Client's goals_____
_____
Functional mobility_____
_____

OBJECTIVE
Involved limb: RF LF RR LR

Range of motion:

Forelimb	**R**	**L**
Shoulder		
1. Flexion	_____/	_____
2. Extension	_____/	_____
Elbow		
1. Flexion	_____/	_____
2. Extension	_____/	_____
Carpus		
1. Flexion	_____/	_____
2. Extension	_____/	_____

Rear limb	**R**	**L**
Hip		
1. Flexion	_____/	_____
2. Extension	_____/	_____
Stifle		
1. Flexion	_____/	_____
2. Extension	_____/	_____
Hock		
1. Flexion	_____/	_____
2. Extension	_____/	_____
Other	_____/	_____

Visual inspection/palpation:_____
_____
_____
_____
_____

Pain score:
0 = No pain on palpation of joint
1 = Mild pain; palpation completed
2 = Moderate pain; palpation completed with obvious discomfort noted
3 = Severe pain; palpation not completed
4 = Pain too severe; restraint/sedation needed to palpate

Limb circumference:

Muscle mass:			*Joint effusion:*		
Affected_____	Unaffected_____		Affected_____	Unaffected_____	
Femur length _____cm	_____cm		Patellar tendon_____ cm	_____cm	
70% of length _____ cm	_____cm		2" above _____ cm	_____cm	
Girth _____ cm	_____cm		2" below _____ cm	_____cm	

Figure 3-5 Orthopedic evaluation form.

Continued

Gait analysis:

Orthopedic lameness

Degree of lameness (stance)	Degree of lameness (walk)	Degree of lameness (trot)
0 = Normal stance	0 = No lameness/weight-bearing on all strides observed	0 = No lameness/weight-bearing on all strides observed
1 = Slightly abnormal stance (partial weight-bearing)	1 = Mild subtle lameness with partial weight-bearing	1 = Mild subtle lameness with partial weight-bearing
2 = Moderately abnormal stance (toe-touch weight-bearing)	2 = Obvious lameness with partial weight-bearing	2 = Obvious lameness with partial weight-bearing
3 = Severely abnormal stance (holds limb off the floor)	3 = Obvious lameness with intermittent weight-bearing	3 = Obvious lameness with intermittent weight-bearing
4 = Unable to stand	4 = Full non-weight-bearing lame	4 = Full non-weight-bearing lame

Post-operative limb use

Degree of limb use (stance)	Degree of limb use (walk)	Degree of limb use (trot)
0 = Normal stance	0 = No lameness	0 = No lameness
1 = Slightly abnormal stance (partial weight-bearing)	1 = Lame but weight-bearing on >95% of strides	1 = Lame but weight-bearing on >95% of strides
2 = Moderately abnormal stance (toe-touch weight-bearing)	2 = Lame but weight-bearing on >50% and <95% of strides	2 = Lame but weight-bearing on >50% and <95% of strides
3 = Severely abnormal stance (holds limb off the floor)	3 = Lame but weight-bearing on >5% and <50% of strides	3 = Lame but weight-bearing on >5% and <50% of strides
4 = Unable to stand	4 = Non-weight-bearing lame or weight-bearing on <5% of strides	4 = Non-weight-bearing lame or weight-bearing on <5% of strides

Gait deviations:_____

ASSESSMENT_____

PLAN OF CARE_____

Therapist Signature_____

Figure 3-5, cont'd

CANINE NEUROLOGIC/SPINAL REHABILITATION EVALUATION FORM

Client_____ Patient_____ Date_____

Breed_____ Age_____ Sex_____ Date of Sx/Onset_____

Referring vet/clinic:_____ Diagnosis_____

History:_____

_____

_____

Prior surgeries:_____

Follow-up visit:_____ Medications:_____

Client's goals:_____

_____

OBJECTIVE

Involved area(s) : RF LF RR LR Spine_____

Mental status: Alert Depressed Stuporous Comatose Aggressive Fearful Disoriented

Palpation/postures:_____

_____

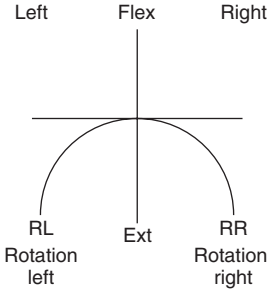
_____

_____

_____

Active movements:

Left Flex Right



KEY:
Affected Body Segment
X Tender
O Center of pain
≡ Spasm
/// Guarding
* Reflex contraction
∞ ROM

Limb circumference:

Muscle mass:

Affected_____		Unaffected_____
Femur length_____cm		_____cm
70% of length_____cm		_____cm
Girth	_____cm	_____cm

Figure 3-6 Neurologic evaluation form.

Continued

Gait analysis:
Involved limb(s):_____

Degree of deficit (stance) _____ Degree of deficit (walk) _____ Degree of deficit (trot) _____

5 = Normal strength and coordination
4 = Can stand to support; **minimal paraparesis** and ataxia
3 = Can stand to support but frequently stumbles and falls; **mild paraparesis** and ataxia
2 = Unable to stand to support; when assisted, moves limbs readily but stumbles and falls frequently; **moderate paraparesis** and ataxia
1 = Unable to stand to support; slight movement; when supported **severe paraparesis**
0 = Absence of purposeful movement; **paraplegia** or **tetraplegia**

Deviations:_____

Proprioception: (+ intact; – absent)

RF	LF	RR	LR

Spinal reflexes:
Key: 0 = Absent
+1 = Depressed
+2 = Normal
+3 = Exaggerated
+4 = Clonus
NE = Not examined

Crossed extensor: _____ Forelimb
+ Present _____ Hindlimb
– Absent

Forelimb	RF	LF	RR	LR
Flexor reflex				
Biceps (C6-C8)				
Triceps (C7-T2)				
Ext carpi rad (C7-T1)				
Deep pain				
Hindlimb				
Flexor reflex				
Patella (L4-L6)				
Cranial tibial (L6-L7)				
Gastrocnemius (L7-S1)				
Sciatic (L6-S1)				
Perineal (S1-S3)				
Deep pain				
Cutaneous trunci				

ASSESSMENT_____

PLAN OF CARE_____

Therapist Signature_____

Figure 3-6, cont'd

CANINE ORTHOPEDIC REHABILITATION FLOWSHEET

PATIENT NAME:_____ CLINICAL CONDITION:_____ SX DATE:_____

CLIENT NAME:_____

TIME	Date	Date	Date	Date	Date

Reassessment: Date / /

ROM Shoulder R_____ L_____ Hip R_____ L_____

Elbow R_____ L_____ Stifle R_____ L_____

Carpus R_____ L_____ Hock R_____ L_____

Weight-bearing status:_____

Gait analysis:_____

_____

Proprioception: RF LF RR LR

OTHER:_____

_____

_____

_____

PLAN:_____

_____

_____

_____

Limb circumference

Affected limb _____ Unaffected limb _____

_____ _____

_____ _____

Reflexes: R L

Forelimb flexor

Rear limb flexor

Patellar

Therapist Signature_____

Figure 3-7 Daily flowsheet form.

CANINE REHABILITATION CLINIC

{Date}

{Address}

Dear Dr. { },

Thank you for referring "Star" to Physical Therapy at The Canine Rehabilitation Clinic and Veterinary Hospital. Star has been treated for 3 days with ultrasound to help resolve the contracture of the digits of the right forelimb and the underwater treadmill to help regain full function of the left hindlimb. We are encouraging Star to use the leg in a buoyant environment to avoid exacerbating the contracture; second, the thermal effects of the water will help with improving joint mobility, and finally, the resistant property of the water will help to strengthen the muscles of the limb. I have also reiterated to her owner, Diane, to take Star for short walks (no more than 10 to 15 minutes at a time) no more than 2 to 3 times per day (on the off days from physical therapy).

Dr. Smith has left a message outlining the rehabilitation process and changes in medications to avoid further GI upset for Star. So far, we have seen no adverse reactions to the medications.

Again, thank you for this referral. We will continue physical therapy for 2 days this week and will provide the owner with a home care program to continue strengthening activities for Star. We expect that she will have progressive improvement.

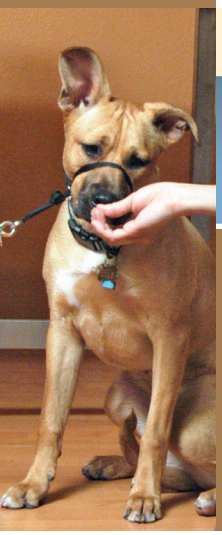
Please do not hesitate to call if you have any questions or concerns: {Phone number}.

Sincerely,

Figure 3-8 Referral letter.

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SECTION II

Basic Science of Veterinary Rehabilitation

4 Canine Behavior

Julie Albright

Optimal benefits of physical rehabilitation are achieved with a willing and relaxed patient. An animal that has enjoyable experiences and trusts the handler is more inclined to comply with rehabilitation procedures, which may result in greater success. For example, muscle contraction that occurs in a tense animal is contraindicated during passive range-of-motion exercises.[1] Similarly, a patient's avoidance of aversive handling could interfere with the detection of pain, an indicator of potential tissue damage during cryotherapy[2] or neuromuscular electrical stimulation sessions.[3] A physical rehabilitation session can subject an animal to potentially painful or frightening procedures that may result in aggressive behavior. Therefore making physical rehabilitation a pleasant experience for the animal can also decrease the risk of injury to the therapist and pet. Pet owners are more inclined to continue rehabilitation if their animals do not appear distressed or hesitant about the facility or personnel. Interpreting body postures and vocalizations in our patients is essential in recognizing fear and stress. Therefore this chapter begins with a basic review of canine and feline communication. Humane handling and behavior modification techniques to decrease fear and ensure a willing patient are also discussed.

Canine Communication

Body posture and facial expressions are usually the first clue to a dog's emotional state. Overt postures such as stiff, forward body position with erect ears and tail signal alertness and offensive aggression (Figure 4-1, dog 6). An offensively aggressive dog's jaw is squared and only front teeth are exposed (Figure 4-2, dog 3). Conversely, a dog that is trembling, in a crouching position, or on its side with its ears and tail tucked is easily recognized as a fearful dog (see Figure 4-1, dogs 4, 5, and 9; Figure 4-3). A fearful or defensively aggressive dog is also lower to the ground compared with assertive dogs (see Figure 4-1, dog 8), often approaches and retreats, and has looser facial expressions that allow all the teeth to be visible (see Figure 4-2, dogs 8 and 9). More subtle behaviors such as panting, lip licking, and yawning can also indicate stress in a dog. During or between procedures, one may notice the dog sitting or lying very still with eyes closed. This is another sign of stress or conflict that may be mistaken for a state of sleepiness or relaxation. While performing potentially painful or threatening procedures, handlers should not only watch for overt body postures, such as lip lifting (snarling), but also less perceptible signals such as an immobilization of the body or a pause in panting. Both of these behaviors may indicate the dog has just become more aroused and aggression may follow.

Two actions often misinterpreted are the tail wag and piloerection (or "raising hackles"). Although the tail wag can indicate a friendly dog and piloerection can indicate an aggressive dog, this is an oversimplification. A dog with a loose body posture and low, loose wagging tail may indeed be friendly, but a dog with a stiff body and a rapidly wagging tail, especially if isolated to the tail tip, is probably agitated.[4,5] Piloerection is triggered by a sympathetic response and signifies a reactive animal that may be fearful or imminently aggressive. Some contend that hair raised across the entire dorsum may be indicative of the latter and piloerection concentrated on just the dorsal hips or shoulders is a more fear-motivated response.[5]

Vocalizations are also helpful indicators of a dog's mood. In general, harsh, low-frequency sounds are an attempt to make the receiver back away, and high-pitched tonal vocalizations are more friendly or appeasing in nature.[6] Barking in dogs can convey a wide range of emotions such as alarm, attention-seeking, distress, threat, or warning. A dog distressed by isolation will give a high-pitched bark, whereas alarm or aggressive barks are lower in frequency and harsher sounding.[7,8] Aggressive barks are usually given in short, rapid sequence.[8] Growling occurs in aggressive and nonaggressive contexts among dogs, but when targeted to humans, growling is typically a low-level threat that can be indicative of defensive or offensive

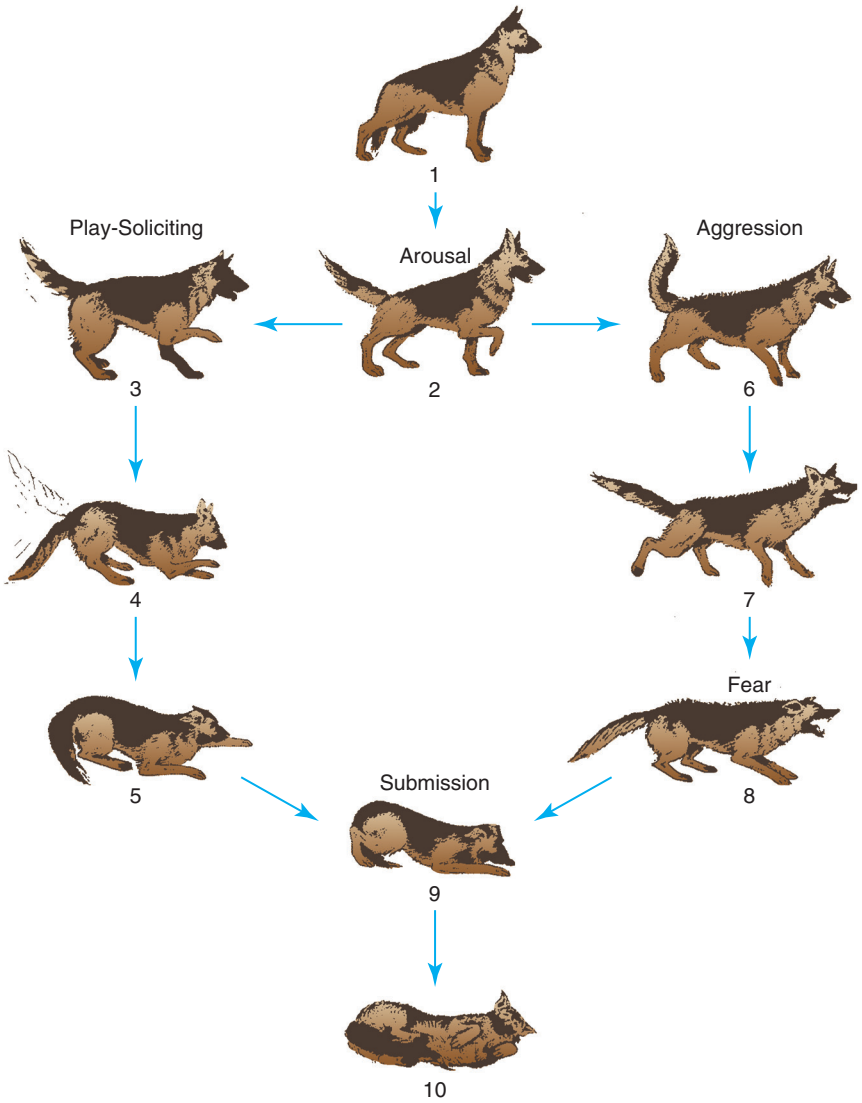


Figure 4-1 General body postures of dogs. Dog 1 shows a relaxed dog. Dog 2 is alert. Dog 3 shows playful behavior. Dogs 4 and 5 show increasing fear and submission. Dog 6 displays offensive aggression. Dog 7 shows mixed motivations of offensive and defensive aggression. Dog 8 shows defensive aggression. Dogs 9 and 10 show fear and/or submission. (From Overall KL: *Clinical behavioral medicine for small animals,* St Louis, 1997, Mosby.)

aggression.[9] A whine has cyclic frequencies and, in adult dogs, is typically made when the dog is in pain or frustrated.[10] Adult dog yelps are barklike and signify similar motivations as whines when directed at humans. Yelps are often heard during situations of greeting, submission, and play solicitation.[11] Howling is more common in nondomestic *canids* species such as wolves and coyotes and occurs as a long-distance call among pack members as a group vocalization to warn an approaching stranger, or possibly to promote pack affinity.[12] In domestic dogs, howling may be a response to a sound similar to a howl (e.g., siren or singing owner) and may represent a group vocalization.[11,12] In a physical rehabilitation facility, barks or howls may occur as a distress behavior when the dog is isolated in a cage or run. Barks are most commonly excited or aggressive displays in response to the presence of people or other animals. The motivation to vocalize should be reduced to decrease distress in the dogs and also the damage to human and patient hearing.

Dogs have an amazing sense of smell; thus urine and other olfactory stimuli are probably the most salient form of communication to the canine sensory system. In contrast, they may be the least recognized by humans in interspecies communication with dogs. In the rehabilitation facility, a dog that urinates, defecates, or expresses anal sacs, especially while displaying fearful body postures, is very distressed or excited. As a territorial behavior, some dogs may feel the need to urine mark, especially vertical surfaces previously urinated on by other dogs. In the clinic setting, stress may also be a contributing factor.

Feline Communication

As with dogs, a cat's vocalizations may help us to recognize its inner state.[10,13] The most common and variable vocalization is the meow and is given to seek attention or greet a social partner. A soft, rhythmical murmur is also given during greeting and social interactions. Purring is a

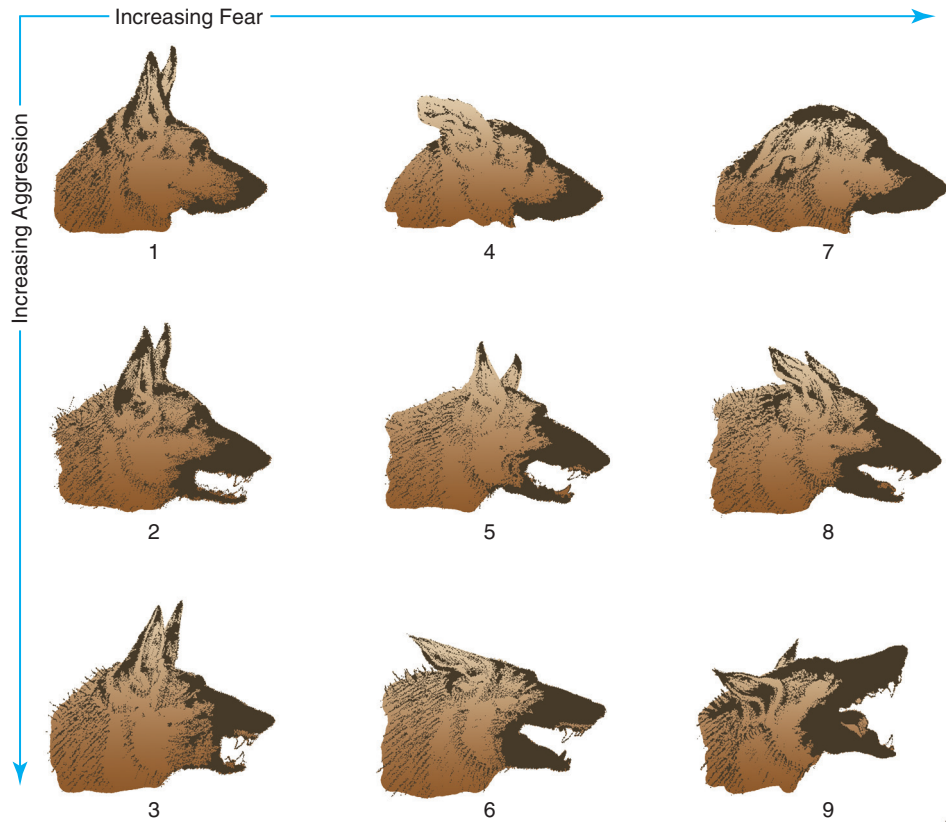


Figure 4-2 Facial postures of dogs. Figures from left to right show increasing fear. Figures from top to bottom show increasing aggressive motivation. Dog 1 is an alert dog. Dog 3 is offensively aggressive. Dog 7 is fearful and/or submissive. Dog 9 is defensively aggressive. All others are intermediate in fear and/or aggression. (From Overall KL: *Clinical behavioral medicine for small animals,* St Louis, 1997, Mosby.)

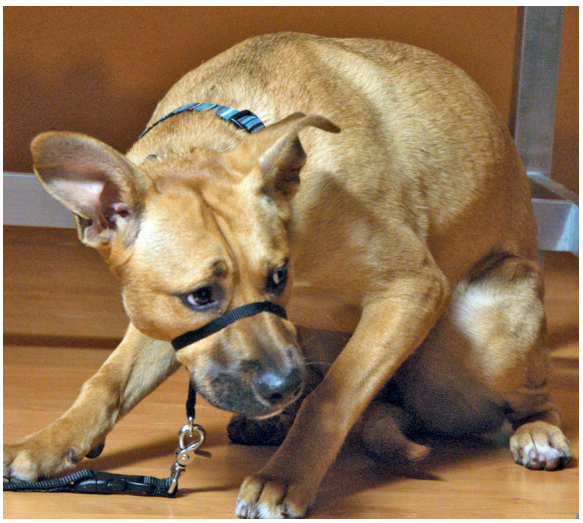


Figure 4-3 Fearful dog crouching, furrowing brows, and averting gaze. (Photo used by permission from Yin S: *Low stress handling, restraint and behavior modification of dogs and cats,* Davis, Calif, 2009, Cattle Dog Publishing.)

unique *felid* sound that is made by rapid contractions of the laryngeal muscles, innervated by a free-running neural oscillator that contracts and releases every 30-40 milliseconds.[14,15] People often assume a purring cat is a content cat, but this behavior can also occur in an anxious, painful, or sick cat. A cat purring during these stressful times is perhaps trying to solicit attention just as the purr from a very young kitten solicits attention from its queen. Aggressive cats may produce loud, harsh howls and growls, as well as hisses and spits. The growl is lower in frequency and longer in duration than the howl. The hiss is an open-mouthed sound invariably displayed in a defensively aggressive cat. During a painful event, a cat may give a loud, quick, high-pitched shriek, probably intended to startle the attacker into release.[16]

Emotions can also be visually communicated through body and facial postures.[5,10,16] A cat with an erect but curved tail, head up, and relaxed ear position is displaying a friendly greeting. Conversely, a cat with a vertically raised, piloerected tail with an arched back, and ears swiveled down or flattened against the head is defensively aggressive. As a cat becomes more frightened, its tail moves into a concave position or is tucked between the

legs. An offensively aggressive cat's tail is lowered, but stiff and lashing. The body appears to be slanting down from tail to head and erect ears rotated back so the inside of the pinna is visible. An offensively aggressive cat's pupils are constricted but become more dilated with a feeling of fear and defense.

Patient Fear and Aggression during Rehabilitation

In a physical rehabilitation or veterinary setting, fear is the predominant motivation for aggression. Most animals show clear signs of fear and defense, but some may appear offensively aggressive. It is tempting to ascribe "dominance" to these animals, but on closer observation one usually sees the subtle and often fleeting signs of defensive aggression such as alternation of approach and retreat, gaze aversion, and ears shifting back. The dog motivated by fear often adopts more assertive postures when confronted with fear-producing stimuli, especially if the aggression successfully drove away the stimulus during previous encounters.

Dominance is a description of a relationship between individuals in which agonistic encounters (aggression and submission) establish who has priority to valued items, such as food or mates.[17,18] Using dominance as a static attribute of an individual's personality is incorrect. Dominance and territorial aggression are highly unlikely to be motivators for pets in a therapy facility. This is because they do not have an extended social relationship with personnel, nor have they likely spent enough time in the facility to become territorial. However, frustration and arousal caused by confinement can trigger cage or kennel aggression in an animal distressed by the sights, sounds, and smells of a facility. This is often misidentified as territorial aggression. Fear is the most reasonable impetus for aggression considering the circumstances.

Irrespective of an animal's emotional state, forceful or pain-inducing handling of animals is never warranted and is usually counterproductive.[18,19] Harsh methods of restraint can escalate a fear response to fear aggression, causing injury to the pet and handler. Once a pet has a fearful or aggressive response in the facility, subsequent visits will result in an even more extreme reaction, thus preventing the therapist from accurately assessing and performing treatments or exercises. Furthermore, owners will be less willing to bring the animal to the clinic for future treatments because of the animal's behavior and welfare. Clients will be more likely to endure the inconvenience of bringing a pet to rehabilitation if they feel the pet is being handled humanely and the pet seems to enjoy trips to the clinic.

Learning and Behavior Modification

Management of patient behavior begins with an understanding of how an animal learns. Learning is basically how consequences influence behavior and is broadly categorized as *nonassociative* or *associative*.[20] Nonassociative learning can be further categorized into *habituation* or *sensitization. Operant conditioning* and *classical conditioning* are the two types of associative learning.

Habituation occurs when an animal learns not to respond to an inconsequential stimulus on repeated exposures. *Flooding* is a term often used to describe exposing an animal to a high-intensity stimulus in the hope that the animal will stop reacting. Instead of habituation, *sensitization,* or an increase in the animal's reaction to a stimulus, may result. The nature of the stimulus (e.g., loud noise) and the level of stress the pet is experiencing are important factors in determining whether the result will be habituation or sensitization. Furthermore, an animal that is suffering from prolonged distress may experience *dishabituation* of stimuli and react fearfully toward objects or people to which it previously seemed indifferent.[21]

An example of nonassociative learning is a nervous dog that may be hesitant when urged to enter an underwater treadmill. The human handler detects nothing frightening about the apparatus so gently forces the dog into the treadmill with the expectation it will "just get over it" (i.e., flooding in an attempt to habituate). However, the stress the dog is experiencing may cause sensitization to the treadmill, handler, or even generalize to the entire facility, resulting in a dog that balks more vehemently and potentially becomes aggressive each time it is near the treadmill.

In this example, the dog has associated the surrounding stimuli such as the staff or room containing the treadmill with unpleasant physiologic effects of fear previously experienced in the treadmill. In this *classical conditioning* (or *Pavlovian learning*), a neutral stimulus is paired with a stimulus that naturally triggers a physiologic response (such as fear, pleasure, or excitement). After repeated pairings, the neutral stimulus alone involuntarily evokes the same response. Conversely, operant *conditioning,* also termed *instrumental learning,* is the voluntary performance of a behavior based on previous consequences. For example, an animal will try to repeat a behavior that results in a reward (*reinforcement*) and avoid those that cause unpleasant sensations such as pain or fear (*punishment*). Most animal training is based on this type of conditioning.

Reinforcement or punishment can be further qualified as negative or positive depending on whether something was applied or removed, not the emotion of the animal or trainer. In other words, a positively reinforced behavior is one repeated because the animal previously received something desirable immediately after performing that action. A behavior may also recur because the animal has learned that action will cause something aversive to be removed. This is negative reinforcement. A head halter is often used to decrease pulling on a leash (see Humane Control and

Safety Devices later in this chapter for more detailed description). Walking close to the handler is negatively reinforced because the pressure on the nose loop is released when the dog stops pulling and he learns that remaining near the human handler prevents the nose pressure. This is often confused with positive punishment, or decreasing the likelihood of a behavior by inflicting something aversive on the animal after it has performed an undesirable behavior. In the same example, the pulling behavior decreases as the walk proceeds because the dog learns jerking ahead of the handler exacts pressure on the nose. Pulling is positively punished, and remaining near the human is negatively reinforced. A behavior can also become less likely through negative punishment, or removal of something the animal considers rewarding when it performs an unwanted behavior. Most dogs consider their owners' affections enjoyable so removing this stimulus when the dog is barking or jumping for attention decreases this pestering behavior. The best result comes from well-timed positive reinforcement (giving attention) when the dog is behaving appropriately (sitting) in addition to the immediate negative punishment (removal of attention) of the unruly behavior (jumping).

Success of behavior modification depends on the timing and relevancy of the behavior with the punisher or reinforcer. The stimulus must be applied within just a second or two of the animal's action and be applied consistently for the animal to be able to predict which behaviors elicited the pleasant or unpleasant consequences. Inappropriately applied rewards can result in problems such as rewarding the incorrect behaviors and potentially increasing anxiety. However, poorly applied punishment does not just result in poor efficacy, but also becomes a welfare concern. The inability of an animal to predict and control when it will receive something that induces fear or pain quickly creates a distressed animal. Animals that perceive an aversive event as unavoidable may become apathetic and not attempt escape, a phenomenon called *learned helplessness.*[22] This indifference could be mistaken for relaxation, but short- and long-term activation of the stress neuroendocrine system may lead to compromised health and mental well-being in therapy patients.

Positive punishment is often the technique first employed to try to change an animal's unwanted behavior. However, for the reasons previously discussed, this is rarely appropriate in a clinic or therapy setting. Subjecting an animal to discomfort or verbal reprimand that induces an unpleasant emotional state may suppress behavior (operant conditioning), but also classically conditions fear of the facility and staff. The use of positive reinforcement, negative punishment, and environmental management (e.g., quiet facility and training of staff to correctly read subtle animal body postures) is strongly recommended in a clinic to prevent fear and escape behaviors. The inconvenience, dietary restrictions of some patients, and bias against "bribing" our pets for certain behaviors often limit the use of food in the clinic. Anything the pet finds pleasurable (toy, presence of owner, an activity like "fetch") can be used as a reward, and thus given as a reinforcer or removed as a punisher. In reality, however, food is an intrinsic need and the most effective, consistent motivator for our patients. Clients can be instructed to consult with a reputable behaviorist or trainer[23] regarding the importance of setting behavior criteria and reward schedules to avoid "bribing" for good behavior. But this is of little concern in the clinic because maintaining a relaxed emotional state in an environment fraught with novel and potentially frightening experiences requires a large "pay check" for the pet. If obesity is a concern, the owner can adjust the diet at home to ensure the appropriate caloric intake. Unless a patient cannot be fasted, the owner should be instructed to feed the animal a maximum of half the morning meal; very small treats are provided during the sessions so the pet remains motivated to take the food. A variety of very small treats should be kept on hand in each therapy room. Common favorite foods are real meat or fish, semimoist treats, or sticky foods like peanut butter, meat-flavored baby food, or flavored pastes in nozzle cans.*

The food can be used to *countercondition* or change the animal's emotional response to the target that elicits fear. As a form of associative learning, counterconditioning can be classical or operant in nature. Classical counterconditioning involves simply providing the dog with rewards in the presence of the fear-producing target with the expectations of replacing the fear response with a pleasant emotion. Often the outcome is more successful if the dog is asked to perform a previously trained command that is incompatible with the fear response while in the presence of the stimulus (operant counterconditioning). Again using the underwater treadmill as an example, a dog that is trembling or attempting to flee as the water fills the chamber could be rewarded for moving forward as the water flow begins by targeting this skill that has been previously mastered outside the apparatus (see Handling of the Physical Rehabilitation Patient later in this chapter). In both classical and operant counterconditioning protocols, the emotional state is being changed. Frequently the target produces such a level of stress that getting the animal focused on a command or a reward may be very difficult. Thus most behaviorists advocate a *systematic desensitization* protocol in which the animal is habituated to a very low stimulus level (e.g., expose the dog to the treadmill at great distance), and then the intensity level is gradually increased (the dog is brought closer to treadmill) with the goal of never triggering fear. Typically a dog is asked to perform commands for treats as the distance is decreased. This is called a *desensitization/counterconditioning protocol* and is the crux of most behavior modification.

*Kong Easy Treat, Kong Company, Golden, Colorado.

Setting the Stage

Preventing anxiety rather than trying to calm a highly distressed dog is a more successful strategy in maintaining a compliant patient. This begins long before the dog enters the treatment room. A dog that has car distress or motion sickness on the way to the clinic is more likely to be stressed during the session. The dog's regular veterinarian should be consulted regarding antiemetic medications if nausea or vomiting during the car ride is a consistent problem. Other strategies to decrease car distress include desensitization to the car itself and then the motion of riding in the car.[18] Visual restriction (e.g., Calming Cap* as shown in Figure 4-4), stabilization via a crate or seatbelt, as well as pheromone therapy[24,25] and lavender aromatherapy[26] can have a calming effect on dogs showing excitable or stress behaviors in the car.

The reception area and therapy rooms should be designed to minimize stimuli that can cause excitement. The waiting room can be made less stressful by instructing staff to move slowly and minimize noise. Arranging seating and providing solid structures in the sitting area so fearful or reactive animals can be visually isolated can be very helpful. Visual sequestration is also recommended for the cage and kennel area as well. Cats should always be provided with hiding places and dogs in cages and larger runs may also feel more secure if a partial visual barrier is provided.

First impressions with staff are important in gaining the patient's trust. Quickly approaching, leaning over, and reaching out are considered threating actions to a dog (Figure 4-5, *A*). Instead, the staff member should provide enough time and space for the dog to acclimate to its new surroundings (Figure 4-5, *B*). A crouching or sitting human who is avoiding eye contact is much less threatening. Treats can be gently rolled to the dog from a distance as

*Calming Cap, Premier Pet Products, Midlothian, Virginia.



Figure 4-4 A Calming Cap can decrease visual stimulation and stress during car rides. (Photo courtesy of Thunderworks, Durham, North Carolina.)



Figure 4-5 **A,** Incorrect greeting: the staff member leans and reaches over the dog. The dog perceives this action as a threat and reacts with fearful body postures and avoidance of the approaching person. (Photo used by permission from Yin S: *Low stress handling, restraint and behavior modification of dogs and cats,* Davis, Calif, 2009, Cattle Dog Publishing.) **B,** Correct greeting: approach dog backwards or from side in a kneeling position and offer treat by slowly extending forearm out to the dog. Allow the dog to approach instead of cornering it. (Photo used by permission from Yin S: *Low stress handling, restraint and behavior modification of dogs and cats,* Davis, Calif, 2009, Cattle Dog Publishing.)

the staff member greets and gets a history from the owner. The dog is less fearful if encouraged with treats and praise to voluntarily approach the unfamiliar person. The dog may feel trapped and defensive if the unfamiliar person invades its personal space. After the animal is in the therapy area, staff members can continue to make the visit enjoyable by speaking to the dog in an upbeat voice and luring it around the facility with small food treats.

Animal facilities have an abundance of smooth, cleanable surfaces that can amplify sound waves. Barking in dogs can easily reach the 90-decibel limit of the Occupational Safety and Health Act.[27] Human ears can be protected with safety devices, but the welfare of our patients is still compromised if noise is not reduced. An architect familiar with noise-reducing design and building materials should be consulted for new facility construction.[28] Although retrofitting existing facilities to reduce noise and visual contact can be more challenging, cleanable but sound-absorbing rubber and certain fiberglass panels can be used in most areas.

Classical music may promote quiet and resting behavior, whereas heavy metal music seems to agitate dogs.[29] Barking and other anxiety-related behaviors may also be decreased by occupying the dog with treat-dispensing objects (e.g., Kong toys* or Premier Pet Products Busy Buddy line of toys†) filled with palatable food. Decreasing the motivation to bark by alleviating underlying distress is preferable to aversive methods that may increase anxiety, but persistent barking by just one or two dogs can increase the stress level of all other patients and staff. A spray (citronella or scentless) bark collar for specific offenders may be helpful in these situations.[30]

Pheromones are another simple modality that may be used to reduce stress in kennel and therapy areas.[31] Feliway is a synthetic version of the feline facial pheromone. The diffuser can be used in wards or treatment rooms, and the spray form can be applied to towels or hands several minutes before the procedure. Adaptil‡ is a synthetic version of a lactating bitch mammary pheromone and is also available as a diffuser or spray. The spray reapplied every few hours to a towel or bandana could potentially provide better pheromone concentration and efficacy for that individual dog or cat. Collars that are impregnated with pheromones and claim to emit the chemical for approximately 1 month are also available (Adaptil‡ and Nurturecalm§).

Finally, nonslip flooring and substrates are essential to creating a relaxed patient. A dog that feels unsteady is more likely to panic and injure itself or staff during restraint, and become an uncooperative participant. Thick rubber mats afford both stable footing and cushioning of pressure points that may become uncomfortable during recumbent procedures. Small dogs and cats should be placed on a soft but secure towel on the examination table.

Humane Control and Safety Devices

Appropriate control over the dog helps to prevent unruly behavior, such as pulling, jumping, or aggressive lunging. Head halters are an excellent tool and are underused in the clinic setting. There are several styles and brands of head halters with slightly different designs, but all have two loops—one behind the ears and one around the nose. With most designs the leash attaches under the jaw. This provides better control because the dog's pulling away from the handler puts pressure on the nose strap and pulls the nose back to the handler. Although the pressure is aversive, it is safe and not painful when applied correctly. The owner should be encouraged to acclimate the dog to the head halter at home by associating it with treats and other enjoyable activities, like walks (Figure 4-6). However, this can usually be done in just a few minutes at the clinic. Head halters can be extremely useful during the session. A dog that becomes distressed will often attempt to escape from the situation. The handler may, in turn, react by speaking sharply to the dog or taking a "death grip" on the patient. This reaction may suppress the struggle in that moment but it will only increase the patient's fear and make subsequent interactions more difficult. Some dogs may appear calmer when simply wearing a properly fitted head halter. If a dog does become overly aroused, judicious use of pressure and release of pressure from the nose loop can quickly calm the dog by preventing escape and allowing the handler to remain calm and quiet.

A head halter can prevent low levels of aggression, but dogs with a biting history may require a muzzle. A basket muzzle is preferred in most situations. A basket muzzle allows the dog to take treats and pant, whereas a sleeve muzzle can exacerbate distress by restricting panting and breathing. Although there may be no obvious difference for brief examinations or events like a venipuncture, extended procedures are more common in physical rehabilitation, thus making basket muzzles more desirable. As with the head halters, animals will accept the muzzle more willingly if a desensitization program is outlined for the owners and performed before it is used in the session. In short, place spreadable food such as peanut butter, canned cheese, or meat-flavored dog pastes (e.g., Kong liver flavor paste) into the muzzle. Encourage, but do not force the dog to put its nose in the muzzle for the treats (see Figure 4-6, *A*). When the dog does not hesitate to put its nose in the muzzle and keep it there, the strap behind the ears can be fastened. The time the dog wears the muzzle can be gradually increased by a few seconds as long as it

*Kong Rubber Toys, Kong Company, Golden, Colorado.
†Busy Buddy Toys, Premier Pet Products, Midlothian, Virginia.
‡Adaptil, Ceva Sante Animale, Rutherford, New Jersey.
§Nurturecalm 24/7 Pheromone Collar, Meridian Animal Health, Omaha, Nebraska.

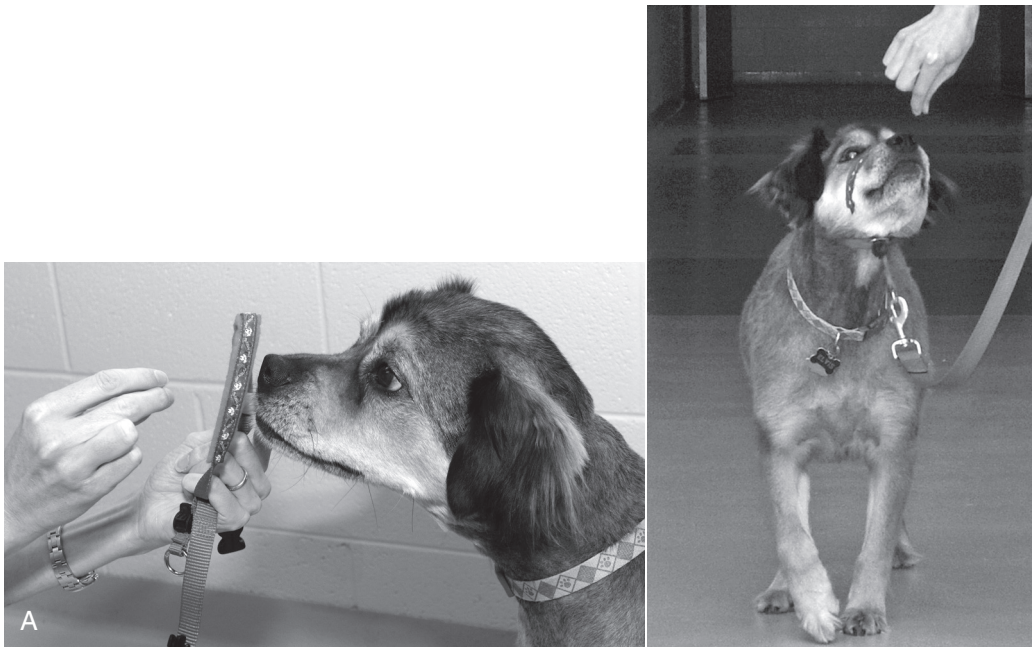


Figure 4-6 **A,** Acclimate the dog to the head halter by initially rewarding with treats when he puts his nose through the loop. **B,** Continue to associate the head halter with rewards by treating the dog for calmly walking by your side.

is relaxed. Continue to provide the treats while the dog is wearing the muzzle.

Handling of the Physical Rehabilitation Patient

Physical therapists and their staff are encouraged to read, watch videos, and practice handling techniques that are extensively detailed in other publications to supplement the material in this chapter. In *Low stress handling, restraint, and behavior modification of dogs and cats,*[18] Yin outlines 10 basic principles of low-stress handling, including proper body support, decreasing fear to reduce patient struggling, and minimizing the duration of restraint. Most importantly, handlers should become highly skilled at recognizing subtle signs of fear and distress in pets. When fear is first detected, handling strategies can then be adjusted, allowing the animal to remain relaxed and avoid extreme escape behaviors and aggression.

Choke chains and slip leads should be avoided. As discussed previously, head halters provide the most humane control. Treats and the head halter can be used to lure the dog from one position to another and minimize the need for physical positioning. However, the use of hands and body to properly place the animal is usually necessary at some point during a therapy session. When moving a dog from one position to the next, motions should be slow and smooth, and the handler's body should be a solid support, not painfully forcing the patient into a certain position. Staff should become adept at towel-wrap restraints for cats



Figure 4-7 Secure and proper towel-wrap restraint can allow access to both the head and hind-end while decreasing the cat's stress and injury to the handler.

so uncomfortable scruffing and stretching can be avoided (Figure 4-7). The tight wraps provide security and ample access to the needed area while also decreasing agitating sensory overstimulation.[18]

Figures 4-8 and 4-9 show how the head halter can be used to keep the dog focused on the reward during a procedure. Note the point of control is on the dog's head. It would be difficult for him to turn his mouth far enough to bite; however, the body restraint is minimal so any discomfort from the ultrasound may be easily detected. Figure 4-8 is appropriate for a dog with a history of aggression. The leash is kept between the dog and the wall, preventing the mouth from reaching the handler and therapist. An Elizabethan collar or rolled towel wrapped around the neck can also be used to block bites, particularly in cats and



Figure 4-8 The handler uses the head halter and treats to distract and control the dog during a procedure. This low-restraint method keeps the dog calm and also prevents the dog from turning to bite the handler or therapist. However, a basket muzzle can be placed over the head halter for added safety.



Figure 4-9 Restraint for examining the hind limbs or back of an aggressive dog. The dog is placed against a wall and one handler stands close to the dog's shoulder. This handler keeps the leash of the head halter between the dog and the wall, thus preventing the dog from turning to bite. Another handler is offering treats during the examination or procedure to encourage the dog to remain stationary. A basket muzzle may be placed over the head halter.

brachycephalic (very short muzzled) dogs for which a head collar can be difficult to fit. Also note that treats are given liberally throughout the procedure. Handlers often wait until after the procedure to reward the dog for good behavior. As discussed in Learning and Behavior Modification earlier in this chapter, the action the animal is performing within 1 second of receiving the reward or punishment is the behavior being operantly conditioned. Thus a treat and verbal praise given as the procedure is ending and restraint is removed is rewarding the pet for moving away from the handler. Although the procedure room and staff are possibly being associated with a pleasant emotion through classical conditioning, remaining calm and still *during* the procedure is not being reinforced.



Figure 4-10 Luring a dog into the water treadmill using a previously conditioned hand-targeting command. The dog mastered targeting in quiet areas and then near the treadmill before he was lured into the apparatus.

Tasty treats or other motivators like a favorite toy or person can be used to lure a hesitant dog onto apparatuses like inclines or treadmills. Asking the animal to perform a command for the reward often improves compliance by focusing the dog on an alternate activity. A correctly performed counterconditioning with systematic desensitization may take more time to complete during the initial training phase, but it is a time saver, as well as being more humane, over the long term. Figure 4-10 demonstrates a dog being lured into the water treadmill using a hand target command. The dog was previously taught to touch the hand with his nose when giving the visual cue of the hand and the verbal command "touch." This dog is nervous about approaching the treadmill. The targeting and other commands are initially asked at a great distance from the apparatus. The handler and dog close the distance to the treadmill by a foot or two when the dog appears relaxed and focused on the handler. If the apparatus has a potentially frightening sound, the first phase of the desensitization should be the gradual approach with the machine off and quiet. The next stage is repeating the session with the motor turned on. The dog should not be forced into approaching. A dog too frightened, tired, or unmotivated to ambulate on the treadmill is not receiving the full benefit of the exercise and may injure itself. Verbally praising the dog and luring with the target and treats increase the dog's motivation and classically condition the patient to the treadmill with an enjoyable emotion. This procedure can be used for a myriad of active exercises such as incline walking, swimming, dancing, and cart exercises. Target training and desensitization, along with good control using a head halter, can also successfully condition the pet to participate willingly in other exercises, such as the balance platform and exercise ball, both of which can be disconcerting because of the imbalance they create.

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5 Canine Anatomy

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This text is intended for people who already possess knowledge of either veterinary or human anatomy. To assist communication among human rehabilitation and veterinary colleagues, some anatomic terms used for dogs appear in regular print with the analogous terminology for humans in parentheses following the canine term. These comparisons have been minimized, as this is a chapter about canine anatomy and not a chapter about comparative anatomy. Comparative anatomy between dogs and humans has been described in other sources.[1-3]

We have chosen to use some terms consistently throughout the chapter, rather than use equally acceptable synonyms. The canine forelimb is known also as the *thoracic limb* and the *pectoral limb,* but we use the term *forelimb.* The canine hindlimb is known also as the *pelvic limb* or *rear limb,* but we use the term *hindlimb.* Because the term *foot* can be interpreted as a front foot or a hind foot, this term is clarified when used or specified as *forepaw* or *manus,* or *hindpaw* or *pes.* The terms *trunk, neck,* and *head* refer to the same body segments in dogs and humans. The word *canine* is an adjective and the word *dog* is a noun; these terms are used in this consistent grammatical form throughout the chapter.

Directional Terms and Anatomic Planes

Directional Terms from Normal Stance (Anatomic Position)

The dog stands upright on digits or phalanges of each forepaw or manus and each hindpaw or pes (Figure 5-1). This type of stance is termed a *digitigrade stance.* The human stands upright on the feet, with the plantar aspect of the feet contacting the floor and adjacent to each other. The upper limbs hang at the sides of the body, palms facing forward. This type of stance is called a *plantigrade stance.*

Directional terms from anatomic position in dogs are more directly compared with the directional terms in humans when the human is in a quadruped position or the dog is in an upright stance posture. Directional terms include *cranial, caudal, rostral, dorsal, palmar, plantar, medial,* and *lateral.* Other specific directional terms include (1) radial and ulnar to indicate toward the radius and ulna, respectively; (2) axial and abaxial to indicate toward or away from the axis of the digits, which is between the third and fourth digits of the forepaw, and the third and fourth digits of the hind paw, respectively; and (3) tibial and fibular to indicate toward the tibia and fibula, respectively.

Anatomic Planes

The main planes of motion for dogs are as follows (see Figure 5-1):

- The *sagittal plane* divides the dog into right and left portions. If this plane were in the midline of the body, this is the median plane or median sagittal plane.
- The *dorsal plane* divides the dog into ventral and dorsal portions.
- The *transverse plane* divides the body into cranial and caudal portions.

Axes of Rotation

Motion may occur in any of three planes of motion or some combination. Joint motion within a plane usually occurs around an axis of rotation, which may be centered within the joint space or within the bone comprising the joint. Some joint motions are planar or gliding motions and do not occur around an axis of rotation.

An axis of rotation for a joint motion is a straight line or rod that is 90 degrees to the plane of motion. For each axis of rotation listed in the next section, the plane of motion around which joint motion occurs can be viewed from Figure 5-1.

Axes of Rotational Joint Motion

The axes of rotational joint motion are as follows:

- *Transverse axis:* Sagittal plane motion occurs around an axis of rotation that is directed mediolaterally.
- *Ventrodorsal axis:* Dorsal plane motion occurs around an axis of rotation that is directed ventrodorsally.
- *Craniocaudal axis:* Transverse plane motion, such as rotation of the trunk, occurs around an axis of rotation that is directed craniocaudally.

Most joints allow motion in more than one plane. Dogs and humans have the ability to selectively produce motion in one, some, or all of the planes of motion at one time.

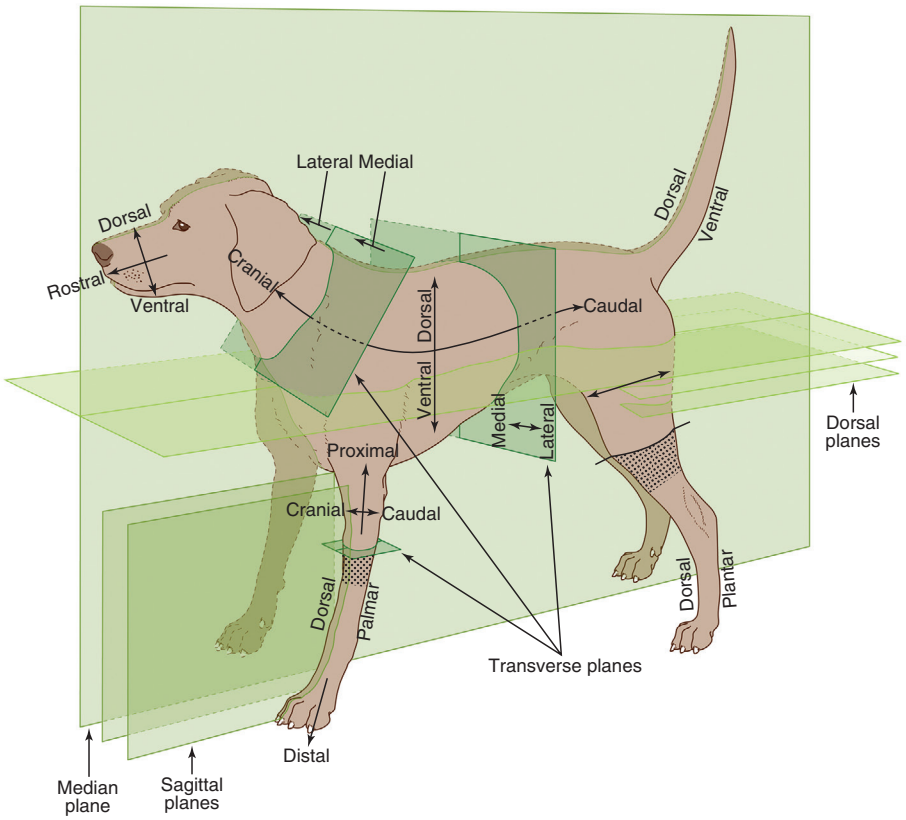


Figure 5-1 Orientation to planes of motion and directional terms for the dog. (From Dyce KM: *Textbook of veterinary anatomy,* ed 4, St Louis, 2010, Saunders.)

Dogs have much more limitation in motion in the dorsal and transverse planes.

Bones

The bones of the dog skeleton and limbs are illustrated in Figures 5-2, 5-3, and 5-4. Bony landmarks on the bones of the limbs are shown in Figures 5-5 through 5-10.

Forelimb

The forelimb skeleton consists of the thoracic or pectoral girdle and bones of the forelimb (see Figures 5-5 and 5-6). The size of forelimb bones varies a great deal, because of the greater variation in size for breeds of dogs. The forelimbs bear 60% of the dog's weight.

The canine scapula is positioned close to the sagittal plane. Dogs have an abbreviated clavicle that does not articulate with the rest of the skeleton. It is a small oval plate often 1 cm or less in length and ⅓ cm wide, located at the tendinous intersection of the brachiocephalicus muscle. The adult canine clavicle is mostly cartilage and is usually not visible on radiographs.

The canine humeral head is less rounded compared with the human head, to assist with weight bearing. Distally, there is an olecranon fossa and supratrochlear foramen for the secure positioning of the protruding anconeal process of the ulna for more stability in weight bearing.

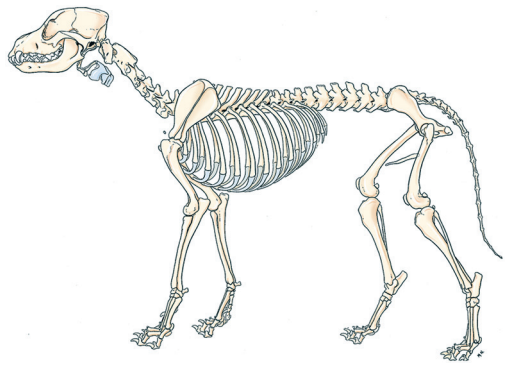


Figure 5-2 Skeleton of a male dog, left lateral view. (From Evans HE: *Miller's anatomy of the dog,* ed 4, Philadelphia, 2013, WB Saunders.)

The radius is the medial forearm bone and is the main weight-bearing bone of the antebrachium distally. The proximal surface of the radius articulates with the humeral capitulum, which is not as prominent as in the human. The canine distal radius has distinct facets for articulation with carpal bones, providing stability in weight bearing.

The ulna is the lateral forearm bone and has a very prominent olecranon process, which allows secure attachment for the large triceps brachii muscle, needed as an antigravity muscle for weight bearing in dogs. The ulna is the longest bone of the canine body. It articulates distally with the ulnar carpal and accessory carpal bones by two distal facets and does not have an articular disk. The dog has an anconeal process, which is near the attachment site of the anconeus muscle. The anconeal process is needed for stability in weight bearing.

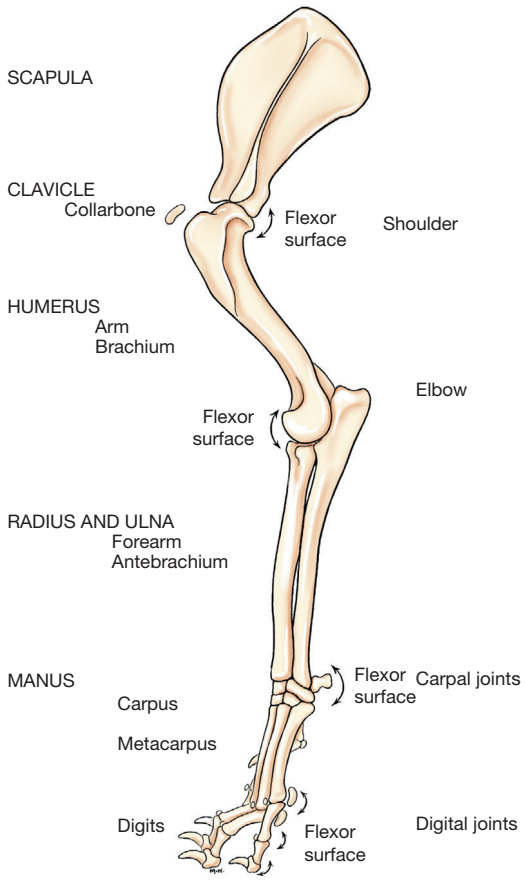


Figure 5-3 Left forelimb skeleton, noting joints and flexor surfaces. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

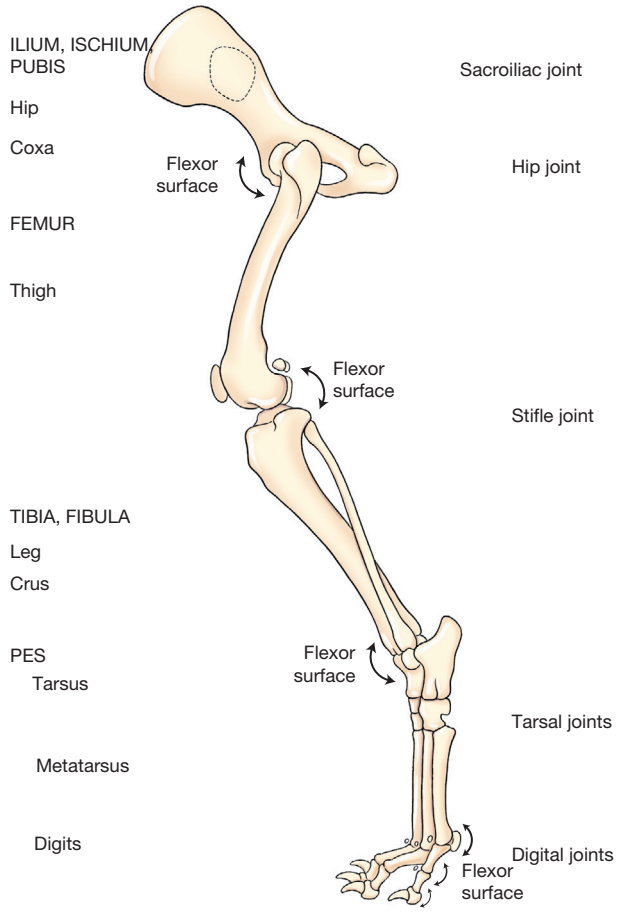


Figure 5-4 Left hindlimb skeleton, noting joints and flexor surfaces. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

At the carpus or wrist (see Figure 5-7), there are seven carpal bones. The radial carpal bone is analogous to the fused scaphoid and lunate. There are five metacarpal bones. The first metacarpal is short and nonfunctional. Dogs have many sesamoid bones that are embedded in tendons or near them. Sesamoid bones occur when there are significant changes in directions of pull on tendons in addition to the tensile forces produced during muscle contractions. They allow for constant, biomechanically advantageous alignment of angles of insertion of tendons at their attachment sites, which helps relieve stress on the tendinous insertions for animals that walk on their digits. Dogs are digitigrade animals and bear weight on digits II to V, with the main weight bearing occurring on digits III and IV. The sesamoid bones at the dorsal surface of each metacarpophalangeal joint align the extensor tendons for optimal muscle action. Those on the pad surface of the manus align the flexor tendons.

Hindlimb

The hindlimb skeleton includes the pelvic girdle, consisting of the fused ilium, ischium, and pubis, and the bones of the hindlimb (see Figures 5-8 and 5-9). The size of hindlimb bones varies a great deal, because of the great variation in size for breeds of dogs. The hindlimbs bear 40% of the dog's weight.

The canine pelvis is positioned between the dorsal and transverse planes and closer to the dorsal plane. The canine pelvis is relatively small and narrow. The canine ischiatic or ischial tuberosities are wide and project caudally to form a broad ischiatic table. The canine pelvis shape from a ventral view resembles a rectangle. The symphysis pelvis is relatively long and has two portions, the symphysis ischii and symphysis pubis, compared with the relatively shorter joining of the anterior aspect of the human innominates at the symphysis pubis. The distinction of the shape of the male and female pelvic inlet and outlet in humans is not made in dogs.

The canine femur is the heaviest[4] and largest[5] canine bone. In most dogs, it is slightly shorter than the tibia and the ulna and approximately one-fifth longer than the humerus. The average canine angle of inclination or cervicofemoral angle is 144.7 degrees.[5] Dogs have an average

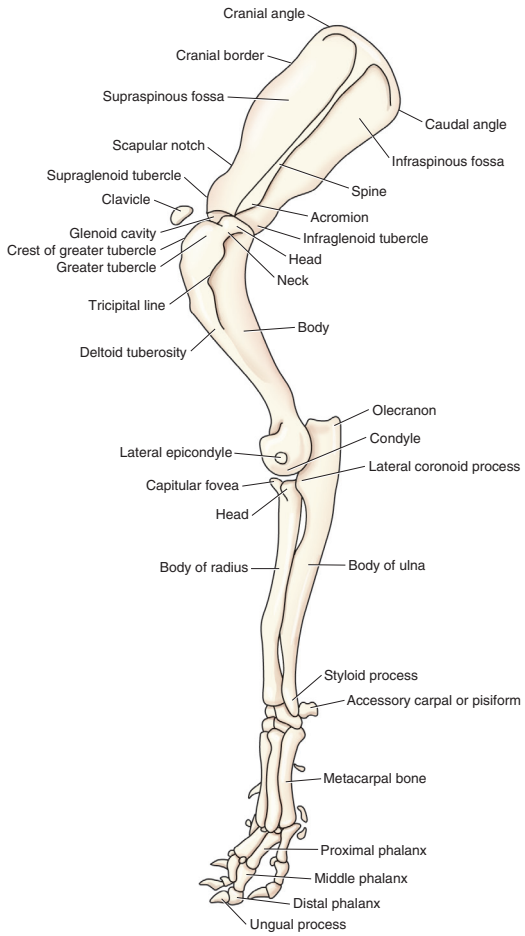


Figure 5-5 Skeleton of the lateral forelimb of the dog. (Adapted from Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

degree of anteversion or positive femoral torsion of +27 to 31 degrees, when measured from a direct radiograph or with a method using trigonometry and biplanar radiography, respectively.[5] The canine femur has a relatively thick and short femoral neck, a caudomedially located lesser trochanter, a prominent lateral greater trochanter, and a relatively short and wide shaft with a narrow isthmus in the middle. The greater trochanter has a craniolateral prominence called the *cervical tubercle.* Dogs have a third trochanter, which is the attachment site of the superficial gluteal muscle. Canine medial and lateral femoral condyles are equally prominent, but the articular surface of the medial femoral condyle projects more cranially than that of the lateral femoral condyle.

There are three sesamoid bones in the caudal stifle joint region. Two are located in the heads of the gastrocnemius muscle caudal to the stifle joint and are called *fabellae.* The sesamoid in the lateral head is the largest, is palpable, and articulates with the lateral femoral condyle, whereas the one in the medial head is smaller and may not have a distinct facet on the medial femoral condyle. The third is the smallest, is located in the proximal attachment of the popliteus muscle, and articulates with the lateral tibial condyle.

The canine patella, or kneecap, is the largest sesamoid bone in the body. It is an ossification in the quadriceps femoris muscle. The patella alters the pull, increases the moment arm, and protects the quadriceps tendon, as well as provides a greater contact surface for the tendon on the trochlea of the femur than would exist without the patella. The canine patellar articular surface is mildly convex.

The canine tibia is the major bone in the crus. The triangular proximal tibia is wider than the distal cylindrical tibia. Medial and lateral tibial condyles, an intercondylar eminence, and a tibial tuberosity are on the proximal tibia. The tibial plateau slopes distally from cranial to caudal. The extensor groove, on the cranial tibia and lateral to the tibial tuberosity, provides a pathway for the long digital extensor muscle. There is a popliteal notch on the caudal tibia in the midline, where the popliteal vessels course. The tibia articulates with the fibula proximally, along the interosseous crest, and distally. The tibial cochlea articulate with the trochlea of the talus to form the talocrural joint.

The canine fibula is a long, slender bone that articulates with the tibia and also serves as a site for muscle attachment. There is a distinctive groove in the lateral malleolus, the sulcus malleolaris lateralis, through which course the tendons of the lateral digital extensor and peroneus brevis muscles.

The tarsus, or hock, consists of the talus, calcaneus, a central tarsal bone, and tarsal bones I to IV (see Figure 5-10). The talus articulates with the distal tibia and has prominent ridges. At the talocrural joint, two convex ridges of the trochlea of the talus articulate with two reciprocal concave grooves of the cochlea of the tibia. The orientation of the grooves and ridges deviates laterally approximately 25 degrees from the sagittal plane. This deviation allows the hindpaws to pass lateral to the forepaws when dogs gallop.[4] The calcaneus is large and serves as the insertion of the common calcaneal tendon. The central tarsal bone lies between the talus and the numbered tarsal bones I to III. Tarsal IV is large and articulates with the calcaneus and metatarsal bones, spanning this entire region.

The canine hindpaw has five metatarsal bones; however, the first metatarsal can be short or absent. Dogs have many sesamoid bones that are embedded in tendons where there are significant compressive and tensile forces produced during muscle contractions. The sesamoid bones at the dorsal surface of each metatarsophalangeal joint align the extensor tendons for optimal joint action. The sesamoid bones on the plantar surface of the hindpaw align flexor tendons.

Spine

The spine consists of five areas of the vertebral column: the cervical vertebrae and its articulation with the head,

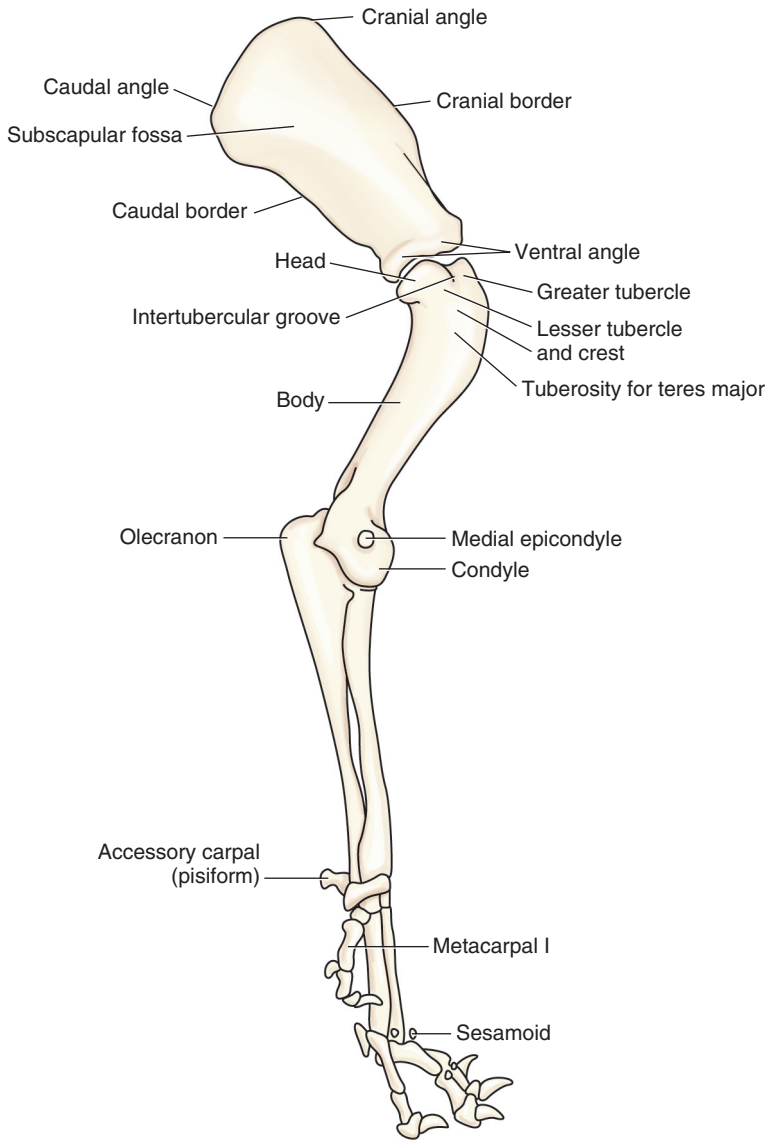


Figure 5-6 Skeleton of the medial forelimb of the dog. (Adapted from Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

thoracic vertebrae, lumbar vertebrae, sacral vertebrae, and the coccygeal vertebrae (Figures 5-11 through 5-14). The number of vertebrae is listed in Box 5-1.

All vertebrae, except the sacral vertebrae, remain separate and form individual joints. Four sites with limited motion exist within the canine spine.[6] These sites occur at areas where the cranial and caudal articular surfaces are inclined in a nonparallel manner and in different directions. The nonparallel alignment of the articular surfaces markedly restricts joint accessory motions, such as glides. The restricted joint motions and areas resulting from these joint alignments include atlantoaxial motion other than rotation, the cervical (C) 7-thoracic (T) 1 junction, the caudal thoracic region, and the sacrum.

Individual vertebral bone size and shape vary among breeds. For any one breed, canine cervical through lumbar vertebrae are fairly consistent in size. The consistent size in dogs reflects the relatively equivalent cranial-to-caudal compressive loading. Because dogs are quadruped, there is weight bearing on all four limbs. There is cervical spine compression as a result of the positioning of the dog's head as a cantilever, which requires cervical extensor muscle activity to maintain head posture. The massive cervical extensor muscle activity requires relatively large and strong cervical vertebrae to support the muscle mass. Canine intervertebral disks likewise change little in size from the cervical through the lumbar vertebrae. The C5-C6 area is a site of relative hypermobility in large dogs. The spinal cord ends at lumbar (L) L6-L7.

The canine atlas, or C1 vertebra (see Figure 5-12), has a transverse foramen in each transverse process, a craniodorsal arch, and right and left lateral vertebral foramina

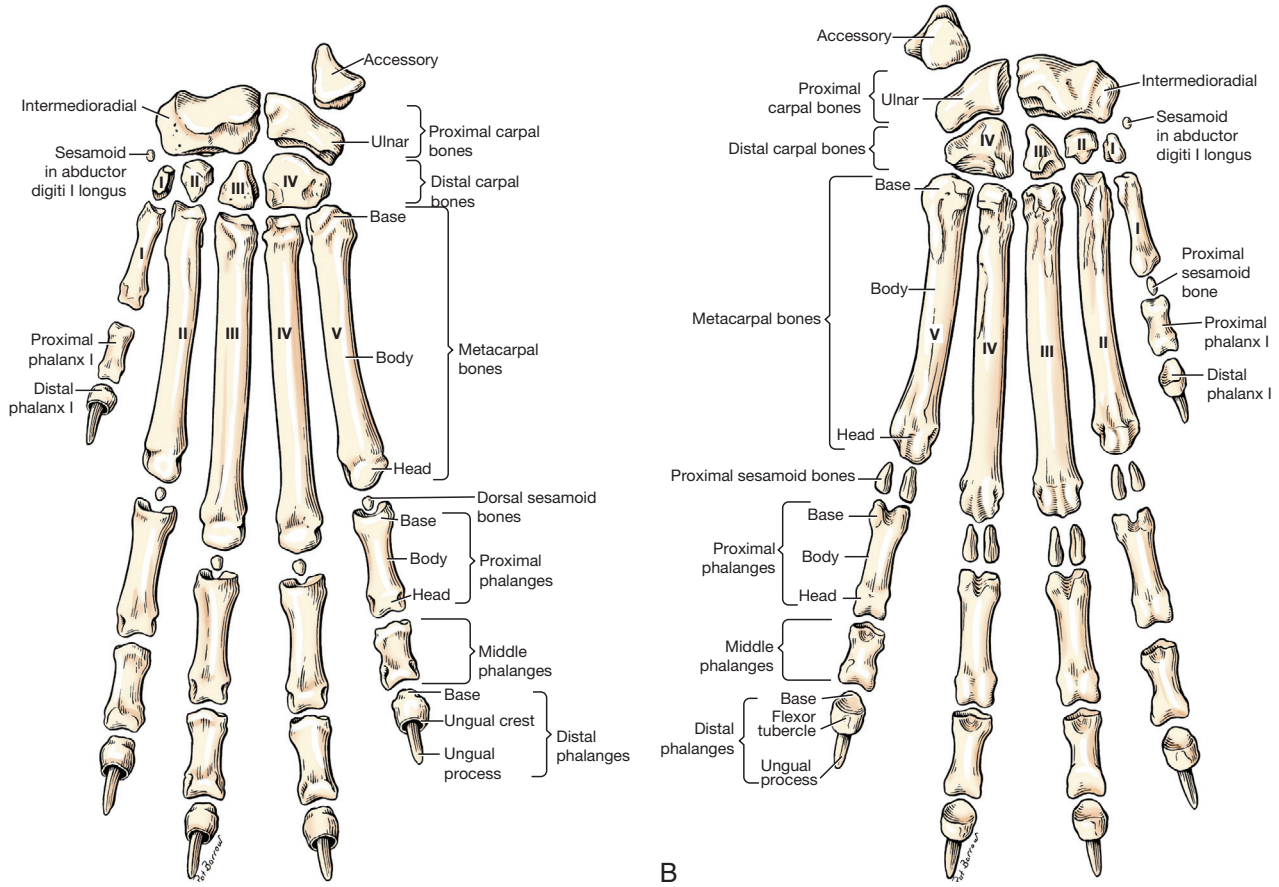


Figure 5-7 Skeleton of the left dorsal (*A*) and left palmar (*B*) forepaw of the dog. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

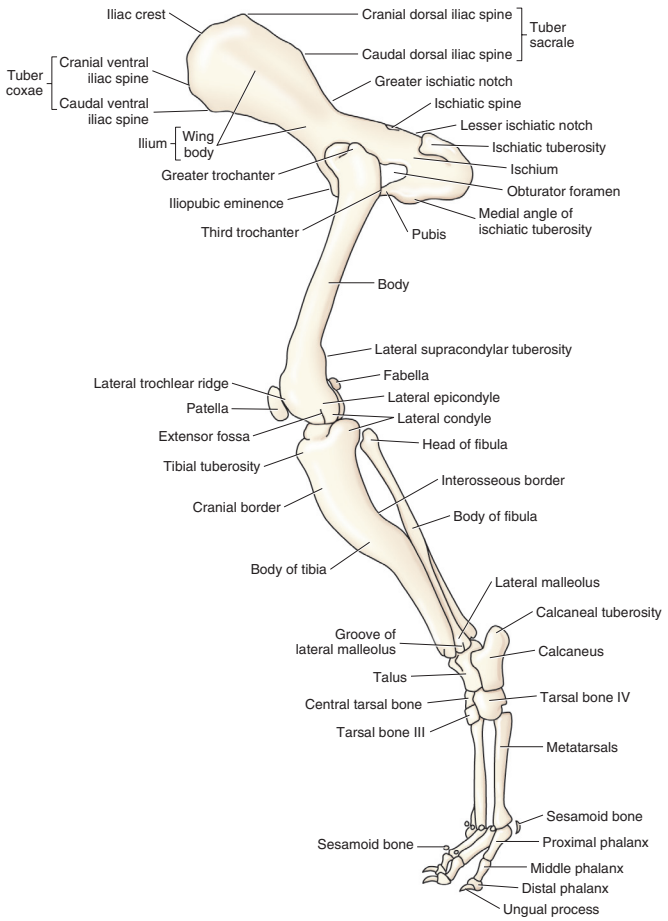


Figure 5-8 Skeleton of the lateral hindlimb of the dog. (Adapted from Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

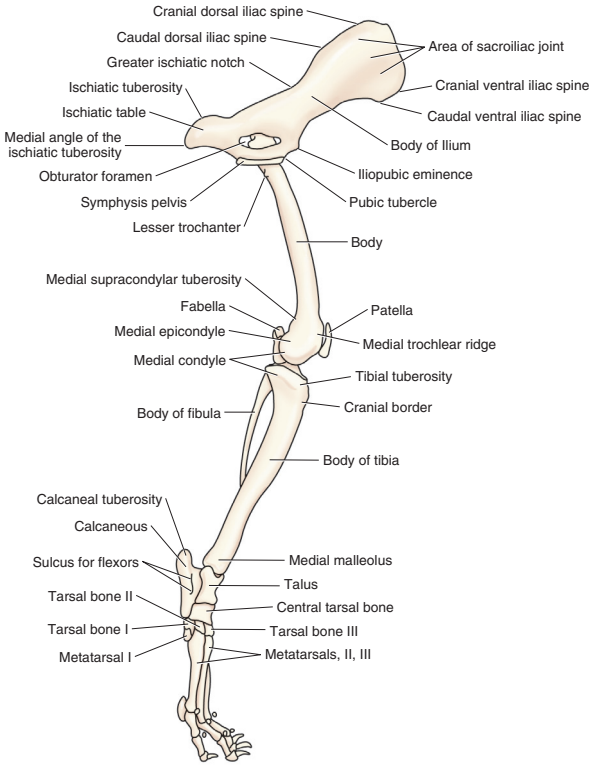


Figure 5-9 Skeleton of the medial hindlimb of the dog. (Adapted from Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

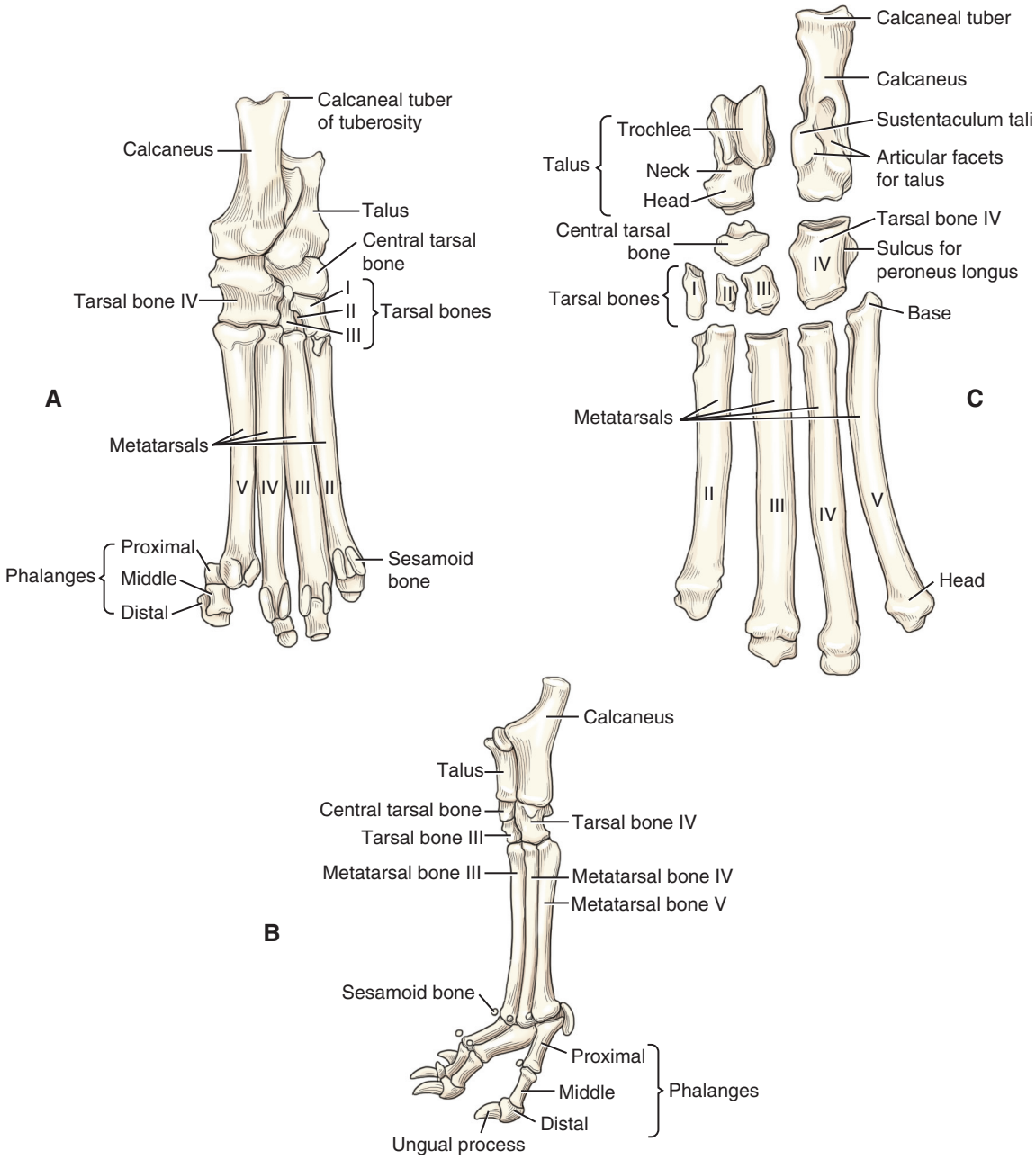


Figure 5-10 Skeleton of the left plantar (*A*), left lateral (*B*), and left dorsal (*C*) hindpaw of the dog.

for the passage of cervical spinal nerve 1. The atlas has correspondingly shaped condyles for articulation with the occiput. The canine lateral wings or transverse processes are prominent and easily palpable from the skin surface. The canine axis or C2 has a large spinous process with an expanded arch, a wide body, and large transverse processes (see Figure 5-12). The spinous process is nonbifid. The canine axis is very large relative to the size of other canine cervical vertebrae. The axis has a dens, which projects cranially to allow pivotal motion between the atlas and axis. The condyles are oriented near the transverse plane to allow cervical spine rotation. The C3-C6 vertebrae have nonbifid spinous processes, large and flat spinous processes, caudal and cranial articular surface facets that are narrower than the transverse processes, large transverse processes, and transverse foramina for the passage of vertebral arteries. Caudal and cranial articular surfaces are oriented between the dorsal and transverse planes to facilitate cranial and caudal glides needed for cervical spine flexion and extension. The C7 vertebra has a similar shape, a large prominent nonbifid spinous process, and caudal and cranial articular surfaces, which are oriented nearly craniocaudally.

Thoracic vertebrae (see Figure 5-13) have small bodies relative to the size of the entire vertebrae. Canine spinous

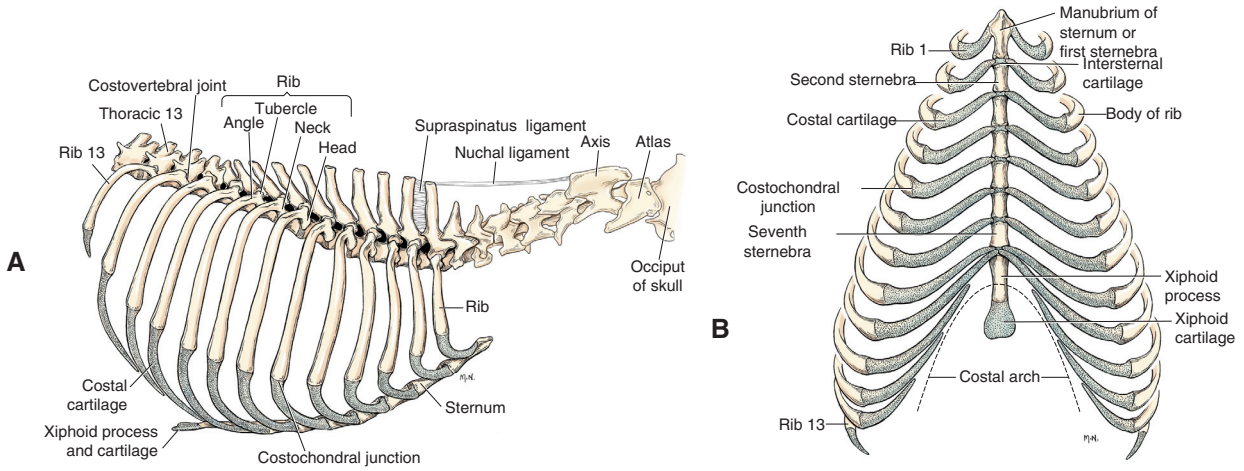


Figure 5-11 **A,** Identified portions of the axial skeleton cranial to the thirteenth thoracic vertebra. **B,** Ribs and sternum, ventral view.

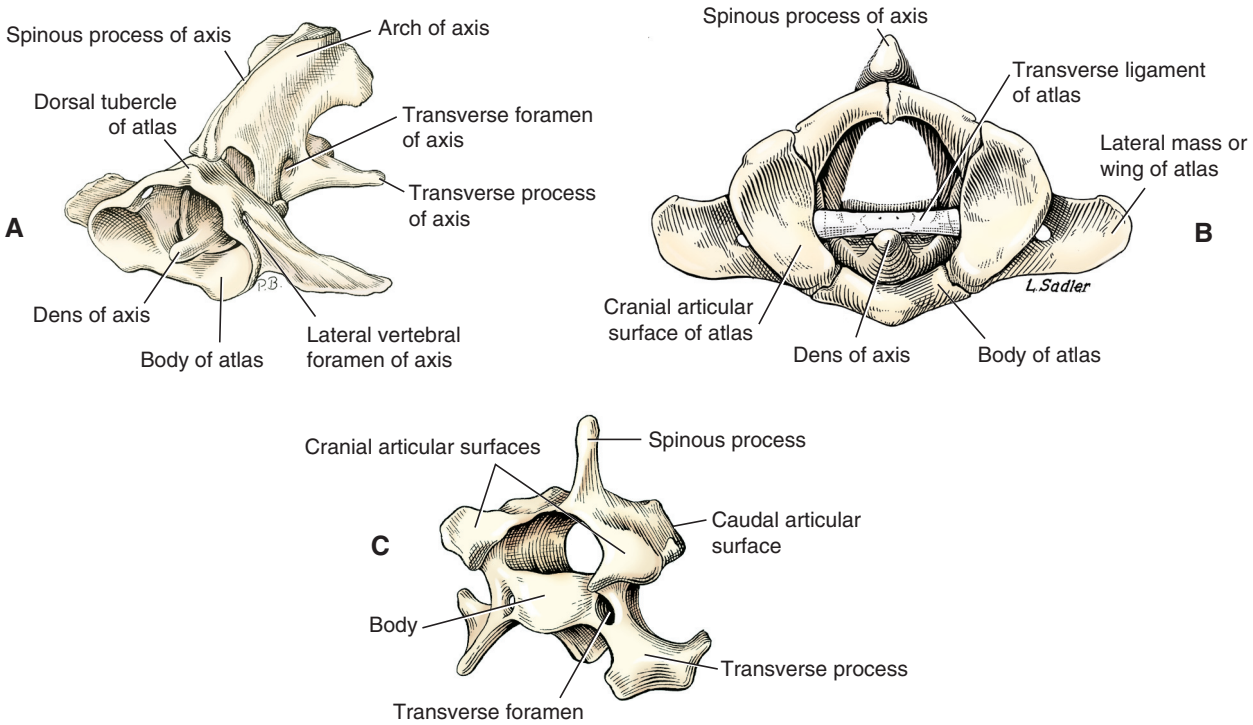


Figure 5-12 Detailed skeletal anatomy of the atlas and axis from a craniolateral view (*A*), atlas and axis from a cranial view (*B*), and C5 vertebra from a craniolateral view (*C*). (**A** from Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, St Louis, 2010, WB Saunders.)

processes are relatively long. The spinous processes block excessive extension of the thoracic spine. At T10, the size of the body begins to increase and the length of spinous process decreases. The spinous processes are oriented close to the transverse plane. Cranial to T11, the spinous processes project caudally, but caudal to T11, they project cranially. Caudal and cranial articular surfaces are oriented close to the dorsal plane.

Lumbar vertebrae (see Figure 5-13) have bodies that are larger than thoracic vertebral bodies. Canine lumbar transverse processes are long and thin, and they project lateroventrocranially. In the cranial lumbar spine, cranial and caudal articular surfaces are oriented between the transverse and sagittal planes, which facilitate lumbar spine flexion and extension. The L7-S1 joint appears to orient between the sagittal and frontal planes to allow more rotation at this intervertebral level. The canine sacrum is relatively narrow and is linked to the pelvis with sacroiliac joints (see Figure 5-14).

Caudal (Cd) vertebrae (see Figure 5-14) have distinct bodies and transverse processes. The cranial articular surfaces are similar to those in more cranial vertebrae in

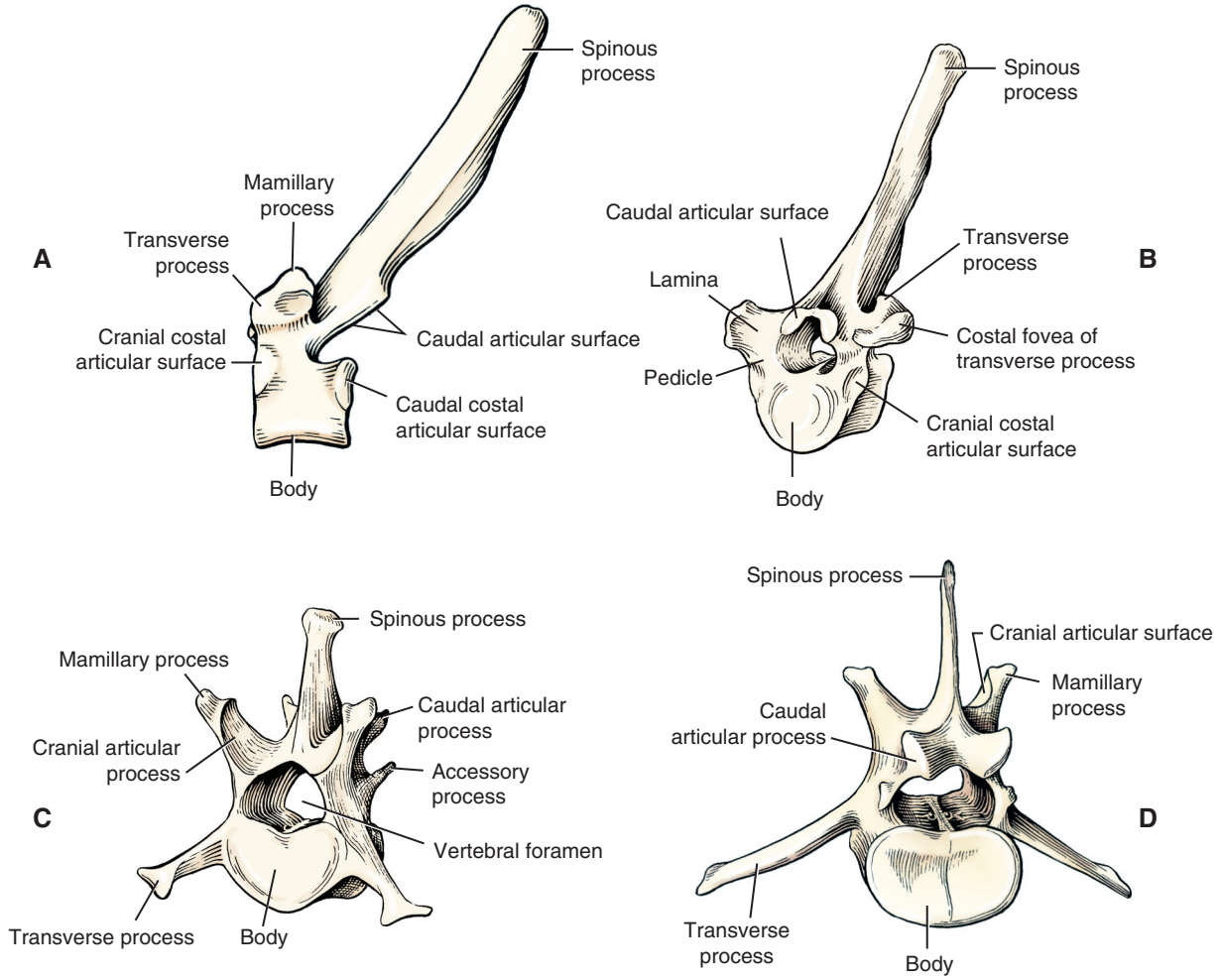


Figure 5-13 Detailed skeletal anatomy of T6 vertebra from a lateral view (*A*) and craniolateral view (*B*), L1 vertebra from a craniolateral view (*C*), and L5 vertebra from a caudolateral view (*D*).

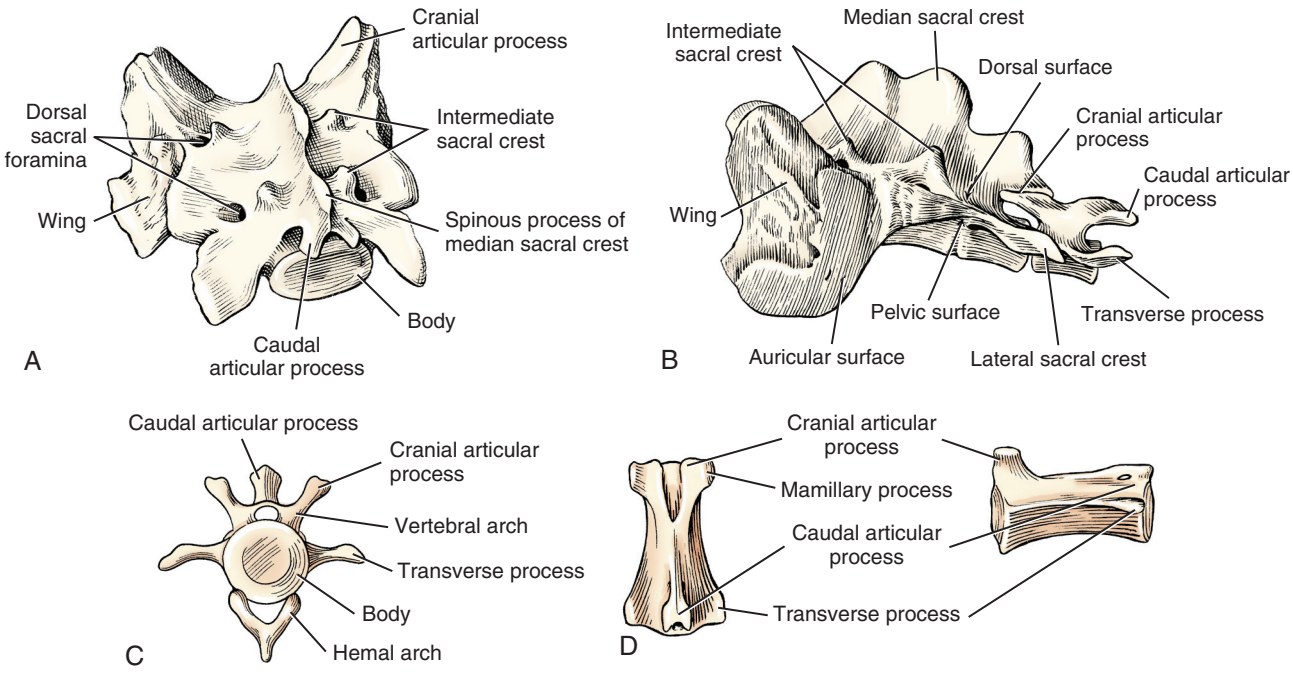


Figure 5-14 Detailed skeletal anatomy of the sacrum from a caudolateral view (*A*), sacrum and caudal 1 or Cd1 vertebra from a lateral view (*B*), Cd4 vertebra from a cranial view (*C*), and Cd6 vertebra from a dorsal view (*D*). (From Evans HE: *Miller's anatomy of the dog,* ed 4, Philadelphia, 2013, WB Saunders.)

Box 5-1 Body Segments

Forelimb

- Forelimb: Arm, forearm, and forepaw
- Thoracic or pectoral girdle
  Scapula, clavicle
- Arm or brachium: Shoulder to elbow
- Forearm or antebrachium: Elbow to carpal joint
- Forepaw or manus
  Carpus or carpals
  Metacarpus or metacarpals
  Phalanges or digits
- Sesamoid bones or cartilages
  Dorsal on MCP joints in common digital extensor tendons of digits II to V; one per digit; small
  Pad surface on MCP joints in interosseous tendons of digits II to V; two per digit; smaller
  Dorsal and palmar on DIP joints of digits I to V; cartilage; small
- One sesamoid bone in the tendon of the abductor pollicis longus
- Digits or phalanges I to V, numbered medial to lateral
  No common names for digits
  Anatomic name: pollex for digit I
- Dewclaw or pollex or digit I with 2 phalanges
- Pads on the paws or digital pads: Weight-bearing pads
  Carpal pad: Small pad palmar to the carpus
  Metacarpal pad: Largest pad palmar to the MCP joints; triangular in shape
  Digital pads: Palmar to the DIP joints; ovoid and flat
- Ungual process: Extension of the phalanx into the claw
- Nails or claws

Hindlimb

- Hindlimb pelvic limb, or rear limb
  Thigh, leg, hindpaw
- Hip bone or os coxae
  Ilium, ischium, pubis
- Pelvic girdle
  Right and left hip bones and sacrum
- Thigh: Hip to stifle or knee
- Leg or crus: Stifle to talocrural joint
- Hindpaw or hind foot or pes
  Tarsus or tarsals (hock area)
  Metatarsus or metatarsals
  Phalanges or digits or toes
- Sesamoid bones or cartilages
  Dorsal on MTP joints in long digital extensor tendons of digits II to V; one per digit; small
  Plantar surface on MTP joints in interosseous tendons of digits II to V; two per digit; large
  Digit I: One per digit, smaller
  Dorsal and plantar on DIP joints—cartilaginous; one per digit I to V; small
- Digits or phalanges or toes
  I to V
- Dewclaw or digit I or hallux—may be absent, fully developed and articulating with a metatarsal, or may be a vestigial, that is, a trace or rudimentary structure, with a terminal phalanx and no proximal phalanx or metatarsal bone
- Digital pads or pads on the hindpaws—weight-bearing pads
  Tarsal pad: Small pad plantar to the talocrural joint
  Metatarsal pad: Largest pad plantar to the MTP joints; triangular in shape
  Digital pads: Plantar to the DIP joints; ovoid and flat
- Ungual process: Extension of the distal phalanx into the nail
- Nails or claws

Spine

- Trunk
  Borders: Inguinal ligament to C7-T1 disk
- Neck or cervical spine
- Head
- Tail
- Spinal regions
  Cervical: C1 through C7
  Thoracic: T1 through T13
  Lumbar: L1 through L7
  Sacral: S1 through S3
  Caudal or coccygeal: Cd1-Cd20; some dogs have more or fewer
- Ribs: 13
- Bones in the dog skeleton (excludes auditory ossicles)
  Vertebral column: 50
  Skull: 49
  Hyoid bone: 1
  Ribs: 26
  Sternum: 8 fused bones—manubrium or first sternebra, 6 additional sternebrae, and the xiphoid process
  Forelimbs: 90
  Hindlimbs: 96
  Total: 320
  Other: os penis in males—1
- Hip bone or os coxae
  Ilium, ischium, pubis
- Sacrum
- Pelvic girdle: Right and left hip bones and sacrum
- Pelvic complex: Hip bones, lumbar spine, sacral spine, caudal spine, sacroiliac joints, and hip joints

C, Cervical; *Cd*, caudal; *DIP*, distal interphalangeal; *L*, lumbar; *MCP*, metacarpophalangeal; *MTP*, metatarsophalangeal; *S*, sacral; *T*, thoracic.

shape and location; however, the caudal articular processes are bifid and are more centrally located, whereas articular processes in more cranial vertebrae are located more laterally. Hemal arches are separate bones that articulate with the ventral surfaces of the caudal ends of the bodies of Cd4-Cd6. The hemal arches provide protection for the median coccygeal artery, which is enclosed by the arches. In vertebrae caudal to Cd6 and in relatively the same position as the hemal arches are the paired hemal processes, which extend from Cd7-Cd17 or Cd18.

The ribs have vertebral attachments (see Figure 5-11). There are nine pairs of vertebrosternal, or true, ribs and

four pairs of vertebrocostal, or false, ribs. The sternum is relatively long and has a manubrium and xiphoid process, with a prominent xiphoid cartilage. The ribs limit overall thoracic spine motion and protect internal organs.

Joint Motion

The body segments of the forelimb and hindlimb are illustrated in Figures 5-3 and 5-4, respectively, with the major joints and their flexor and extensor surfaces. Body segments are listed and defined in Box 5-1. Types of joints are listed in Box 5-2.

Joint Motion and Shape of Articular Surfaces

The shape of articular surfaces of bones helps define the motions available for a joint. Articular surfaces of two bones forming a joint are usually concave on one bone and convex on the other bone. Some articular surfaces are flat. Occasionally adjacent bones are convex on both joint surfaces. Intraarticular structures, such as the medial and lateral menisci in the stifle joint, may modify adjacent surfaces. Understanding the concave-convex relationships as a guiding principle in determining joint motion allows prediction of possible joint motions based on articular surface shape. Ligamentous and other soft tissue around the joint guide and restrict the motion that would be possible based on articular surface shape alone.

Joint motions are named, most commonly, by movement of the distal bone relative to the proximal bone. For example, cranial movement of the tibia on a stable femur is named *stifle joint extension.* The major direction of motion, such as flexion of the stifle, is physiologic or osteokinematic motion.

Accessory, or arthrokinematic, motion is smaller in magnitude and less observable. Examples of accessory motions are glide or slide, rotary motion, distraction or traction, and compression or approximation. A normal amount of glide occurs in normal functioning joints. Glides are shear type or sliding motions of opposing articular surfaces. Rolls involve one bone rolling on another. Gliding motion in combination with rolling is needed for normal physiologic joint motion. Spins are joint surface motions that result in continual contact of articular cartilage areas on opposite sides of a joint. Distraction or traction accessory motions are tensile or pulling-apart movements between bones. Compressive or approximation accessory motions are compressive or pushing-together movements between bones.

Normal joint motion involves both physiologic motion and accessory motion. Physiologic motion in joints with opposing concave and convex articular surfaces involves both roll and glide. Roll occurs in the same direction as the movement of the moving segment of the bone, but glide directions differ based on whether the moving articular surface is concave or convex. A glide is described by identifying the joint motion, the direction of the glide, and which bone is moving. For example, stifle flexion involving the tibia and femur is termed *caudal glide of the tibia on the femur.*

Joint Motion in the Limbs and Spine

Joint motions are named by one body segment approaching or moving away from another body segment or movement of some referenced body landmark. Joint motions are named in the following sections and described (see Figures 5-3 and 5-4) as they refer to the limbs, starting from normal stance. Limb motion is usually described by motion of the joint rather than a body segment. For example, elbow flexion is recommended rather than forearm flexion. Occasionally, body segment motion is used to describe limb motion when motion does not involve axial motion with a joint as a pivot point. For example, rotation of the forelimb might be observable when pronation at the radioulnar joint would be difficult to observe clinically.

Flexion

During flexion, a limb is retracted or folded, a digit is bent, and the back or neck is arched dorsally (i.e., the convex portion of the arch is directed dorsally). In the limbs, flexion motion occurs as the bones on either side of a joint move closer together and the joint angle becomes more acute. Flexion may also be referenced to limb motions involving closing angles during the swing phase of gait. Flexion motions of the limb joints are noted in Figures 5-3 and 5-4. In the spine, flexion occurs as the back or neck arches dorsally (i.e., the convex portion of the arch is directed dorsally).

A notable difference between dogs and humans is the meaning of *shoulder flexion.* In dogs, caudal retraction of the humerus in relation to the scapula is shoulder flexion, whereas cranial motion of the humerus in relation to the scapula is shoulder extension. The direction of shoulder flexion motion is opposite to this in humans. The terminology used in dogs is consistent with naming flexion as described previously.

In normal stance, as shown in Figure 5-2, a dog's spine is flexed at the atlantooccipital and atlantoaxial joints, straight (neither flexed nor extended) in the remainder of the cervical spine, extended at the cervicothoracic junction, slightly lordotic in the thoracic spine, and flexed or normally kyphotic in the lumbar spine. There is either a slightly flexed or extended sacrum on the lumbar spine, depending on the tail posture. The flexed canine lumbar spine is beneficial to running speed. During running, the lumbar spine moves through varying degrees of flexion as running speed changes.

Extension

During extension, the limb reaches out, the digit is extended, and the back or neck is less arched dorsally or

Box 5-2 Types of Joints

Forelimb

- Artificial joint: Not described as a joint
- Ball and socket: Shoulder
- Hinge: Elbow, metacarpophalangeal I
- Pivot: Proximal, and distal radioulnar
- Syndesmosis: Middle radioulnar
- Hinge with lateral motion: Carpal
- Ellipsoid: Antebrachiocarpal, radiocarpal
- Plane: Middle carpal or midcarpal, intercarpal, intermetacarpal
- Saddle plane: First carpal with MC I
- Plane: Second carpal with MC II, third carpal with MC III, fourth carpal with MC IV and V intermetacarpal
- Hinge: Metacarpophalangeal I
- Condylar or condyloid: MC II to V with the same numbered proximal phalanx
- Saddle/condylar
  Proximal interphalangeal II to V
  Distal interphalangeal II to V
  (Interphalangeal of thumb)

Hindlimb

- Synovial and fibrous: Sacroiliac
- Symphysis: Symphysis pelvis
- Ball and socket: Hip or coxofemoral
- Complex condylar: Stifle (the term *knee* is used commonly with an animal's owner)
- Plane
  Patellofemoral
  Tarsal joints or hock joints (this joint is referred to as the *hock joint* in common usage)
  Talocalcaneal
  Talocalcaneocentral and calcaneoquartal joints combined
  Proximal intertarsal or talocentral
  Calcaneoquartal
  Calcaneocentral
  Centroquartal
  Centrodistal
  Distal intertarsal: Central bone with tarsal III
  Tarsal I with II, II with III
  Tarsal III with IV
  Tarsometatarsal
  Tarsal I with MT I
  Tarsal II with MT II
  Tarsal III with MT III
  Tarsal IV with MTs IV and V
  Intermetatarsal
- Tibiofibular
  Synovial: Proximal and distal tibiofibular
  Syndesmosis: Middle tibiofibular
- Hinge: Talocrural, tarsocrural, tibiotarsal (the tarsocrural has been referred to as the talocrural and the talocalcaneal joints combined) or ankle joint (the term *ankle* is commonly used with an animal's owner)
  Metatarsophalangeal I—MT I with digit I
- Condylar: MT II to V with the same numbered digit
- Saddle
  Proximal interphalangeal II to V
  Distal interphalangeal II to V
  Interphalangeal of hallux

Spine

- Part synovial and part fibrous: Sacroiliac
- Condyloid: Atlantooccipital
- Pivot: Atlantoaxial—dens of C2 and atlas
- Plane
  Atlantoaxial—articular surfaces
  Between cranial and caudal articular surfaces
  Costovertebral
  Sternocostal: Sternum and true ribs
  Synchondrosis: Costochondral—ribs with cartilage

MC, Metacarpal; *mT,* Metatarsal.

arched ventrally. Extension is motion in the sagittal plane in the direction opposite to that of flexion motion. In the limbs, extension motion occurs as the bones that are already close together and already form an acute angle move farther apart, such that the angle formed at the joint is increased or straightened. Extension beyond normal is sometimes termed *hyperextension.* The carpus normally has greater than 180 degrees of extension. In the spine, extension occurs as the back or neck is arched ventrally (i.e., the convex portion of the arch is directed ventrally).

Abduction

Limb joint abduction is movement in the transverse plane such that the distal aspect moves away from the midline of the body. Abduction of the digits is not routinely measured in dogs. Separation of the toes may be noted as splayed toes or abducted toes. The reference line for abduction of the digits passes between digits III and IV. Digit I, or the dewclaw, in dogs is nonfunctional and is not considered in a discussion of skeletal motion.

Adduction

Limb joint adduction is movement in the transverse plane such that the distal aspect moves toward the midline of the body. Adduction of the digits is not routinely measured in dogs. The reference line for adduction is the same as for abduction.

Side Bend

In the trunk and head, side-to-side movement occurs in the dorsal plane such that lateral aspects of adjacent areas of the spine approximate or move away from each other. For example, side bend to the right involves the right side of adjacent spinal areas approximating each other.

Circumduction

Circumduction is movement at a joint, during which a bone or body segment outlines the surface of a cone or circle. This motion does not require rotation at the joint, but rotation at the joint may occur. Forelimb circumduction occurs primarily at the shoulder. Hindlimb circumduction occurs at the hips. Although circumduction at individual limb joints is possible in dogs, veterinarians commonly observe and document circumduction of the entire limb, without distinction of the joints that contribute to the overall motion.

Rotation

Internal rotation of a limb occurs with reference to the cranial aspect of the limb rotating toward the midline of the body or median plane and continuing in that direction. External rotation of a limb occurs with reference to the cranial aspect of the limb rotating away from the midline of the body and continuing in that direction. Limb rotational motions occur in the dorsal plane. With reference to the ventral aspect of a vertebral body, rotation to the right or left is called *rotation to the right or left,* respectively. Spinal rotations occur in the transverse plane.

Supination

In the limbs, supination is external rotation of the limb, such that the pad surface of the paw faces medially. Supination motion therefore could involve forelimb carpal and forepaw supination, shoulder external rotation, or hindlimb talocrural and hindpaw supination, as well other motions such as hip joint external rotation. Scapular motion and glenohumeral external rotation occurring as part of supination occur in the dorsal plane. However, dorsal plane scapular motion is blocked by contact of the scapula with the trunk.

Pronation

In the limbs, pronation is internal rotation of the limb, such that the pad surface of the paw faces laterally. Pronation motion therefore could involve forelimb carpal and forepaw pronation, as well as other motions, opposite to those of supination.

Other Shoulder Girdle Motions

Other shoulder girdle motions that are defined for humans exist in dogs, but have not been defined and measured in dogs. They include the human motions of scapular protraction, retraction, upward rotation, downward rotation, elevation, depression, anterior tilt and return from anterior tilt of the scapula, and glenohumeral horizontal abduction and horizontal adduction. Human scapulohumeral rhythm has not been defined in dogs, but may exist.

Pelvic and Sacroiliac Motions

Pelvic and sacroiliac motions and postures are less well defined in dogs than in humans. Human pelvic and sacroiliac motions and postures are described in depth, although terminology is not consistent among authors and clinicians. When pelvic and sacroiliac motions and postures are analyzed and described in dogs, the following terminology could be useful for correct identification and for accurate communication. Pelvofemoral rhythm, the human lower-limb counterpart to human scapulohumeral rhythm, has not been defined in dogs, but may exist.

Pelvic Motion

Ventral and Dorsal Pelvic Tilt

Orientation of the pelvis with respect to the sagittal plane describes tilt of the pelvis. Ventral tilt involves approximation of the ventral pelvis and cranial femur, and dorsal tilt involves the ventral pelvis moving away from the cranial femur. These motions occur with the hip joints as the axes of rotation. Relative to a longitudinal axis through the entire spine, the canine pelvis appears tilted dorsally on the canine longitudinal axis.

Lateral Tilt of the Pelvis

Lateral tilt is dorsal plane movement such that the lateral aspect of the pelvis approximates the lateral aspect of the trunk. Lateral pelvic tilt to the right, for example, would occur as the cranial border of the pelvis tilts to the right, approximating the right lateral side of the pelvis toward the right trunk.

Pelvic Rotation

With reference to the movement of the ventral aspect of a pelvis, rotation toward the right or left is called *rotation to the right or left,* respectively. This motion occurs in the transverse plane. Pelvic rotation can be referenced to the floor and one lower limb. If this convention were used, internal rotation with reference to the right hindlimb would occur as the ventral pelvis rotates to the left. Internal rotation motion of the right hindlimb would occur in the same direction, that is, with the cranial aspect of the thigh or femur rotating to the left and the ventral aspect of the leg or tibia and fibula rotating to the left.

Sacroiliac Motion

The suggested but as yet undefined terminology uses application of human terminology, with movements of the canine cranial and caudal aspects of the sacrum correlated to movement of the human superior and inferior aspects of the sacrum, respectively.

Sacroiliac Flexion or Nutation

Sacroiliac flexion involves the cranial aspect of the sacrum tilting ventrally.

Sacroiliac Extension or Counternutation

Sacroiliac extension involves the cranial aspect of the sacrum tilting dorsally.

Sacroiliac Side Bend or Lateral Flexion

Sacroiliac lateral flexion involves the cranial aspect of the sacrum tilting to the side. Right side bend or lateral flexion occurs as the cranial aspect of the sacrum tilts to the right.

Sacroiliac Rotation

Sacroiliac rotation involves rotation around a longitudinal axis through the sacrum. Rotation of the sacrum is identified by the motion of the ventral sacrum to be consistent with terminology for humans. For example, rotation of the ventral sacrum to the left is called *sacral rotation to the left.*

Overall spinal motion is determined and affected by many things. Among them are the orientation of the articular surfaces (facets); disk shape and size; structural blockages, such as ribs and orientation of spinous processes; ligament fiber direction, strength, and tightness; and muscle-tendon unit strength and tightness.

Normal Joint Motions and Ranges of Motion

Appendix 2 summarizes normal joint motions and ranges of motion for the peripheral joints.[5,7,8] Joint ranges of motion for the spine have not been documented in dogs.

Distinction of Maintained Joint Postures versus Joint Motion

Motion is described by either referencing the joint or referencing a body segment. Terms to describe motion include *flexion, flexing, abduction, abducting,* and so on. Motion occurs in a consistent direction, independent of the starting point of the motion. For example, the movement of the carpal joint from neutral to flexion is carpal joint flexion. Movement of the carpal joint from full extension to neutral is carpal joint flexion. Postures are maintained positions. Terms to describe postures include *flexed* or *abducted, maintained flexion,* or *abduction.*

Skeletal Alignment

Limbs

Skeletal alignment is important, because it is closely linked to mobility, function, and pathologic conditions. A functional example of the importance of skeletal alignment is seen at the talocrural joint. Alignment at this joint influences angulation of the hindlimbs in relation to speed during the gallop. Figures 5-5, 5-6, 5-8, and 5-9 show the normal alignment of the limbs.

Skeletal alignment is the alignment between bones at joint surfaces or the alignment of one portion of a bone in relation to another portion of the same bone. *Skeletal malalignment* is abnormal alignment of either of these two relationships.[9] Skeletal alignment at any one joint affects the alignment at other joints, especially at joints functioning in weight-bearing or closed-chain activities rather than non–weight-bearing or open-chain activities. Skeletal malalignment can lead to arthritis of a malaligned joint or of a compensating joint.

Normal skeletal alignment results in normal correlated motions and postures, which occur naturally and are normally related to the motion or posture imposed at the joint. Correlated motions and postures may not occur at all but can occur at any or several joints along the chain. This concept is well defined in human physical therapy, although clinicians use different terms to describe it. These concepts have been discussed in relation to dogs, mostly in the context of canine gait and related to correlated motion as a result of a limb with pain or dysfunction, rather than from skeletal malalignment alone.[10] An example of correlated posture or motion is the lateral rotation of the proximal forelimb or hindlimb that occurs when the dog supinates at the paw. Another example is the excessive shoulder or hip adduction and medial positioning of the elbow or stifle joints, which occur when the digits angle or point laterally in the forepaw or hindpaw, respectively.

For any skeletal malalignment or associated abnormal correlated motion or posture, compensatory motions or postures can occur. They occur to normalize mobility, improve cosmetic appearance of the limb, or improve foot contact with the ground. The term *compensation* is used to describe abnormal motion and posture in dogs.[10] An example is excessive weight bearing on one limb when dysfunction exists in the other limb. Another example of a compensatory motion is limb circumduction while advancing that limb during gait, as a result of inability to flex the joints of a limb during swing.

Spine

Complex correlated and compensatory postures and motions occur within the spine and pelvic complex. The pelvic complex for dogs and humans is defined in Box 5-1. Human correlated and compensatory postures and motions related to the spine and pelvis are described in great detail in the physical therapy literature[11-14]; however, there is controversy and even contradiction related to which motions and postures are correlated or compensatory for other motions and postures. This concept has been applied in less detail to spinal and pelvic postures and motions in dogs; however, development and use of terminology are encouraged for analysis of spinal motion and posture.

Fascia (Fasciae)

Fascia (fasciae), formed by flat, enveloping connective tissue, weaves throughout the body and provides structural

connection. With damage, it can become adhered to adjacent structures. Physical therapists and other clinicians intervene in such circumstances with treatments such as stretching, transverse friction massage, and soft tissue mobilization to reduce this adherence.

Forelimb

The superficial and deep fascia of the shoulder and brachium are continuous with the superficial and deep fascia of the cervical spine and thorax. The superficial fascia in the region of the sternum is continuous with the medial brachium, and it covers the cephalic vein during its course. Distal to the elbow, the superficial fascia is more adhered to the deep structures. The cutaneous nerves and vessels of the forelimb travel long distances deep to the superficial fascia before entering the skin.

The deep fascia of the lateral shoulder and brachium, called the *fascia omobrachialis lateralis,* covers the muscles' superficial surfaces and adheres to the spine of the scapula. The deep antebrachial fascia forms a single dense sleeve around the muscles in the caudal antebrachium and is thickest medially. Intermuscular septa extend to the radius and ulna to separate the flexor and extensor carpal muscle groups.

The deep fascia is closely joined with the connective tissue around the tendons of the carpal joint. A thick and strong extension, the flexor retinaculum, bridges the flexor tendons to the carpal joint and digits. On the palmar surface of the metacarpophalangeal joints, thick extensions attach to the sesamoid bones and are called *transverse ligaments.*

Hindlimb

The hindlimb also has superficial and deep layers of fascia. At times the fascial layers are separable, especially with a layer of fat in obese dogs, and at times they are not. In general, the deep fascia is stronger than the superficial.

The superficial fascia on the dorsal trunk is continuous with the superficial gluteal fascia of the hip area, whereas the superficial fascia on the ventrolateral abdomen is continuous as the superficial lateral fascia of the lateral thigh and gluteal region. The superficial fascia encompasses the thigh almost to the patella and is closely connected to the deep fascia of the biceps femoris muscle. The superficial and deep fascia are also closely associated near the proximal sartorius muscle, the femoral canal, and the gracilis muscle. The superficial fascia of the crus, tarsus, and hindpaw is similar to that in the forelimb.

The strong lateral femoral fascia, or fascia lata (iliotibial band in humans), is formed on the lateral thigh from the gluteal fascia and the fascia of the tensor fasciae latae muscle and attaches to the lateral femur by an intermuscular septum between the biceps femoris and vastus lateralis muscles. The more loosely adhered medial femoral fascia joins the lateral femoral fascia to envelop the thigh and attach to the medial femur by a septum caudal to the vastus medialis. Both lateral and medial fascial layers attach to the patella and femoral condyles. The fascia extends around both the medial and lateral aspects of the stifle, but it thins as it merges with the patellar ligament and patella. The deep fascia continues in the crus as the crural fascia. The crural fascia has a superficial layer, which extends to the metatarsus, and a deep layer. The superficial and deep leaves are united near the distal tendons of the biceps femoris and semitendinosus, where they contribute to the common calcaneal tendon. The layers are also united to the distal tendons of the semimembranosus and cranial tibial muscles. The deep crural fascia thickens to form the crural extensor retinaculum, which envelops the long digital extensor and cranial tibial tendons, and the tarsal extensor retinaculum, which envelops the long digital extensor muscle. These retinacula ensheath all tendons and superficial muscles of the hindpaw and secure them to the bones in the hindpaw.

Trunk and Neck

The superficial and deep fascial layers of the neck and head are continuous. Although the deep fascia is stronger than the superficial fascia, it is deeper and more difficult to palpate; therefore the superficial fascia is emphasized here. The superficial fascia of the neck ensheathes the entire neck, is continuous toward the forelimb as the superficial brachial fascia, and is continuous ventrally with the superficial fascia of the trunk in the sternal region. Caudally, the superficial external fascia of the trunk is continuous with the superficial gluteal fascia. At the dorsal midline in dogs, there is no attachment of the superficial fascia to deep structures, allowing the superficial fascia to be lifted in a big fold when the skin is gently lifted. Large quantities of subfascial fat are deposited in areas where the superficial fascia and skin move freely on deeper structures.

The deep external fascia is attached to the supraspinous ligament on the spinous processes of thoracic and lumbar vertebrae and ends cranially on the transverse processes of C7 and T1. In the trunk, it is continuous with the lateral abdominal muscles. The deep fascia attaches to the sternum and costal cartilages cranially and the ilium caudally. The dorsally located thoracolumbar fascia has two layers and lies deep to large amounts of fat in well-nourished dogs. The main layer serves as an attachment site for the serratus dorsalis cranialis and the internal and external abdominal oblique muscles. The deep fascia is continuous with the deep gluteal fascia and other deep fascia of the thigh.

Compartments

Fascial layers and the bones they attach to can form well-defined volumetric spaces called *compartments*. Compartments are described in the limbs and contain muscles and their associated neurovascular supply. Compartments become important when interstitial fluid hydrostatic pressure within the compartment exceeds physiologic levels, causing ischemia, disruption of membrane integrity, edema, and loss of viable muscle tissue.

Four distinct compartments have been described in the dog: (1) craniolateral compartment of the crus, (2) caudal compartment of the crus, (3) caudal compartment of the antebrachium, and (4) femoral compartment.[15] Unlike the others, the femoral compartment is not a single space but consists of three fascial envelopes; one contains the quadriceps muscles, another the hamstring group, and the final one surrounds the adductor muscle.

Muscles

Muscles are characterized as primary movers (i.e., critical in performing joint actions) and secondary movers (less important in performing joint actions). Primary movers are the muscles that produce the necessary moments for normal (physiologic) joint motion and have the best combination of characteristics: large physiologic cross-section, large moment arm, and a good line of action based on the desired joint motions. Primary movers usually cross the joint with the desired motion at the last joint crossed.

Muscle strength depends on the multiplied product of the muscle force applied in the direction of the desired joint motions and the moment arm of the muscle force. The size of the muscle and the direction of its line of action force vector determine its ability to produce specific motion. Muscle force is related to the physiologic cross-sectional areas of muscle fibers. The line of action determines the length of the moment arm, which determines the moment arm's contribution to the muscle moment that can be produced.

Interestingly, some canine muscles have been named according to the number of heads in analogous human muscles, but the actual number of heads making up the muscle may be different. For example, the biceps brachii in dogs has one head and the human biceps brachii has two heads, but both muscles are called biceps brachii.

Forelimb

Muscle number, size, and location are related closely to forelimb function as a weight-bearing limb (Figures 5-15 to 5-21). In addition, the need to suspend the head and neck increases the need for muscle mass and strong ligaments that extend between the head, neck, and forelimb. The

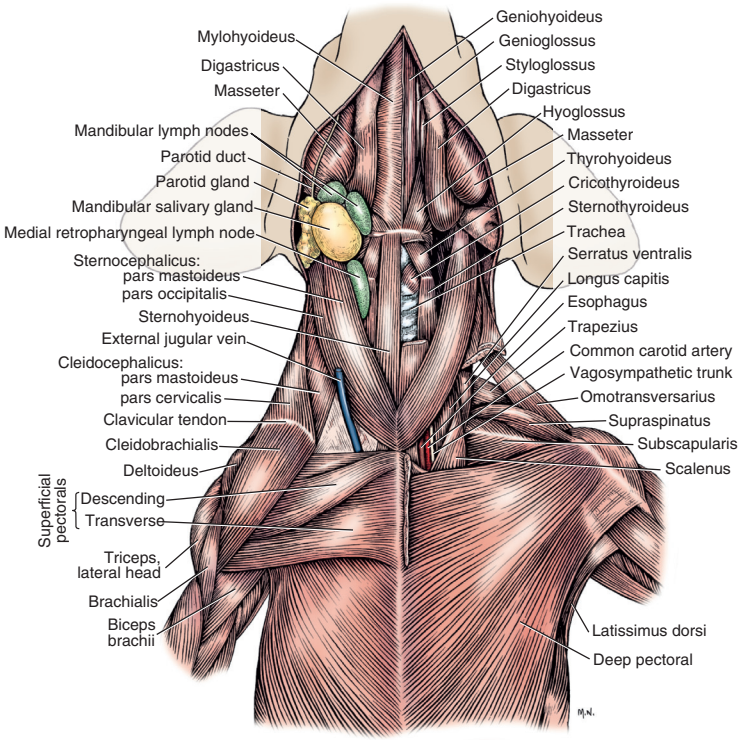


Figure 5-15 Superficial muscles of the ventral shoulder and neck. (From Evans HE: *Miller's anatomy of the dog,* ed 4, Philadelphia, 2013, WB Saunders.)

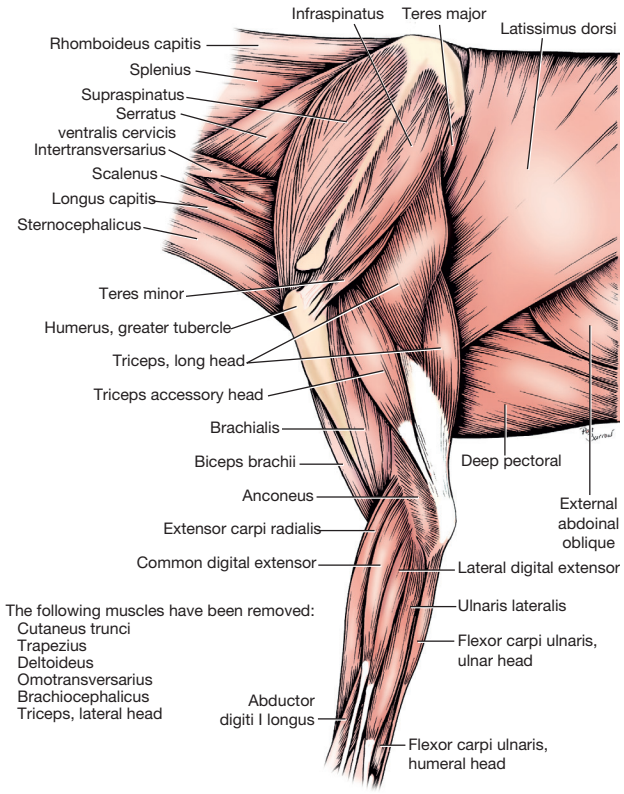


Figure 5-16 Deep muscles of the left lateral forelimb. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

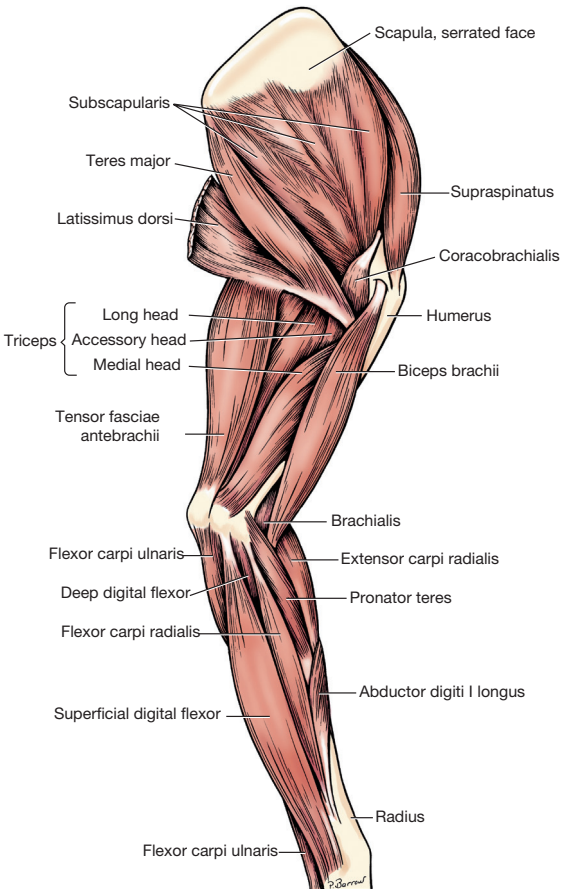


Figure 5-17 Muscles of the left medial forelimb. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

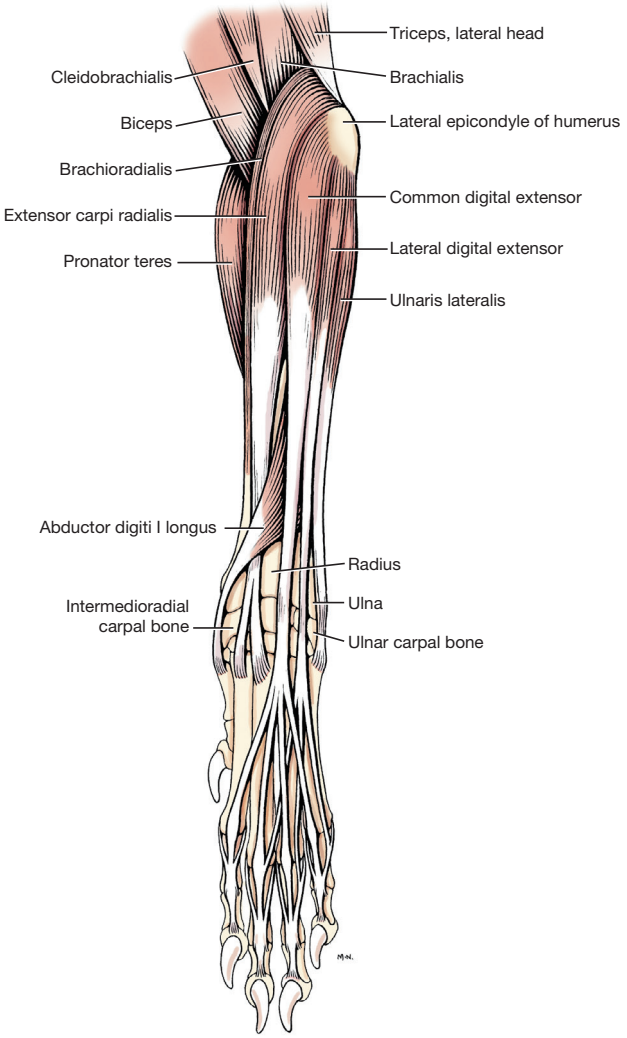


Figure 5-18 Muscles of the left antebrachium and forepaw, cranial view. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

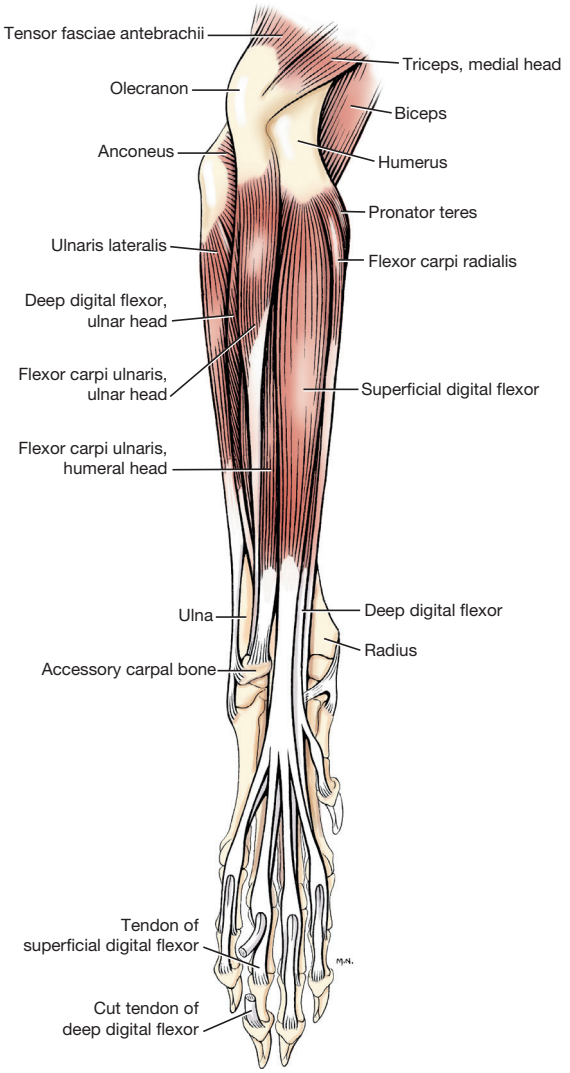


Figure 5-19 Muscles of the left antebrachium and forepaw, caudal view. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

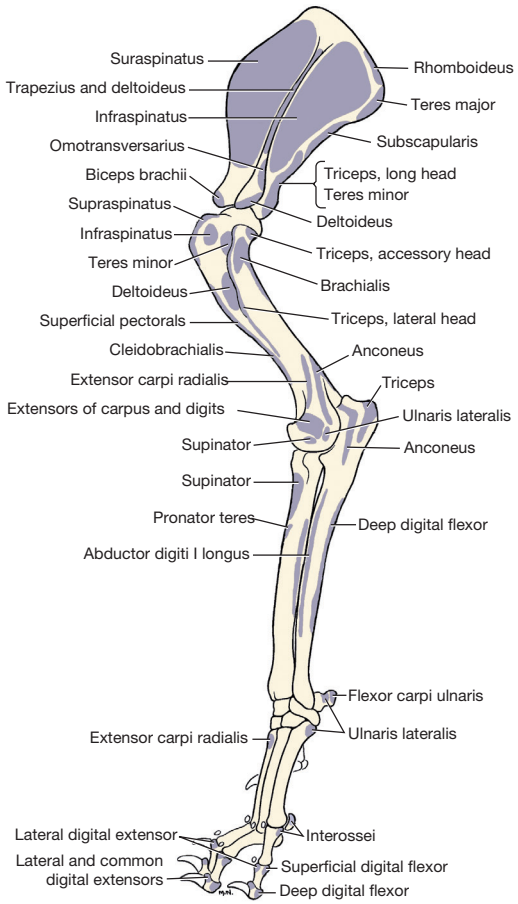


Figure 5-20 Muscle attachment sites on the lateral forelimb skeleton. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

canine head and neck function mechanically like a cantilever beam. The weight of the head at the end of the "beam" produces a large neck flexion moment, which must be balanced by spinal ligaments and neck extensor muscles.

Dogs have a large muscle mass in shoulder adductors and elbow extensors because of the weight-bearing function of the forelimb. These antigravity muscles support the dog in weight bearing. Antigravity muscle groups for the forelimb include the following (see Figures 5-15 to 5-21):

- Shoulder extensors in closed chain and end-range open chain, and shoulder flexors in open chain from normal stance posture
- Shoulder adductors and abductors, depending on the weight-bearing posture
- Elbow extensors in closed chain, and flexors in open chain from stance
- Carpal and digit flexors in closed chain and end-range open chain, and extensors in open chain from normal stance

Prime movers for forelimb muscle groups are as follows:

- Shoulder flexors—latissimus dorsi, deltoideus, long head of the triceps brachii, teres major, teres minor
- Shoulder extensors—cleidobrachialis, biceps brachii, supraspinatus, brachiocephalicus
- Elbow flexors—biceps brachii and brachialis
- Elbow extensors—triceps brachii
- Carpal flexors—flexor carpi radialis, ulnaris lateralis, flexor carpi ulnaris
- Carpal extensors—extensor carpi radialis; carpal flexor and extensor muscles have flexor and extensor retinacula to keep tendons to the digits in proper alignment
- Digit flexors—superficial and deep digital flexors
- Digit extensors—common and lateral digital extensor muscles

Web Appendix 1 contains information on muscles, attachment sites, joint actions, analogous human muscles (muscle that performs a similar function in humans), and

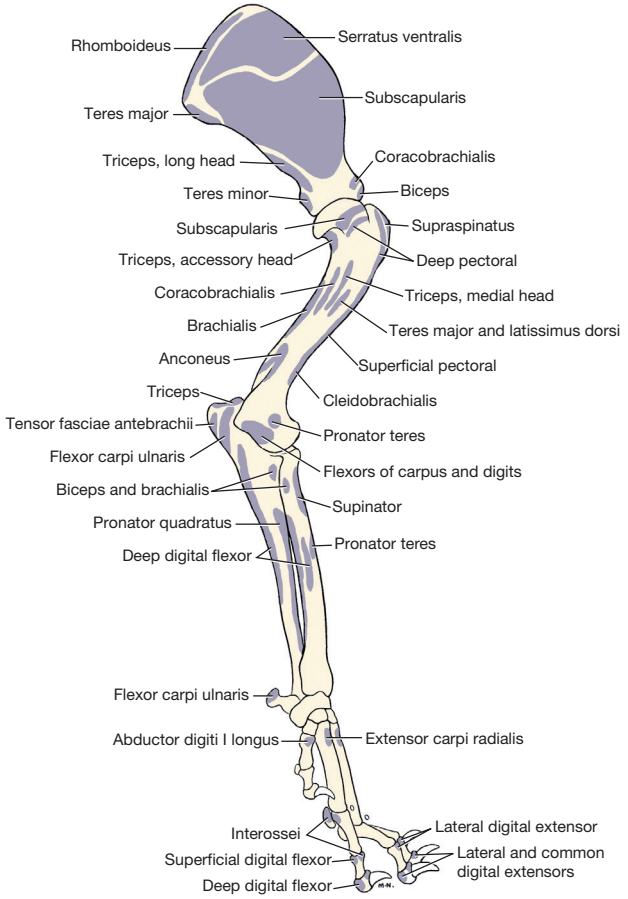


Figure 5-21 Muscle attachment sites on the medial forelimb skeleton. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

innervation for prime movers of joint actions of the forelimb, hindlimb, and trunk and neck.

Hindlimb

The antigravity muscles of the hindlimb support the dog in weight bearing or closed chain (Figures 5-22 to 5-30). In normal stance, the angle of the hip and stifle are both approximately 110 to 150 degrees, with perhaps more flexion in the stifle joint. This posture creates a constant requirement for large hip extensor and stifle extensor moments. The main antigravity muscle groups for closed-chain function in the hindlimb are as follows:

- Hip extensors in the sagittal plane
- Hip adductors in the frontal plane
- Stifle extensors
- Talocrural extensors
- Digit flexors

Prime movers for hindlimb muscles include the following:

- Hip flexors—iliopsoas, sartorius, tensor fasciae latae
- Hip extensors—gluteal muscles, biceps femoris, semitendinosus, semimembranosus
- Hip abductors—middle gluteal

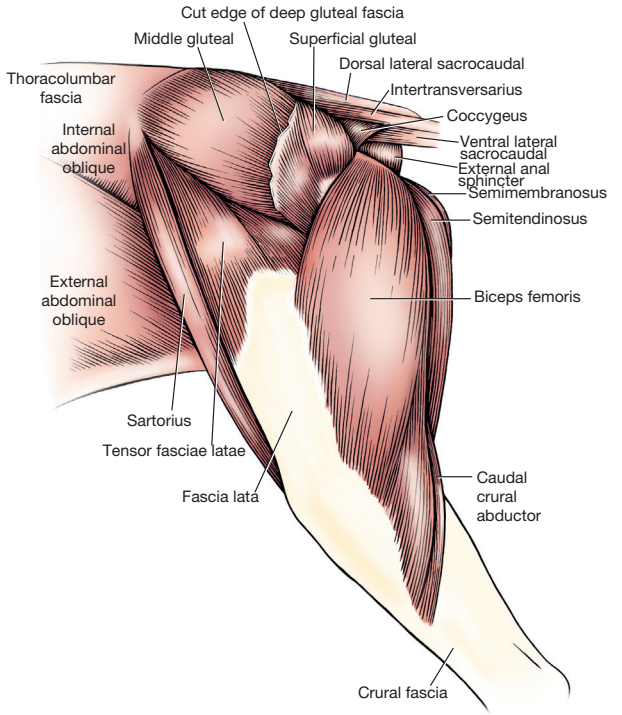


Figure 5-22 Superficial muscles of the left lateral hindlimb. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

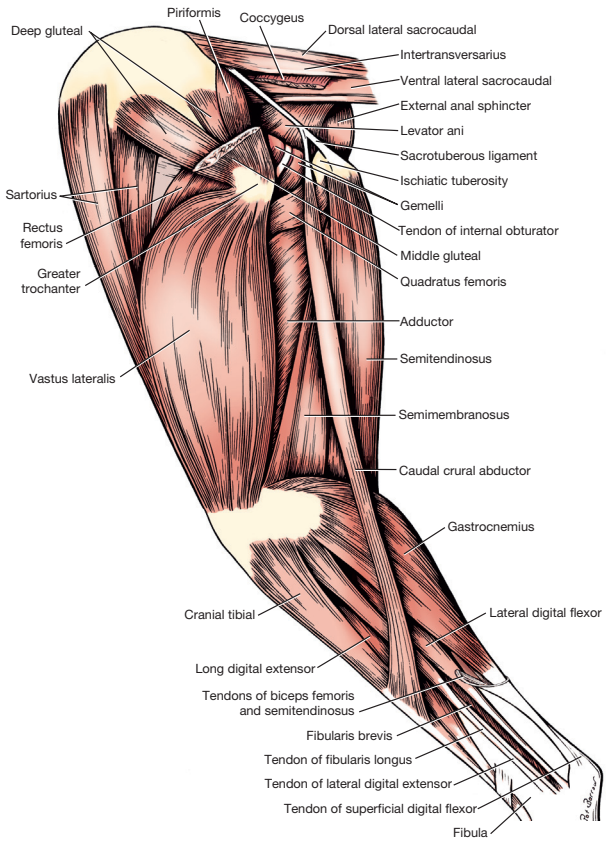


Figure 5-23 Deep muscles of the left lateral hindlimb. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

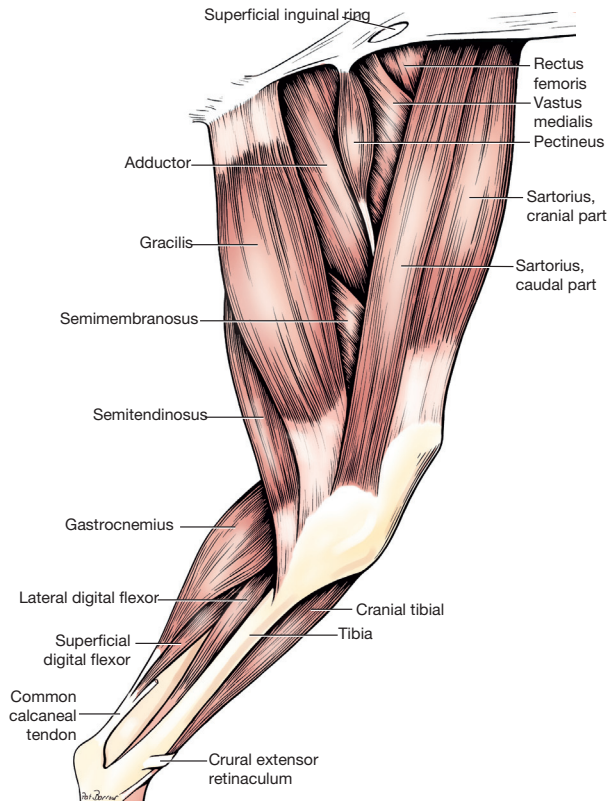


Figure 5-24 Superficial muscles of the left medial hindlimb. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

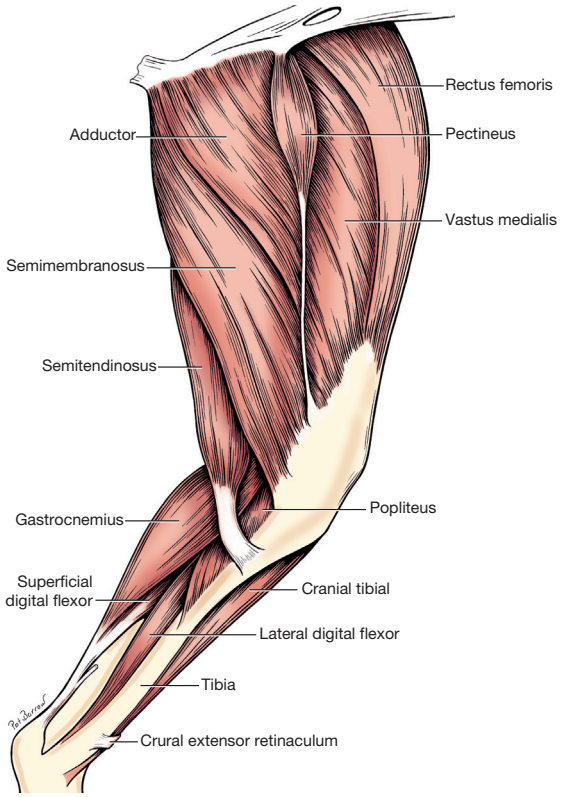


Figure 5-25 Deep muscles of the left medial hindlimb. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

- Hip adductors—adductor magnus et brevis, pectineus
- Hip lateral rotators—internal and external obturator, gemelli
- Hip medial rotators—deep gluteal, semitendinosus
- Stifle extensors—quadriceps femoris: rectus femoris, vastus lateralis, vastus medialis, vastus intermedius
- Stifle flexors—biceps femoris, semitendinosus, semimembranosus
- Talocrural flexors—cranial tibial
- Talocrural extensors—gastrocnemius, superficial digital flexor
- Digit flexors—superficial and deep flexor
- Digit extensors—long digital extensor

Whereas the front limbs bear the majority of the static weight of the dog, the rear limbs provide the necessary propulsion for dynamic activity such as running and jumping.

As a result, the hamstring muscles are large and therefore have a large force capacity. The hamstring muscles in the dog are divided into cranial and caudal groups. The cranial group consists of the cranial parts of the biceps femoris and semimembranosus. The caudal group consists of the caudal parts of the biceps femoris and semimembranosus and the whole semitendinosus. The division into groups is based on electromyographic and functional characteristics. The cranial group functions primarily as antigravity muscles in which contraction is prominent at the point of foot placement and during the stance phase executing extension of the hip, whereas the caudal group contracts during the swing phase executing flexion of the stifle.[16] When compared with the human, the dog's ischial tuberosity is farther from the hip joint. Therefore the origin of the hamstrings on the ischial tuberosity provides a longer moment arm and therefore a relatively larger extensor moment about the hip joint. At the region of the femorotibial joint, the entire biceps femoris muscle courses superficial to the vastus lateralis muscle and, as an aponeurosis, joins the fascia lata and the fascia cruris. Based on these attachments, the biceps femoris is very effective in producing hip extension and stifle flexion, making this muscle an effective source of propulsion. The middle gluteal and adductor muscles are large, thick muscles. The superficial gluteal is small and located more on the lateral hip than the caudal hip.

The quadriceps femoris muscles are large. This is consistent with the constant and large stifle extension moment caused by the stifle position in normal stance.

At the talocrural joint, the canine gastrocnemius muscle is relatively large, and usually there is no soleus muscle. The long digital extensor muscle crosses the stifle and tarsal joints on the cranial surfaces. The tendons of the lateral digital extensor and peroneus brevis muscles travel

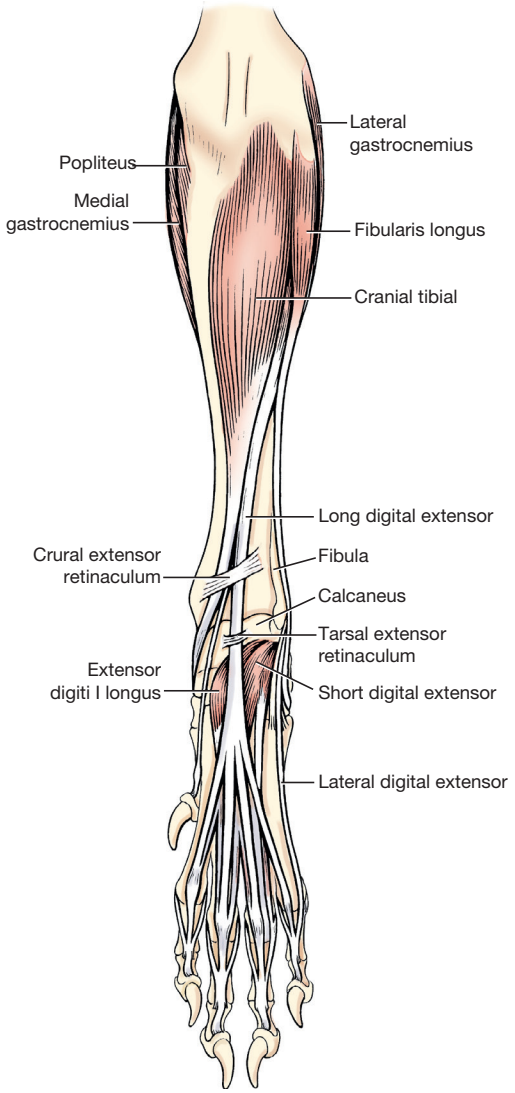


Figure 5-26 Muscles of the left leg and hindpaw, cranial view. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

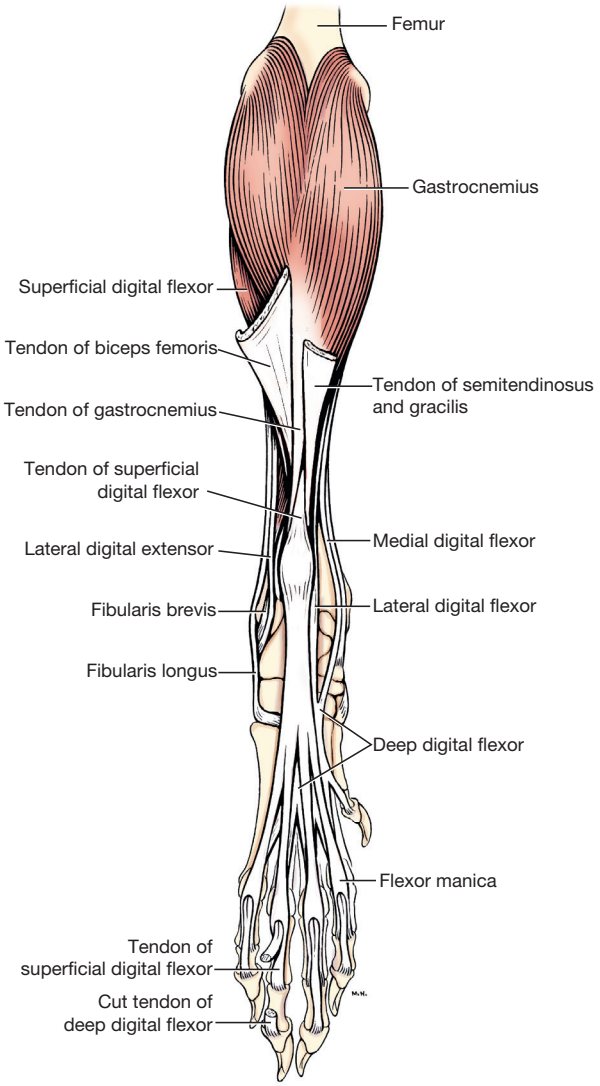


Figure 5-27 Muscles of the left leg and hindpaw, caudal view. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

in a distinctive groove, the sulcus malleolaris lateralis, on the lateral malleolus.

Trunk and Neck

Figures 5-31 to 5-36 display visual information about trunk and neck musculature. Canine muscles of the trunk and neck are characterized by nerve supply and embryologic development into hypaxial and epaxial muscles. The epaxial muscles develop locally in the trunk and are innervated by the dorsal rami of the spinal nerves. They include muscles that lie dorsal to the transverse processes of the vertebrae and have a main action of extension of the spine. The hypaxial muscles lie ventral to the transverse processes, are innervated by the ventral rami, and have a main action of flexion of the spine. The hypaxial group also includes the muscles of the abdominal and thoracic walls. The superficial dorsal trunk muscles act on the forelimb as well as the cervical spine and are innervated by the ventral rami.

Antigravity muscle groups for the spine are the trunk and neck extensors. Prime-mover muscles for spine motion include the following:

- Cervical spine flexion of C1-C2—longus capitis and longus colli
- Cervical spine flexion caudal to C2—sternomastoideus, longus capitis, and longus colli
- Cervical spine extension—splenius
- Cervical spine side bend—scalenus
- Rotation of the cervical spine—splenius and sternomastoideus
- Flexion of the trunk (thoracic and lumbar spine)—rectus abdominis muscle

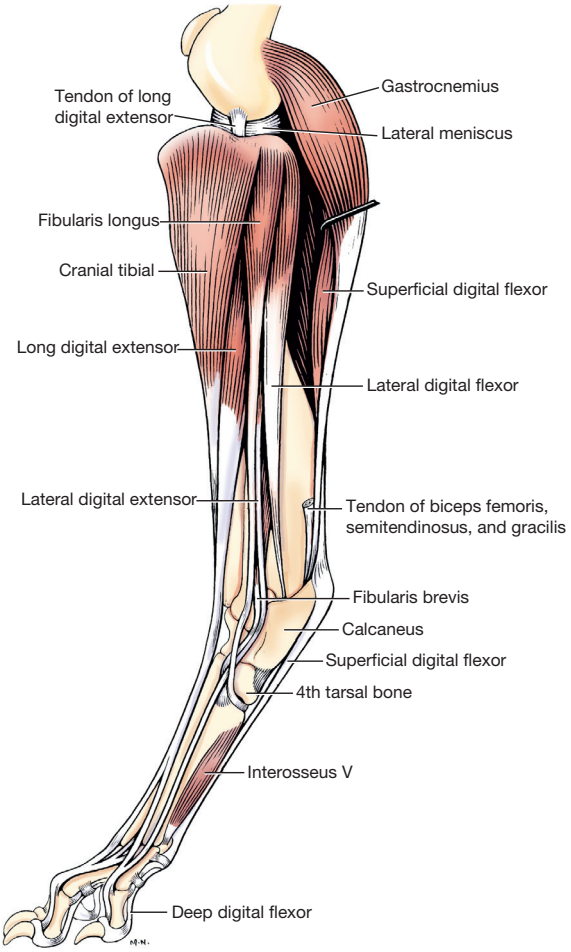


Figure 5-28 Muscles of the left leg and hindpaw, lateral view. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

- Extension of the trunk—sacrospinalis or erector spinae muscles
- Side bend of the trunk—pars lumbalis of the external abdominal oblique
- Rotation of the trunk—longissimus and pars costalis of the external abdominal oblique

The posturing of the dog's head and neck as a cantilever beam produces a large amount of extensor or resisting neck flexion moment on the cervical spine. This posture creates a constant requirement for large neck extensor muscle activity. In response, there is a large amount of neck extensor muscle mass. Trunk flexor and extensor muscle masses are both large, reflecting a large requirement for trunk extensor and flexor muscle activity. The abdominal muscles have a big role in supporting the weight of the internal organs.

The deep dorsal trunk musculature, the extensor muscle groups, consists of three longitudinal muscle masses, each having many overlapping fascicles. The three masses are the iliocostalis muscle system laterally, the longissimus muscle system intermediately, and the transversospinalis muscle system medially. Different arrangements of fused portions of these three primary segmental muscle masses exist, based on the running abilities of the breed. Dogs that run fast may have longer fascicles, which allow muscles to attach to the spine for increased leverage. These breeds have fused caudal portions of the iliocostalis muscle with the longissimus muscle to form the erector spinae or sacrospinalis muscle group.

Bursae

A synovial bursa (*bursae synoviales*) is a walled space containing a viscid fluid. The wall is composed of an outer tough layer, the membrana fibrosa, and a inner delicate layer, the membrana synoviales. Synovial bursae provide a gliding surface and a cushion for soft tissues as they pass over skeletal prominences and are found over bone and under muscle, tendon, ligament, fascia, or skin. Not all bursae are consistent structures, as some are present at birth (congenital) whereas others form later (acquired) in response to mechanical stresses.[17] Bursae can also be eliminated by aggressive inflammation as a result of infection or traumatic injury.

In the front limb of the dog, there is a bursa between the tendon of insertion of the infraspinatus muscle and the greater tubercle of the humerus. Frequently a second more proximal bursa can be found in association with the infraspinatus insertion.[17] Ossification of this bursa and the tendon of the infraspinatus tendon has been reported as a cause of lameness.[18] Diagnosis is suspected when pain is elicited by digital pressure over the site of the bursa and the tendon.

A bursa is present under the origin of the deep digital flexor muscle at the medial epicondyle of the humerus. The distal portion of the muscle is surrounded by a sheath extending from the level of the distal radius to the metacarpus. In some cases the sheath is less well defined with weaker walls and no fluid.[4]

Bursae are commonly associated with the extensor carpi radialis muscle. The joint capsule of the elbow forms a pocketlike structure under the proximal portion of the extensor carpi radialis muscle. Distally the extensor carpi radialis gives rise to two tendons, a medial tendon attaching to metacarpal II and a larger lateral tendon attaching to metacarpal III. In approximately half of all dogs there is a bursa that completely envelops both tendons as a complete fluid-filled tendon sheath. A separate bursa may be present under both tendons or just the lateral tendon at the level of the proximal row of carpal bones. A second bursa can be found under the lateral tendon at the level of the distal row of carpal bones. In other dogs fluid-filled sheaths or sacs are absent with a loosely meshed connective tissue in place.[4]

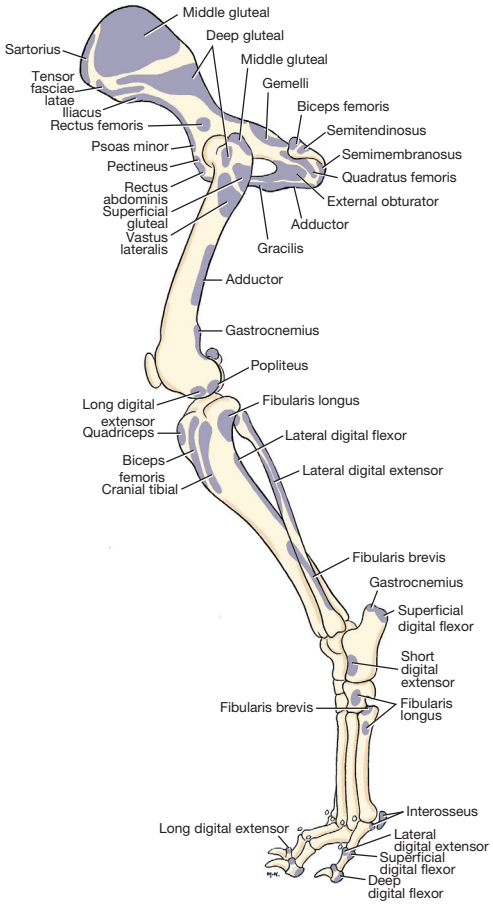


Figure 5-29 Muscle attachment sites on the lateral hindlimb skeleton. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

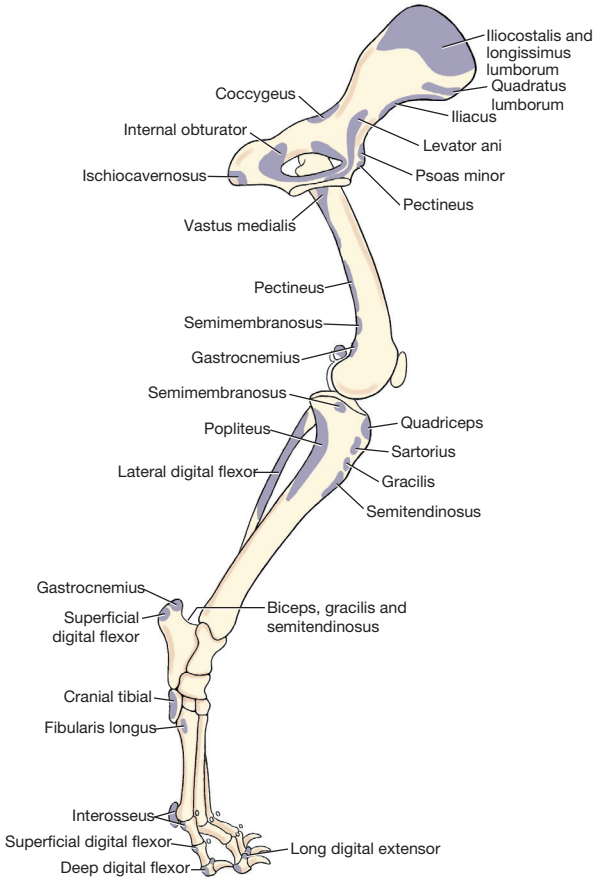


Figure 5-30 Muscle attachment sites on the medial hindlimb skeleton. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

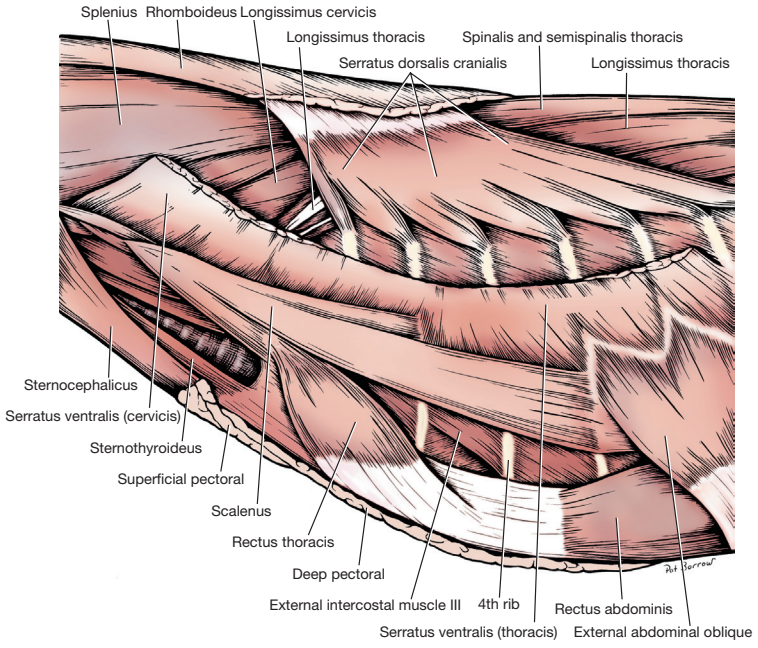


Figure 5-31 Muscles of the left lateral neck and thorax. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

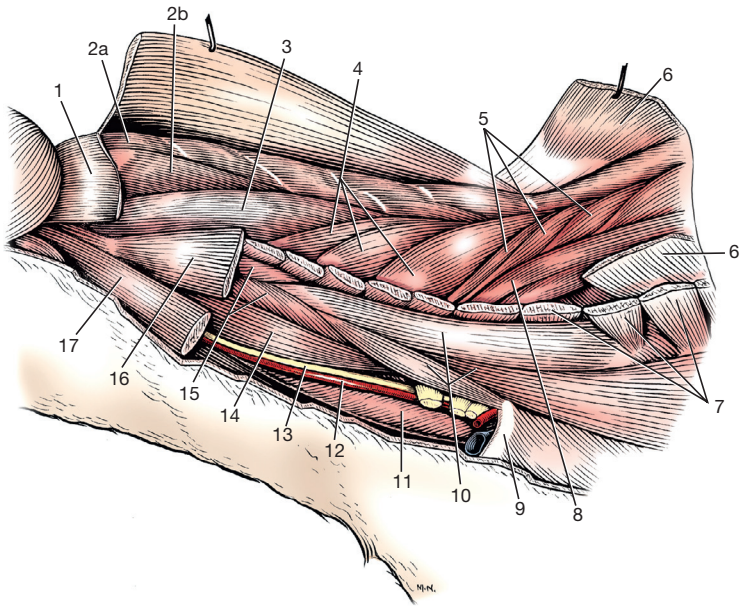


Figure 5-32 Deep muscles of the left lateral neck. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

1. Splenius
2. Semispinalis capitis:
   a. Biventer cervicis
   b. Complexus
3. Longissimus capitis
4. Longissimus cervicis
5. Longissimus thoracis
6. Serratus dorsalis cranialis
7. Serratus ventralis
8. Iliocostalis thoracis
9. First rib
10. Scalenus
11. Esophagus
12. Common carotid artery
13. Vagosympathetic trunk
14. Longus capitis
15. Intertransversarius
16. Omotransversarius
17. Mastoid part of cleidocephalicus

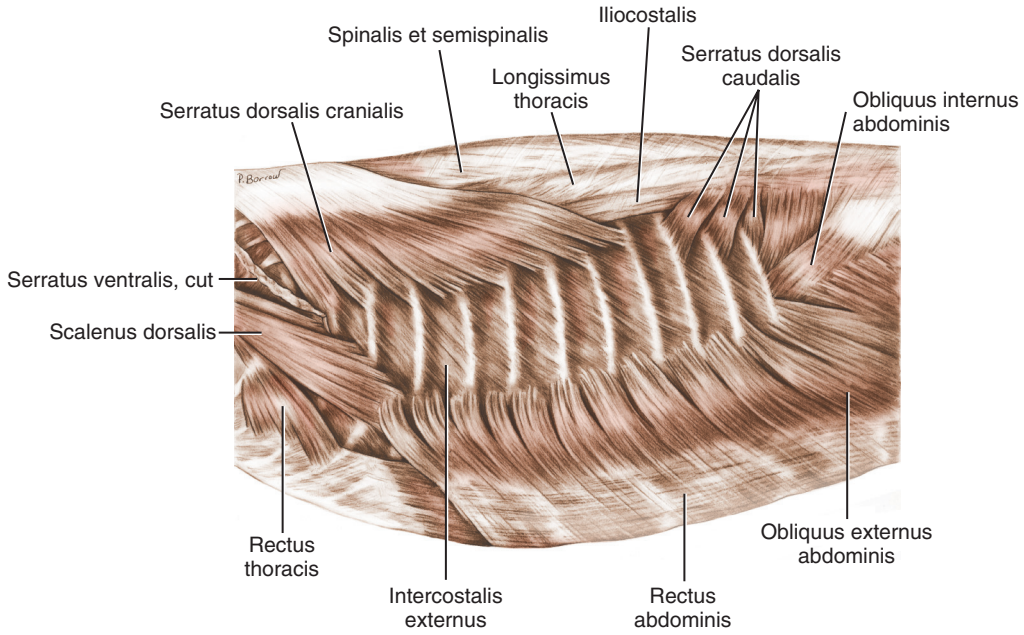


Figure 5-33 Superficial muscles of the left lateral thoracic cage. (From Evans HE: *Miller's anatomy of the dog,* ed 4, Philadelphia, 2013, WB Saunders.)

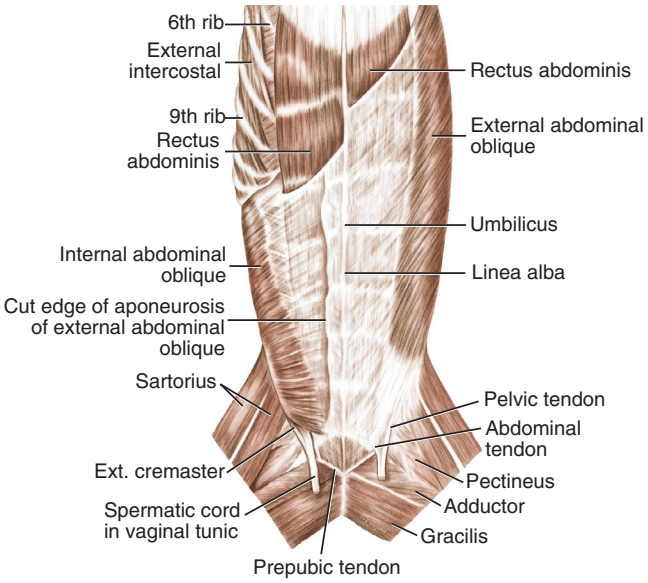


Figure 5-34 Superficial muscles of the ventral trunk. (From Evans HE: *Miller's anatomy of the dog,* ed 4, Philadelphia, 2013, WB Saunders.)

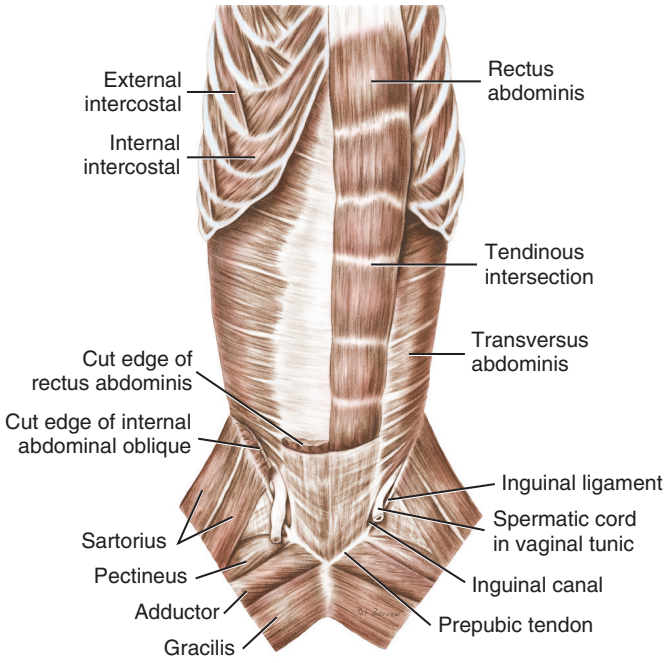


Figure 5-35 Deep muscles of the ventral trunk. (From Evans HE: *Miller's anatomy of the dog,* ed 4, St Louis, 2013, WB Saunders.)

Lymph Node Anatomy

Forelimb

Superficial cervical lymph nodes are located just cranial to the shoulder joint. They are deep to the cleidocephalicus and omotransversarius muscles and are on the lymph drainage path from the cutaneous area of the head, neck, and forelimb. The superficial cervical lymph nodes are almost always palpable. Axillary lymph nodes are located dorsal to the deep pectoral muscle and generally are not palpable unless they are abnormally enlarged. The deep structures of the forelimb, as well as lymph vessels from the thoracic wall, drain into these nodes.

Hindlimb

The popliteal lymph node lies in the fat just caudal to the stifle joint and at the caudal border of the biceps femoris muscle. This is the largest lymph node in the hindlimb. The hindlimb structures distal to the stifle joint drain into this lymph node. The femoral lymph node is not consistently present and is small when present. It lies in the fat deep to the deep medial femoral fascia at the distal part of the femoral triangle. The femoral vessels lie cranial to the femoral lymph node.

Trunk and Neck

The superficial inguinal lymph nodes are important lymph nodes in the trunk. In addition to the lymph nodes, trunk internal organ anatomy that is particularly relevant for rehabilitation includes the lung lobes, the anatomy of which is critical when administering postural drainage of individual lobes for dogs with respiratory insufficiency.[19]

Ligaments

Information about attachment, fiber direction, and motion restricted by the major forelimb, hindlimb, and spinal ligaments is noted in Web Appendix 2.[4,20] Listed order of the fiber direction in three planes is noted by the main orientation of the fibers first and follows in sequence for the second orientation of importance and then the third.

Forelimb

Figures 5-37 and 5-38 display ligament attachment site, location, and line of direction for the major ligaments of the forelimb. The canine shoulder joints have medial and lateral glenohumeral ligaments, which are mildly thickened regions of fibrous joint capsule. The transverse humeral retinaculum holds the tendon of the biceps brachii securely in place. The annular ligament holds the proximal radius securely to the ulna. The medial and lateral collateral ligaments of the elbow are substantial structures and contribute to elbow stability. Although there are no carpal collateral ligaments that span all the joints of the carpus, there are individual collateral ligaments at the various levels that are critical to carpal stability. Palmar ligament and fibrocartilaginous support of the carpus are vital for normal weight bearing.

Hindlimb

Figures 5-39 to 5-41 display hindlimb ligament attachment site, location, and line of direction for the major ligaments

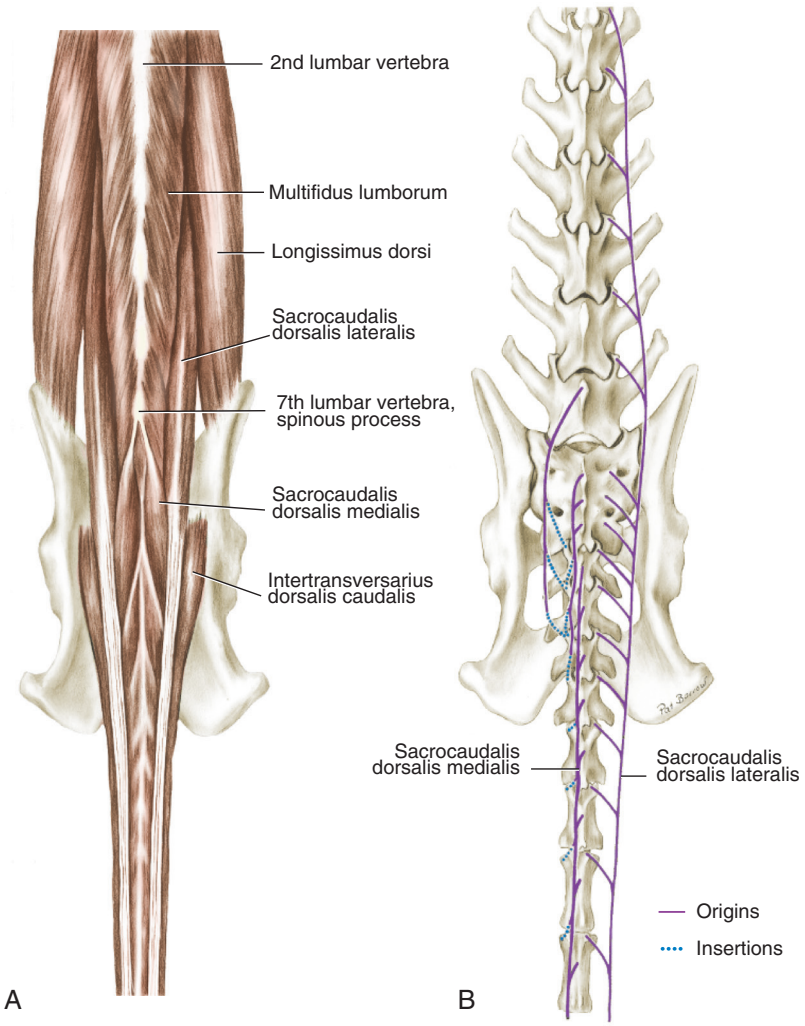


Figure 5-36 Muscles of the lumbocaudal region. **A,** Epaxial muscles, dorsal view. **B,** Diagram of skeleton and sacrocaudal muscle attachments, dorsal view. (From Evans HE: *Miller's anatomy of the dog,* ed 4, Philadelphia, 2013, WB Saunders.)

of the hindlimb. The iliofemoral, pubofemoral, and ischiofemoral ligaments are not present in the dog. The ligament of the femoral head does not contribute to hip stability as much as the capsule, but is important in the development of the hip. The transverse acetabular ligament traverses the acetabular notch and thereby completes the rim of the acetabulum. Without the transverse acetabular ligament, the hip may luxate ventrally. Although the sacrotuberous ligament is a pelvic ligament, rather than a hip-joint ligament, it is mentioned here because of its importance as one of the origins of the biceps femoris muscle.

The cranial (anterior) and caudal (posterior) cruciate ligaments are critical stabilizing ligaments that restrain stifle motion within physiologic limits. The cruciate ligaments are named by their cranial or caudal attachment to the tibia. Injury to the cranial cruciate ligament is common. The cranial cruciate ligament has different fiber bundles, the craniomedial and caudolateral bands. Throughout the normal range of motion, these bands accept varying stress loads depending on the stage of motion. With the stifle in moderate flexion, excessive stifle internal rotation stretches the cranial cruciate ligament under the medial femoral condyle, causing mechanical trauma to the ligament.

The medial and lateral collateral ligaments of the stifle joint are substantial and important to joint stability. The medial meniscus is attached to the tibia cranially and caudally, and to the medial collateral ligament. The lateral meniscus is attached to the tibia cranially and the femur caudally (through the meniscofemoral ligament) and is separated from the lateral collateral ligament. Although the menisci move with flexion and extension, the more fixed medial meniscus is less accommodating than the lateral if subluxation occurs with loss of the cranial cruciate ligament. Therefore the medial meniscus is often damaged in cases of cranial cruciate rupture.

The fibrous extensor retinaculum secures the patella. The contribution from the fascia lata to the extensor

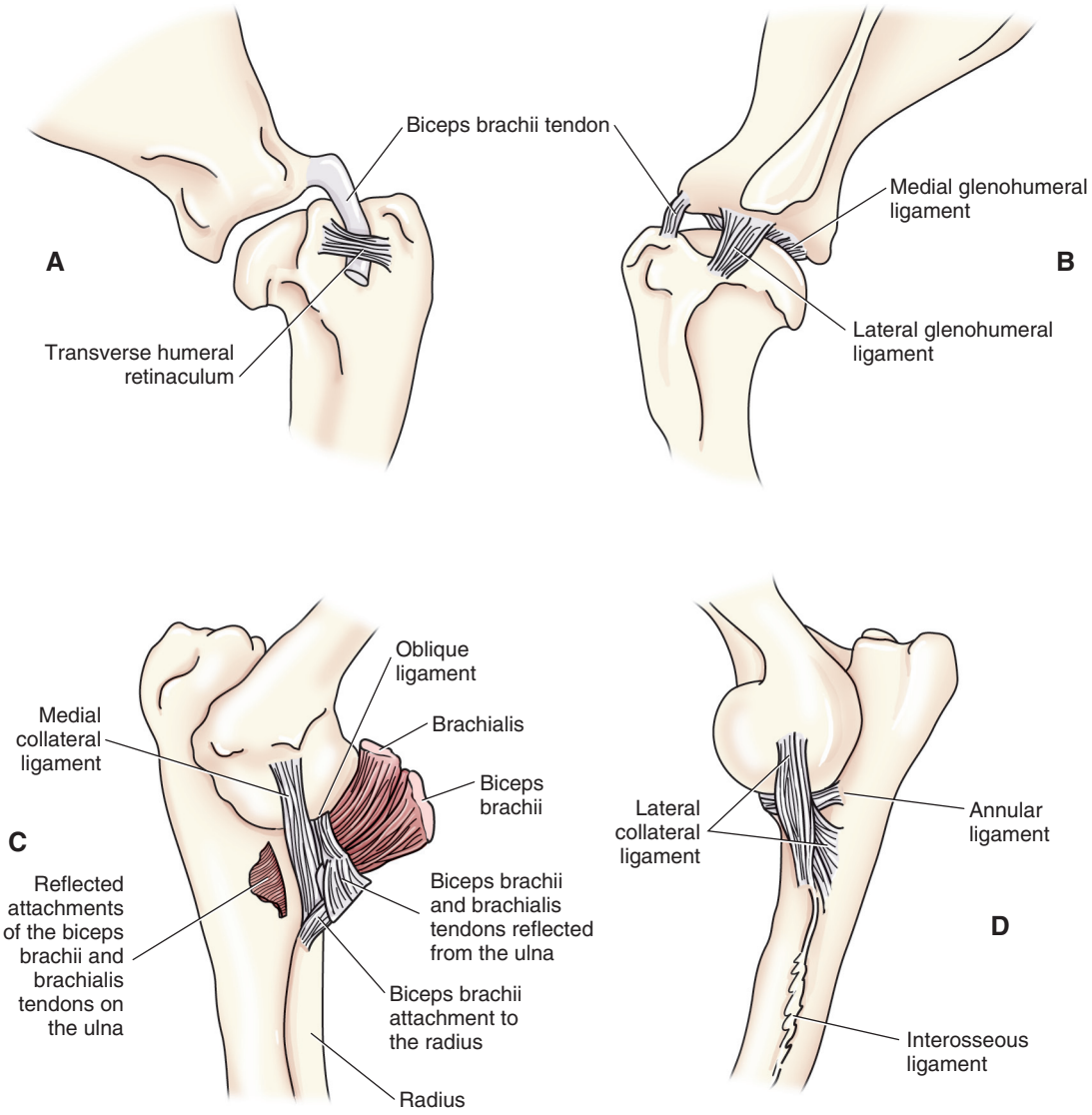


Figure 5-37 Ligaments of the medial (*A*) and lateral (*B*) left shoulder and medial (*C*) and lateral (*D*) left elbow in the dog. (Adapted from Evans HE: *Miller's anatomy of the dog,* ed 4, Philadelphia, 2013, WB Saunders.)

retinaculum is particularly important in holding the patella in the trochlea. Femoropatellar ligaments are mildly thickened regions of the fibrous capsule and are observed only in large dogs. The tendon of the popliteus muscle separates the lateral collateral ligament from the lateral meniscus. The patellar ligament is a strong support between the patella and tibial tuberosity. There is no tendon from the quadriceps muscle to the patella. The quadriceps is attached to parapatellar fibrocartilage.

There are three joint or synovial pouches in the stifle, two femorotibial joint pouches, and one retropatellar joint pouch. The femorotibial joint pouches incorporate the articulations of the sesamoids of the gastrocnemius muscle, but exclude the cruciate ligaments. The lateral femorotibial pouch continues distally through the extensor groove as the tendon sheath for the tendon of the proximal long digital extensor muscle and also surrounds the tendon of the proximal attachment of the popliteus muscle. The tibiofibular joint has ligamentous support from the ligament of the fibular head, the interosseous membrane, and the cranial tibiofibular ligament.

Medial and lateral collateral ligaments of the tarsocrural joint provide stability to the tarsus. There are long and short portions of the collateral ligaments. Plantar ligaments are more developed and substantial in the dog than in the human because of the upright weight-bearing stance in humans. Ligaments in the hindpaw maintain what is described as a *rigid tarsocrural area* in dogs. The tendons coursing into the hindpaw are secured by the fascia pedis, which forms retinacula to stabilize tendons and superficial muscles of the hindpaw and secure them to all the projecting bones in the hindpaw.

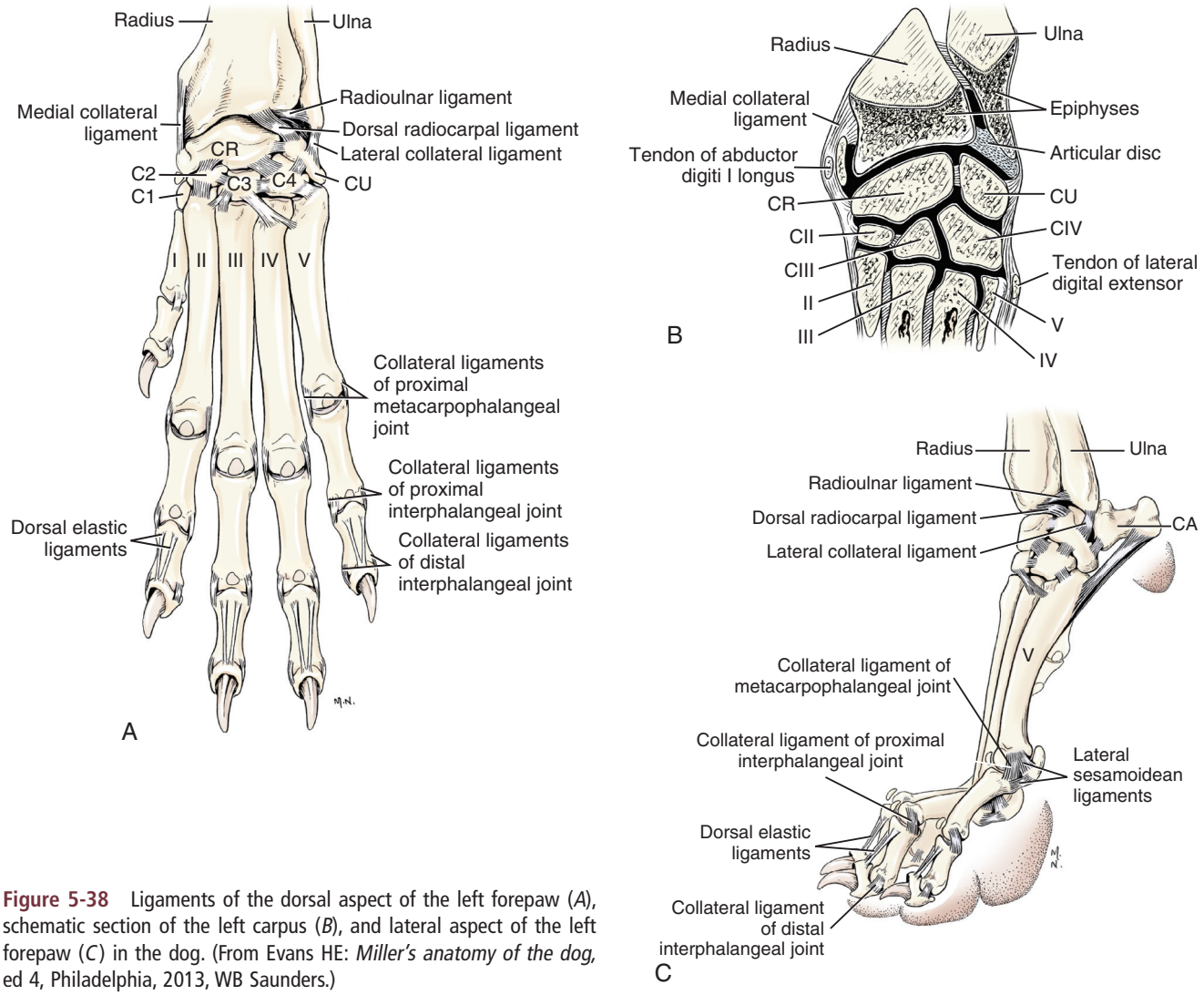


Figure 5-38 Ligaments of the dorsal aspect of the left forepaw (*A*), schematic section of the left carpus (*B*), and lateral aspect of the left forepaw (*C*) in the dog. (From Evans HE: *Miller's anatomy of the dog,* ed 4, Philadelphia, 2013, WB Saunders.)

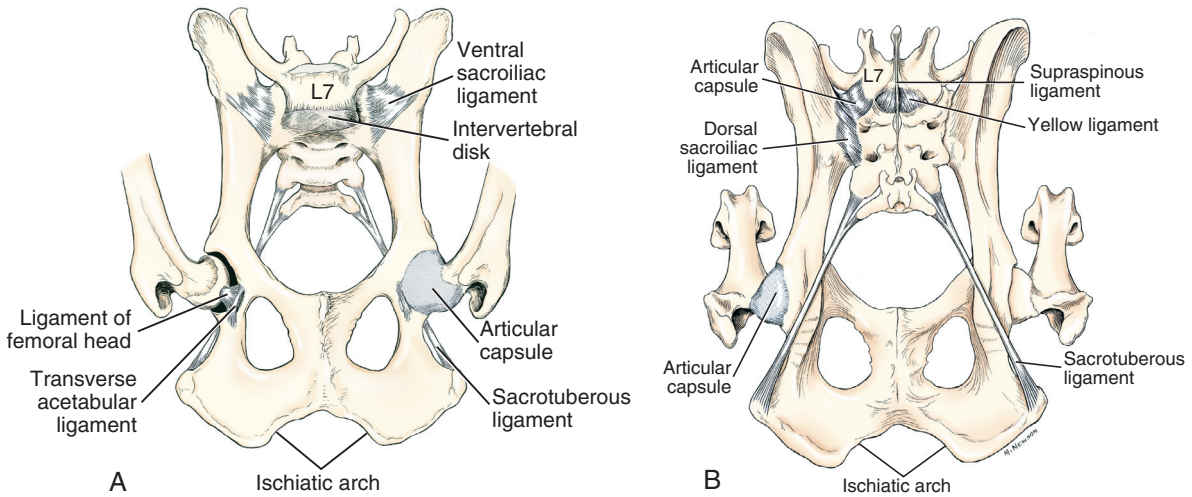


Figure 5-39 Ligaments of the hip and pelvis from a ventral (*A*) and dorsal (*B*) view. (From Evans HE: *Miller's anatomy of the dog,* ed 4, Philadelphia, 2013, WB Saunders.)

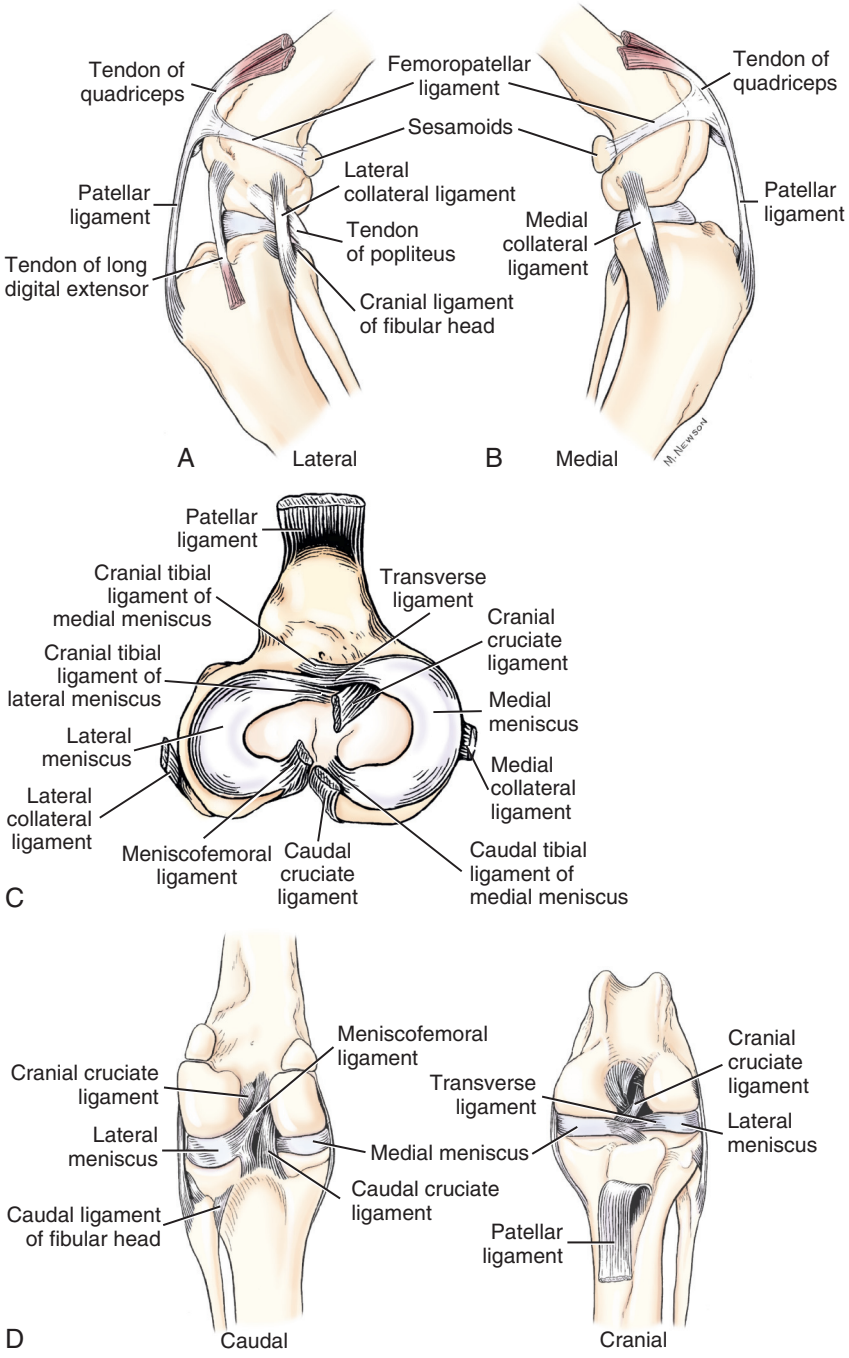


Figure 5-40 Ligaments of the left stifle in the dog from a lateral (*A*), medial (*B*), dorsal (*C*), caudal (*D*), and cranial (*E*) view. (From Evans HE: *Miller's anatomy of the dog,* ed 4, Philadelphia, 2013, WB Saunders.)

Spine

Figures 5-11, 5-12, 5-32, and 5-42 display ligament attachment sites, locations, and lines of direction for some ligaments of the spine. Spinal ligaments are extensive. Strong ligamentous support in the spine is thought to decrease the risk of disk herniation. The ligamentum nuchae, or nuchal ligament, is attached at the cranial and caudal extents of the cervical spine. This strong ligament acts as a tension band to oppose flexion of the head and neck and helps to hold the dog's head up with passive force, sparing muscle activity. This allows the dog relief from muscle fatigue.

General Nerve Supply

Forelimb

Figures 5-43 to 5-45 show the main nerves of the forelimb. The brachial plexus innervates the forelimb. It is formed by ventral rami C6 through T2. Sometimes C5 contributes to the brachial plexus. The nerves appear along the ventral border of the scalenus and enter the forelimb through the axillary space. The nerves of the canine brachial plexus include the suprascapular (C6-C7), subscapular (C6-C7), thoracodorsal (C6-C8), brachiocephalic (C6-C7), lateral

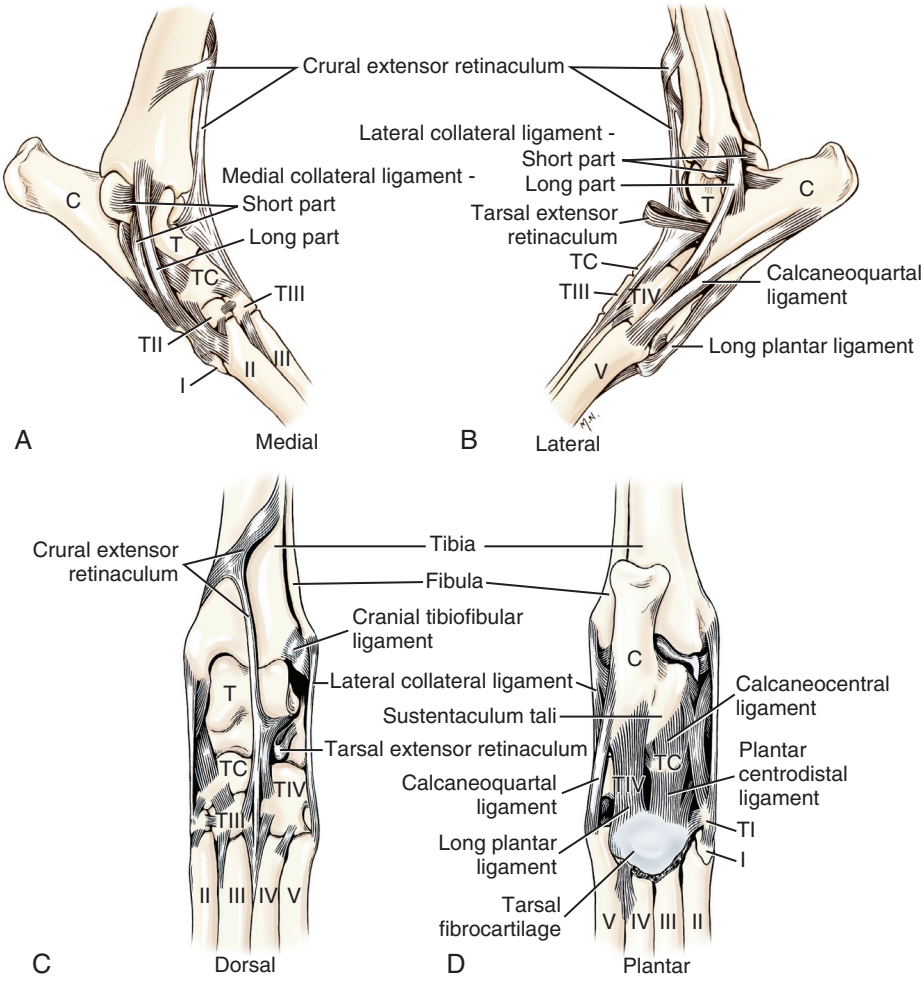


Figure 5-41 Ligaments of the left hindpaw in the dog from a medial (*A*), lateral (*B*), dorsal (*C*), and plantar (*D*) view. (From Evans HE: *Miller's anatomy of the dog,* ed 4, Philadelphia, 2013, WB Saunders.)

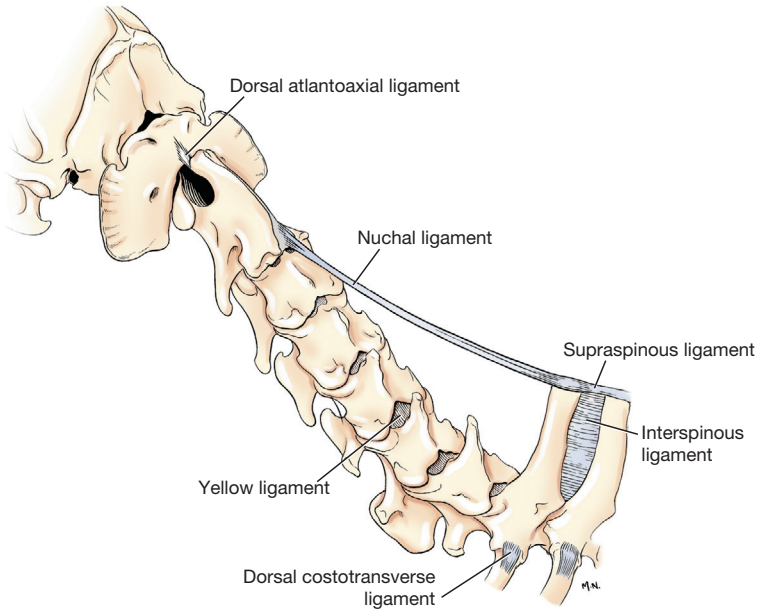


Figure 5-42 Nuchal ligament. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

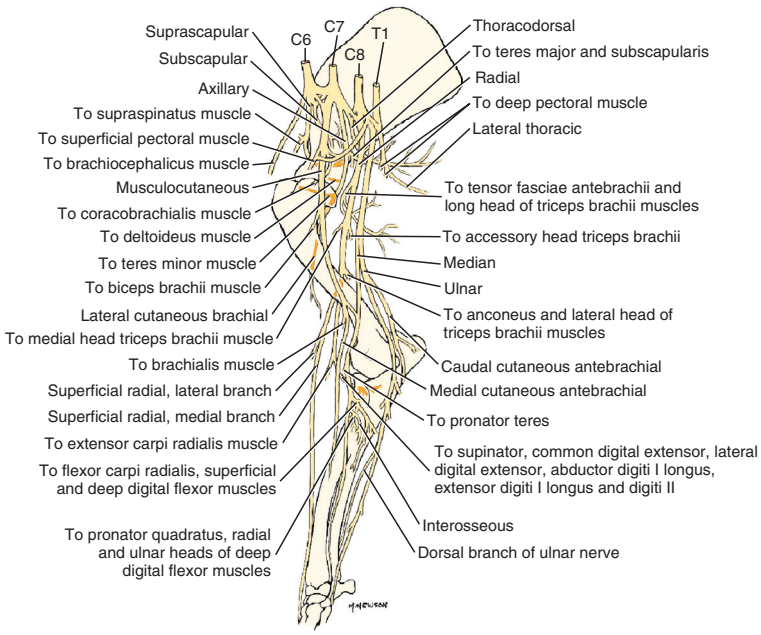


Figure 5-43 Main nerves of the right arm and forearm, medial aspect. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

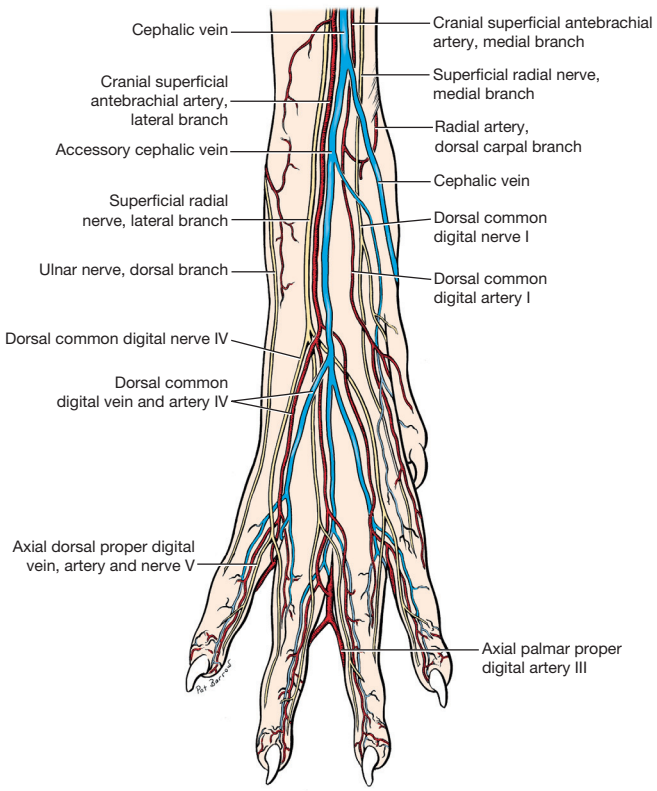


Figure 5-44 Nerves and arteries of the right forepaw, dorsal aspect. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

thoracic (C8-T1), caudal pectoral (C6-C8), musculocutaneous (C6-C8), axillary (C6-C8), radial (C6-T2), median (C7-T1), and ulnar (C8-T2) nerves. Innervations of muscles of the forelimb are included in Web Appendix 1.

Autonomous zones are areas of skin or cutaneous sensation that are innervated by only one peripheral nerve. Main front-limb autonomous zones of the brachial plexus are the regions of the axillary, musculocutaneous, radial, median, and ulnar nerves. Autonomous zones of the front limb and cranial trunk are displayed in Figure 5-46.

Hindlimb

Figures 5-47 to 5-49 show the main nerves of the hindlimb. The lumbosacral plexus innervates the hindlimb. This plexus can be divided into lumbar and sacral plexuses; however, there are always communications between the two plexuses. The plexus consists of ventral rami from the caudal five lumbar, or L, nerves and the three sacral, or S, nerves. There are seven lumbar nerves and three sacral nerves. The lumbosacral trunk is the largest and most important part of the lumbosacral plexus and becomes the sciatic nerve outside the pelvis.

The lumbar plexus is generally formed by ventral rami from L3-L5, sometimes L2-L6. The nerves of the lumbar plexus include the ilioinguinal from L3, genitofemoral from L3 and L4, lateral cutaneous femoral from L3 and L4, femoral from L4-L6, saphenous from L4-L6, and obturator from L4-L6 nerves. Innervations of the rear limb muscles are included in Web Appendix 1.

The sacral plexus is formed by ventral rami from L6-S3. The nerves of the sacral plexus that relate to the hindlimb include the cranial gluteal from L6-S1; caudal gluteal from L7 (S1); caudal cutaneous femoral from S1-S2[21]; sciatic from

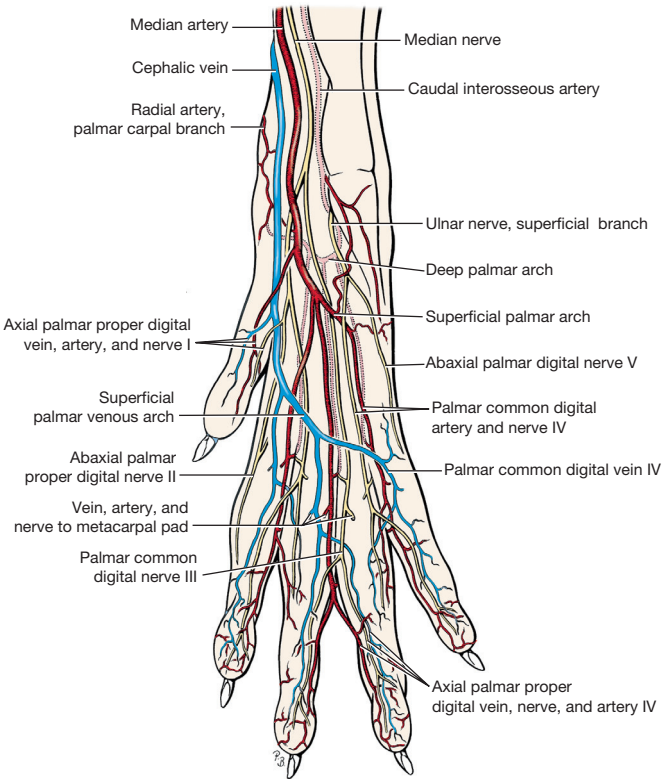


Figure 5-45 Nerves and arteries of the right forepaw, palmar aspect. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

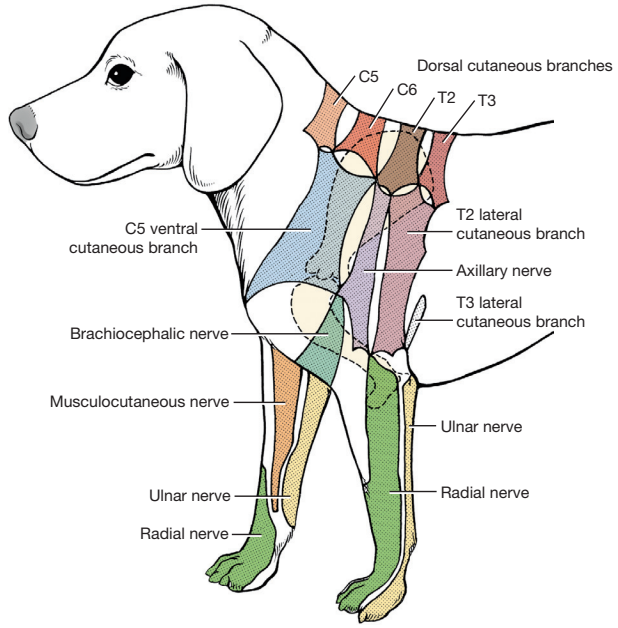


Figure 5-46 Autonomous zones of cutaneous innervation of the cranial trunk and forelimb. (After Kitchell RL et al: Electrophysiologic studies of cutaneous nerves of the thoracic limbs of the dog. *Am J Vet Res* 41:61-76,1980. In Evans HE, deLahunta A: *Miller's guide to the dissection of the dog,* ed. 7, Philadelphia, 2010, WB Saunders.)

L6-S1; common, superficial, and deep peroneal from L6-L7; tibial from L6-S1; proximal caudal cutaneous sural from L6-S1; and lateral cutaneous sural from L6-L7. In the hindpaw the dorsal metatarsal nerves are continuations of the deep peroneal nerve, and the medial plantar and lateral plantar nerves are the distal continuations of the tibial nerve. Innervations of muscles of the hindlimb are included in Web Appendix 1.

Main hindlimb autonomous zones are the regions of distribution of the genitofemoral, femoral, lateral femoral cutaneous, saphenous, caudal cutaneous femoral, sciatic, tibial, and peroneal nerves. Autonomous zones of the hindlimb and caudal trunk are displayed in Figure 5-50.

Trunk and Neck

The superficial muscles of the dorsal and lateral neck and trunk are innervated by the brachial plexus for muscles near the forelimb and by the lumbosacral plexus for muscles near the hindlimb. Most of the deep musculature of the neck and trunk is innervated segmentally by dorsal rami of spinal nerves (see Web Appendix 1). These deep muscles on the dorsal neck and trunk are epaxial muscles; that is, they developed in the dorsal neck or trunk area and received muscular innervation locally from the dorsal rami. The advantage of the segmental innervation from many nerves is that there is less chance of a strength deficit from damage to any one nerve.

The hypaxial muscles of the lateral and ventral abdominal wall developed in the lateral and ventral abdomen and received innervation from the ventral rami (i.e., the branch

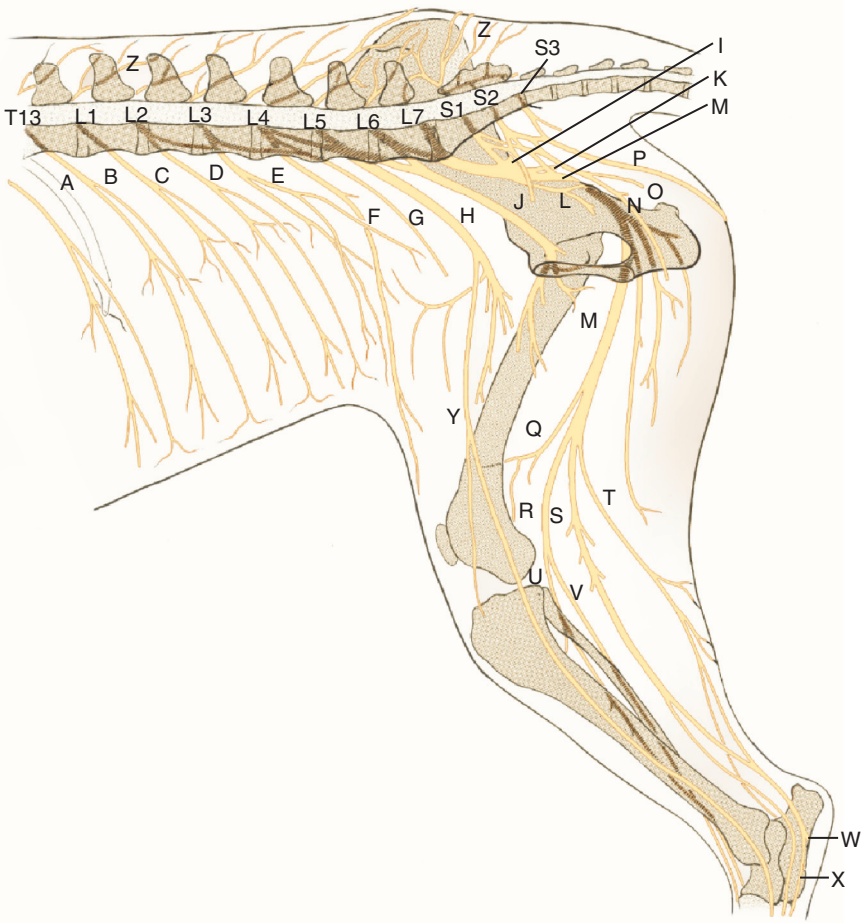


Figure 5-47 Schematic medial view of lumbar and sacral nerves. (From Evans HE: *Miller's anatomy of the dog,* ed 4, Philadelphia, 2013, WB Saunders.)

A. Thirteenth thoracic nerve, ventral branch
B. Cranial iliohypogastric nerve
C. Caudal iliohypogastric nerve
D. Ilioinguinal nerve
E. Lateral cutaneous femoral nerve
F. Genitofemoral nerve
G. Femoral nerve
H. Obturator nerve
I. Cranial gluteal nerve
J. Pelvic nerve
K. Caudal gluteal nerve
L. Nerve to mm. obturator internus, gemelli, and quadratus femoris
M. Ischiatic nerve
N. Pudendal nerve
O. Perineal nerve
P. Caudal cutaneous femoral nerve
Q. Lateral cutaneous sural nerve
R. Common fibular nerve
S. Tibial nerve
T. Caudal cutaneous sural nerve
U. Deep fibular nerve
V. Superficial fibular nerve
W. Lateral plantar nerve
X. Medial plantar nerve
Y. Saphenous nerve
Z. Dorsal branches of lumbar and sacral nerves

of the spinal nerve that traveled laterally and ventrally and innervated the local structures).

The spinal nerves exit between the vertebrae through the intervertebral foramina. All of the spinal nerves, except the cervical nerves, exit the intervertebral foramen caudal to the vertebra of the same number. In the cervical region, there are eight cervical nerves and seven cervical vertebrae. C2-C7 exit the intervertebral foramen cranial to the vertebra of the same number. C1 emerges through the lateral vertebral foramen of the arch of the atlas. C8 exits the intervertebral foramen caudal to C7.

Neck and trunk autonomous zones are circumferential strips of skin sensation around the dorsal neck and trunk. Because of extensive overlap in cutaneous sensation, a sensory deficit does not often occur unless a minimum of two consecutive spinal nerves are damaged.

General Blood Supply

Forelimb

Primary blood vessels to the dog's forelimb are the subclavian, axillary, brachial, common interosseous, and median arteries (Figure 5-51). The axillary artery and branches and the external thoracic, lateral thoracic, subscapular, and cranial and caudal circumflex humeral supply the scapular and shoulder area. The brachial artery and branches, deep brachial, bicipital, collateral ulnar, superficial brachial,

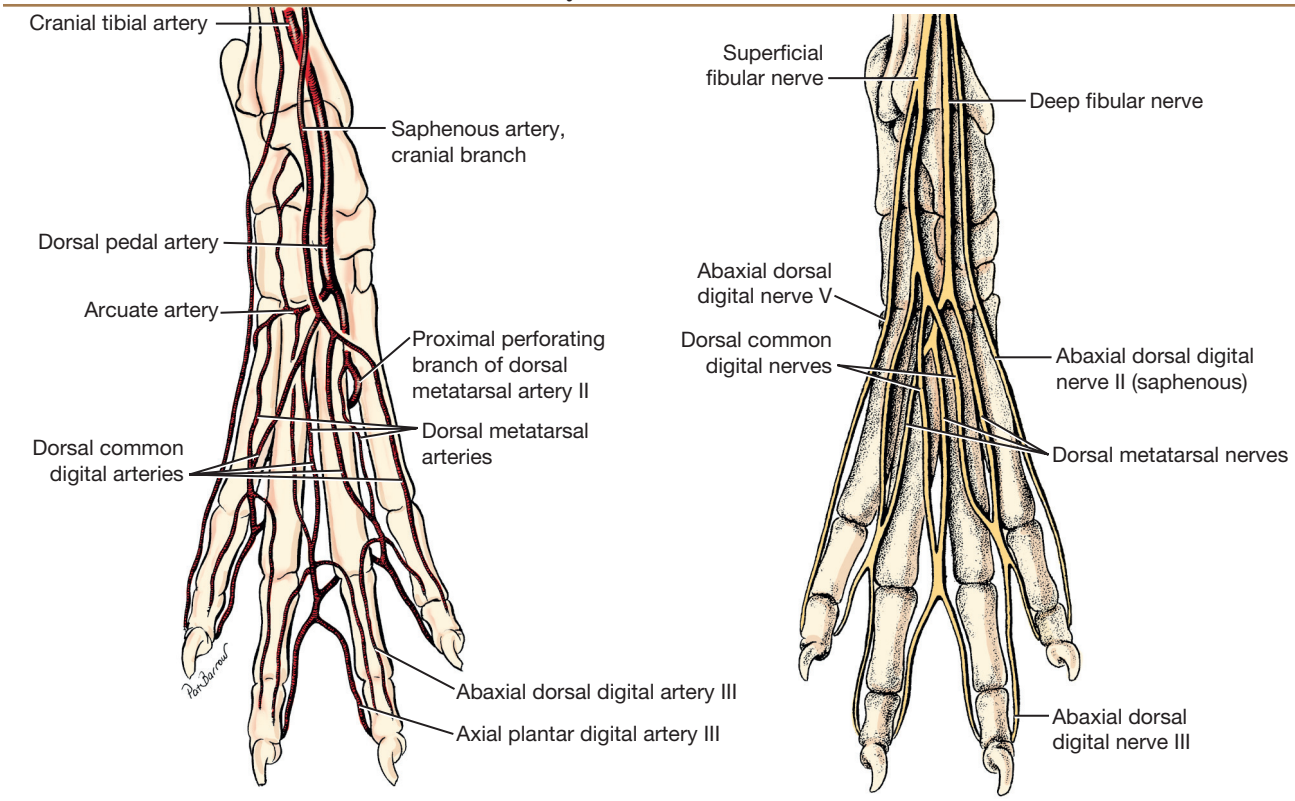


Figure 5-48 Nerves and arteries of the right hindpaw, dorsal aspect. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

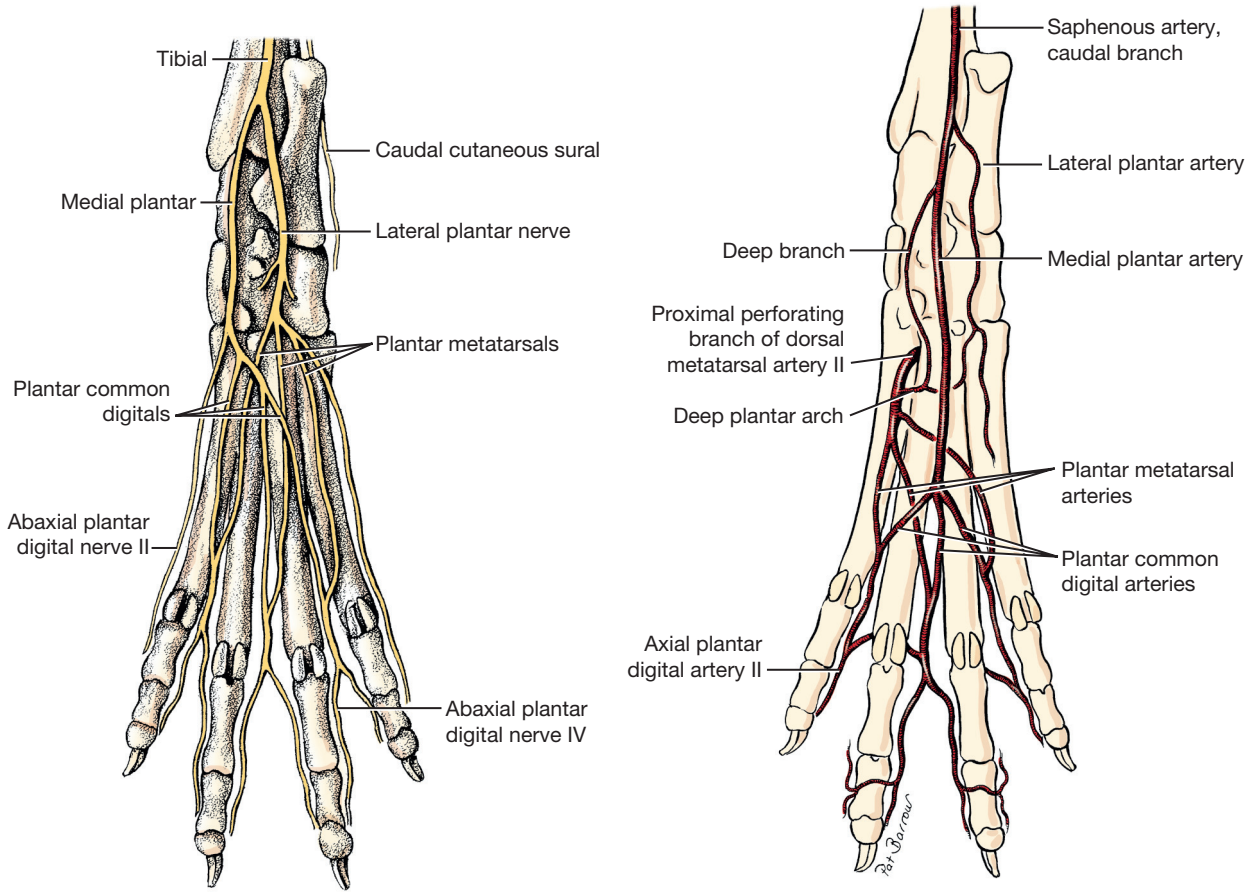


Figure 5-49 Nerves and arteries of the right hindpaw, plantar aspect. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

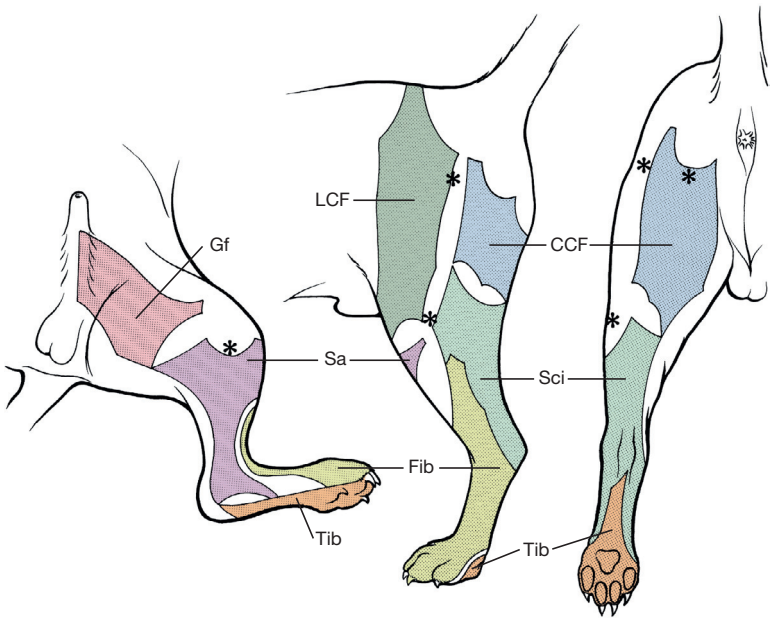


Figure 5-50 Autonomous zones of cutaneous innervation of the caudal trunk and hindlimb. *Asterisks* indicate palpable bony landmarks (medial and lateral tibial condyles, greater trochanter, and lateral end of the ischiatic or ischial tuberosity). The sciatic nerve autonomous zone is for lesions proximal to the greater trochanter and includes the zones for the peroneal and tibial nerves. For sciatic nerve lesions caudal to the femur, the autonomous zone varies, depending on how many of its cutaneous branches are affected. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

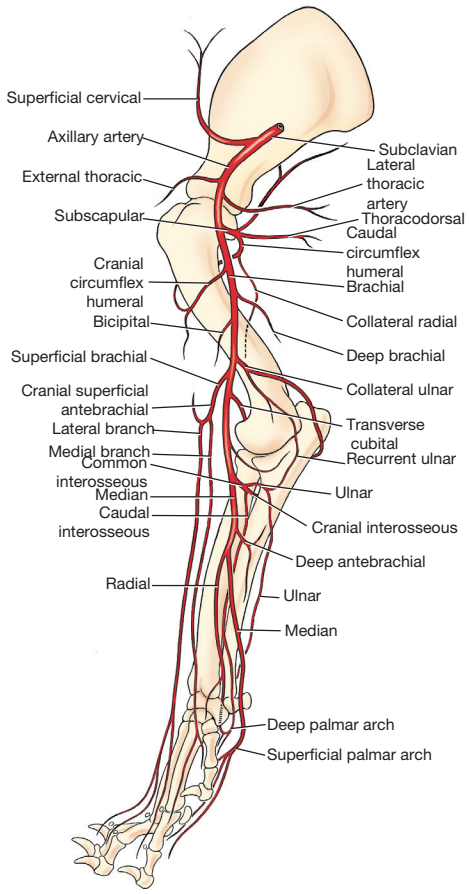


Figure 5-51 Arteries of the right forelimb, medial view. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

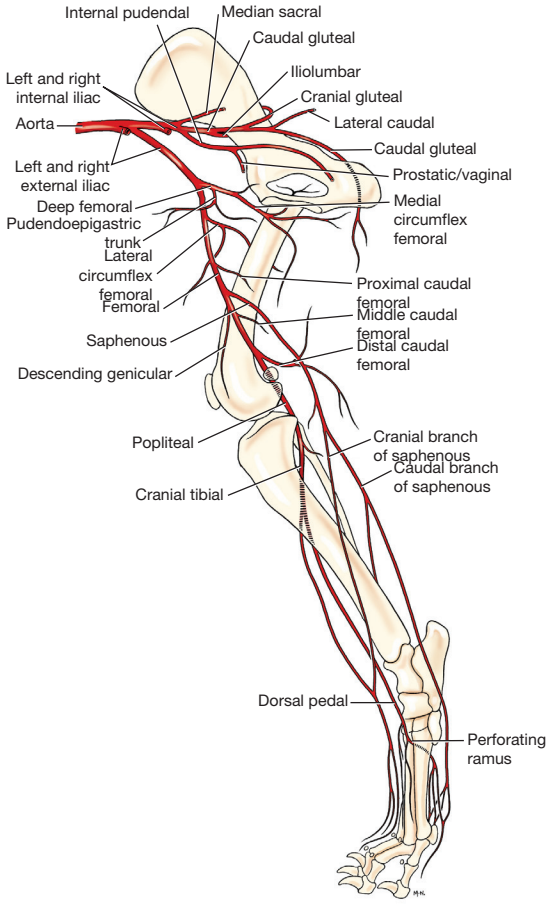


Figure 5-52 Arteries of the right hindlimb, medial view. (From Evans HE, de Lahunta A: *Miller's guide to the dissection of the dog,* ed 7, Philadelphia, 2010, WB Saunders.)

and common interosseous supply the arm and forearm. Common interosseous branches are the ulnar, cranial, and caudal interosseous arteries, which supply the deep forearm. The median artery, a continuation of the brachial artery, has two branches, the superficial palmar arch, which is the distal continuation of the median artery, and the radial artery. Arterial pulses are not easily palpable in the front limb and are not commonly used to assess cardiovascular function.

Hindlimb

The abdominal aorta divides into the common iliac arteries. The two branches of the common iliac arteries are the internal iliac artery, supplying the pelvic area, and the external iliac, which supplies the lower limb (Figure 5-52). The internal iliac artery supplies the caudal thigh via the caudal gluteal artery. The external iliac branches to the deep femoral, which gives rise to the medial circumflex femoral and the femoral arteries. The femoral artery gives rise to the lateral circumflex femoral, saphenous, descending genicular, distal caudal femoral, and popliteal arteries. Arterial pulses are palpable in the femoral artery in the inguinal region. From the caudal stifle area, the femoral artery becomes the popliteal, which divides into the cranial tibial, which continues as the dorsal pedal and arcuate arteries supplying the dorsal hindpaw, and the caudal tibial or posterior tibial arteries. The dorsal pedal artery is the principal blood supply to the hindpaw. The saphenous artery from the femoral sends genicular branches to the stifle before dividing into a superficial and caudal branch. The superficial branch anastomoses with the superficial branch of the cranial tibial artery to form a series of dorsal metatarsal, common digital, and axial and abaxial proper dorsal digital arteries coursing between the toes. The caudal branch of the saphenous artery forms the larger medial and a smaller lateral plantar arteries, and from these are formed a series of plantar common digital arteries. A perforating branch of dorsal metatarsal artery II, which is a branch of the arcuate artery, forms the deep plantar arch, from which form the plantar metatarsal arteries. The plantar metatarsal arteries join the plantar common digital arteries to form axial plantar digital arteries, which travel on the axial sides of the toes.

The cranial branch of the lateral saphenous vein is commonly a site for venipuncture. It is located on the dorsal tarsus and courses proximocaudally along the lateral surface of the leg.

Palpation of the Dog

Structures that are sufficiently superficial may be palpated. Palpation of structures, linked with knowledge of location of structures and the variation of feel with pathologic findings, is an important skill in accurate palpation. Palpation is more difficult in some dogs, depending on the type and thickness of hair, skin, and subcutaneous fat. Structures that can be palpated in dogs are numerous. Almost all figures in this chapter illustrate some palpable structures.

Forelimb

Bony Landmarks

The bony landmarks that are easily palpated include the following:

- *Scapula:* Borders, spine, acromion
- *Humerus:* Greater tubercle, intertubercular groove, deltoid tuberosity, medial and lateral epicondyles
- *Radius:* Head, medial styloid process
- *Ulna:* Olecranon, lateral styloid process

The medial coronoid process is usually covered by too much soft tissue to be palpable, but the location can be painful on palpation if the coronoid is fragmented.

- *Carpus and digits:* Accessory carpal bone, metacarpals, phalanges, metacarpophalangeal and interphalangeal joints, metacarpal pad in relation to the metacarpophalangeal joints, digital pads in relation to the distal interphalangeal joints

Joints

Joint motion can be palpated and observed with flexion and extension of the shoulder, elbow, carpus, and the metacarpophalangeal and interphalangeal joints.

Muscles and Ligaments

The forelimb muscles to be palpated include the following:

- *Scapulothoracic:* Trapezius, rhomboideus
- *Glenohumeral:* Superficial pectoral, deep pectoral, brachiocephalicus, latissimus dorsi, supraspinatus, infraspinatus, deltoideus, long head of triceps, biceps brachii tendon in the intertubercular groove, biceps brachii medially and brachialis laterally on the distal cranial humerus.
- *Elbow:* Triceps brachii and tendon of insertion
- *Forepaw:* Flexor muscles on the medial humeral epicondyle, distal attachments of the flexor carpi ulnaris and ulnaris lateralis on the accessory carpal, flexor carpi radialis as it tightens with carpal extension, extensor muscles, tendons of the extensor carpi radialis and the common digital extensor
- *Digit:* Tendons of the superficial digital flexors at the carpal canal, and digital flexor tendons and extensor tendons as groups
- *Ligaments:* Although the medial and lateral collateral ligaments of the elbow, annular ligament, short radial collateral, and short ulnar collateral ligaments of the carpal joint are not palpable, their integrity can be evaluated by applying controlled stresses to the joints.

Nerves

The ulnar nerve may be palpable caudal to the medial epicondyle. The median nerve is located just cranial to the

humeral medial epicondyle where it is accessible for nerve stimulation but is not distinctly palpable.

Arteries

The brachial artery is located just cranial to the humeral medial epicondyle but may not be palpable. The median artery passes deep to the flexor carpi radialis at the carpal joint, where the pulse may be palpable.

Veins

The cephalic vein becomes prominent for palpation with compression of the cranial aspect of the proximal antebrachium.

Lymph Nodes

The superficial cervical lymph nodes can be palpated cranial to the shoulder.

Hindlimb

Bony Landmarks

The bony landmarks to be palpated include the following:

- *Pelvis:* Iliac crests, sacrum, cranial dorsal and ventral iliac spines, ischiatic tuberosities, ischiatic arch
- *Femur:* Greater trochanter, trochlea of the femur and patella that articulates with the trochlea, femoral condyles
- *Tibia:* Tibial condyles, tibial tuberosity, cranial border, medial side of the body of the tibia, medial malleolus
- *Fibula:* Head of the fibula, lateral malleolus
- *Tarsus:* Trochlea of the talus and lateral ridge, calcaneus, phalanges, metatarsal pad in relation to the metatarsophalangeal joints, digital pad in relation to the distal interphalangeal joints

Cartilage

The parapatellar fibrocartilages sometimes can be palpated.

Joints

Joint motion can be palpated and observed in the hip, stifle, tarsocrural, metatarsophalangeal, and interphalangeal joints.

Muscles and Ligaments

The hindlimb muscles to be palpated include the following:

- *Hip:* Superficial gluteal, middle gluteal, sartorius, biceps femoris, semitendinosus, semimembranosus, gracilis, and pectineus

  In the caudal thigh, the biceps femoris, semitendinosus, semimembranosus, and gracilis are palpated in sequence from lateral to medial.
- *Stifle:* Vastus medialis, rectus femoris, vastus lateralis, biceps femoris, semitendinosus, semimembranosus, medial and lateral gastrocnemius and their associated sesamoid bones (fabellae), gracilis
- *Tarsus:* Craniolateral muscle group that forms the tarsocrural flexors and digital extensors, gastrocnemius muscles, common calcaneal tendon, superficial digital flexor tendon, and portions of the biceps femoris, semitendinosus, and gracilis tendons
- *Digits:* Digital flexors and extensors as groups, digital flexors in the caudal leg and plantar tarsus
- *Ligaments:* The medial collateral and lateral collateral ligaments of the stifle and tarsocrural joints, and the patellar ligament, are palpable. The extensor retinaculum may be palpable.

Nerves

The common peroneal nerve is palpable on the lateral surface of the proximal fibula. The tibial nerve is palpable cranial to the common calcaneal tendon and proximal to the tarsus.

Arteries

The femoral artery is palpated in the area of the medial femoral triangle and just cranial to the pectineus muscle. A pulse is palpable where the cranial branch of the saphenous artery typically crosses the medial side of the middle third of the tibia. The dorsal pedal artery can be palpated on the dorsal tarsus, often between the proximal ends of the second and third metatarsal bones.

Veins

The lateral saphenous vein is palpable and is a common site for venipuncture.

Lymph Nodes

The popliteal lymph node, between the distal ends of the biceps femoris and the semitendinosus, is palpable.

Trunk

Bony Areas

Palpable bony areas of the trunk include spinous processes of thoracic and lumbar vertebrae, transverse processes of the lumbar vertebrae, wings or lateral masses of atlas, spine of the axis, transverse processes of C3-C6, T13 spinous process from location of rib 13, cranial dorsal iliac spine, rib expansion with breathing, iliac crests and L7 spinous process between the iliac crests, sternum from manubrium to the xiphoid process, ribs.

Joints

Joint motion may be palpated and observed during intervertebral joint motion.

Muscles

The cervical and trunk extensor muscle mass and the rectus abdominis muscle are palpable.

Soft Tissue

The lumbosacral space caudal to the last lumbar spinous process is palpable.

Nerves

No nerves are readily palpable in the neck and trunk.

Arteries

The heartbeat can be felt over the left ventral thorax, and the pulse can be felt from the carotid artery. No veins are palpable in the neck and trunk, but with compression at the thoracic inlet, the external jugular vein is palpable.

Lymph Nodes

The inguinal lymph nodes are palpable.

Summary

This chapter presents canine anatomy in the context of association learning and the relevant anatomy for movement rehabilitation. Some of the relevant biomechanics that enhance association learning have been included. Knowledge of relevant anatomy and biomechanics is important in understanding movement function and dysfunction in animals.

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6 Tissue Healing: Tendons, Ligaments, Bone, Muscles, and Cartilage

Andrea Henderson and Darryl Millis

Injury to tissue initiates a complex series of events involving many cellular and biochemical responses that ultimately result in wound healing. The series of events depends on the severity of the injury and the tissues involved. The goal is regeneration or repair of the injured or traumatized tissue. Bone generally restores tissue architecture by regeneration of the normal cell population while other connective tissues respond by repair of the injured area.[1] The wound repair response is generally described by three major phases: inflammation, reparative (proliferative or fibroblastic), and remodeling (maturation).[1-3] These phases of healing overlap, and their sequence and timing vary with the tissue involved and the severity of the injury. The focus of this chapter is the basic wound healing response as well as the specific healing patterns of bone, muscle, tendon, ligament, and cartilage.

General Wound Healing

Inflammatory Phase

Most tissues undergo an initial inflammatory phase that involves an acute vascular response followed by cellular infiltration.[1,3] The immediate vascular component of the response is centered around hemostasis in the wound. Blood vessel disruption within the wound allows extravascular movement of blood elements and exposure of subendothelial collagen to platelets, causing activation of the coagulation cascade.[1,3-5] The result is formation of a fibrin network to support the hemostatic plug and act as a scaffold for cellular infiltration.

The cellular aspect of the inflammatory phase follows the initial vascular response. The release of cytokines associated with the hemostatic plug and increased vascular permeability incites the chemotaxis of cells into the wound environment (Figure 6-1). Neutrophils are the first cells migrating into the area, appearing by 6 hours after injury and peaking over a 2- to 3-day period.[1,3,5] The roles of the neutrophil are initial debridement and phagocytosis of microorganisms, thereby minimizing the potential for infection. The extent of neutrophil action within the wound depends on the severity of the wound and degree of contamination. The contribution by neutrophils in a noncontaminated wound is not essential for normal wound healing.[1,5]

Macrophages appear within the wound approximately 24 to 48 hours after neutrophil migration.[1] The influx of macrophages into the wound appears key to the transition from the inflammatory phase to the reparative phase and appropriate wound repair.[1,3] They are central to five major functions: phagocytosis, wound debridement, matrix synthesis regulation, cell recruitment and activation, and angiogenesis.[3] Phagocytosis and wound debridement occur with the release of oxygen radicals, nitric oxide, and collagenases.[3] Working in conjunction with neutrophils, macrophages create an optimal environment for entering the reparative (fibroblastic) phase of wound healing. This activity is at a maximum in the first 3 to 4 days after injury. In addition, macrophages release a host of cytokines, growth factors, prostaglandins, and enzymes that subsequently activate and mediate angiogenesis and fibroplasia.[3] The macrophage appears to be the central figure in control of cellular and biochemical events in general wound healing.

The presence of macrophages also attracts and activates lymphocytes, whose function remains controversial.[1,3] Lymphocytes secrete lymphokines such as interferons and interleukins that stimulate fibroblast migration and collagen synthesis.[1,3,6] Their activity appears to peak approximately 6 days after injury.[1,5]

Reparative Phase

The reparative (proliferative or fibroblastic) phase of wound healing is characterized by the cellular response of endothelial cells and fibroblasts.[1,3] Fibroblast proliferation and migration predominate and are followed by matrix synthesis, including collagen, elastin, and proteoglycans. The fibroblasts begin appearing in the wound within 3 days of injury; however, there is a lag phase of 2 to 3 days before initiation of collagen production with maximal proteoglycan synthesis not occurring until 2 weeks after injury.[5] Matrix synthesis increases over the following few weeks with concurrent increases in tensile strength.[1]

In addition, endothelial cells adjacent to the wound begin to proliferate and form new capillaries that gradually migrate into the wound. The newly developing capillaries

The authors would like to acknowledge James (Ned) Williams for his work on the previous edition.

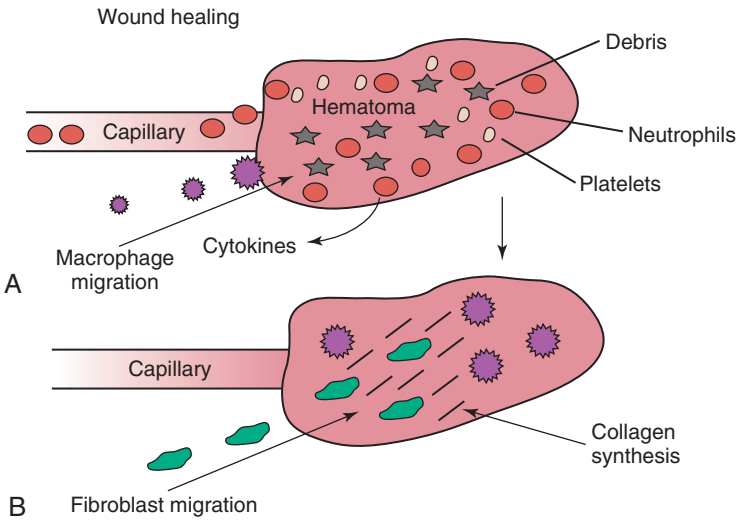


Figure 6-1 Inflammatory (*A*) and reparative (*B*) phases of wound healing.

Box 6-1 Healing Timeline: General

Inflammation
Immediate to 3-5 days

Repair
72 hr to 2 wk

Remodeling/Maturation
Occurs in 2-3 wk (max collagen deposition) to 1 yr

Healing/Strength Characteristics
Varies

Implications for Rehabilitation
N/A
Rates are generalizations of individual tissue healing. Results from each injury vary depending on the severity, chronicity, and age of the individual.

Box 6-2 Healing Timeline: Skin

Inflammation
N/A

Repair
Collagen stabilizes at approximately 21 days

Remodeling/Maturation
N/A

Healing/Strength Characteristics
Occurs in 21 days (20% normal strength) to 1 yr (70% normal strength)

Implications for Rehabilitation
Minimal effect

follow immediately behind the migrating fibroblast and collagen scaffold and continue until normal oxygen tension in the wound is restored.[5] This dense network of macrophages, fibroblasts, and neovascularization during the proliferative phase is generally recognized as granulation tissue.

Remodeling (Maturation) Phase

The remodeling phase is the final aspect of wound healing during which collagen fibers reorient parallel to lines of stress and strain and the fibers cross link in a stable formation.[7,8] This is the most important aspect of wound healing for connective tissues, because appropriate collagen deposition and alignment are critical to adequate tensile strength development in the repaired tissue. Although collagen deposition reaches a maximal point 2 to 3 weeks after injury, tensile strength continues to progressively increase over the course of approximately 1 year (Box 6-1 and Box 6-2). This period allows for removal of biomechanically inferior collagen fibers (type III) and replacement with the fibers suitable for the specific tissue involved (generally type I collagen). In addition, decreasing proteoglycan concentration leads to a decrease in water content, resulting in compression of the collagen fibers. As fibers become closer in proximity because of realignment and compression, increased surface area is available for cross-linking, subsequently increasing tensile strength.[5] Thus a proper equilibrium between collagenolysis and matrix accumulation becomes essential.

Bone Healing

Bone Structure

Bone is a dynamic tissue composed of 35% organic material and 65% mineral.[2,9-11] The organic portion comprises cellular constituents (osteocytes, osteoblasts, and osteoclasts) and matrix components. The osteocytes are cells of mature bone that remain within spaces (lacunae)

surrounded by lamellar bone and are responsible for normal mineral homeostasis. Osteoblasts are responsible for osteoid production, and osteoclasts function in bone resorption.[11,12] A fine balance of bone resorption and deposition is maintained for mineral homeostasis as well as continuous remodeling secondary to mechanical stresses placed on the bone in accordance with Wolff's law which states that a bone in a healthy individual will adapt to the forces under which it is placed.[12,13]

Bone matrix is composed of predominately type I collagen and ground substance containing glycosaminoglycans.[8,11,12] Calcium and phosphorus deposit in association with the collagen fibrils and serve as the primary reservoir for overall calcium homeostasis. Hydroxyapatite crystals of calcium and phosphorus are aligned with the collagen fibrils and provide the structural rigidity to bone.[11,12] This unique arrangement between collagen and mineral creates the viscoelastic property of bone with the crystal supplying resistance to compressive forces and the collagen providing tensile support.[12]

Blood supply to bone is primarily based on the nutrient artery that bifurcates into ascending and descending branches within the medullary cavity (Figure 6-2). In addition, there is vascular support from metaphyseal arteries and periosteal arterioles.[11] The metaphyseal vessels anastomose with the nutrient artery branches within the medullary canal to provide complete collateral circulation. The vascular flow is centrifugal, with elevated pressure in the medullary cavity driving blood toward the periosteum. The efferent system is a venous drainage system using periosteal venules and the medullary venous sinus. Cortical vessels flow through Haversian canals in which small vascular channels are surrounded by layers of lamellar bone.[9-11] These channels in the cortical bone provide perfusion to the osteocytes within their lacunae.[12] The periosteal arterioles are the least important contributors of the vascular supply in healthy intact bone and are primarily of concern in areas of soft tissue attachment. Injury to the bone results in disruption of the vascular supply to varying degrees depending on the severity of the fracture. If the endosteal supply remains intact, it will predominate during healing. However, if the endosteal supply is disrupted, as in most fractures, the periosteal support becomes extremely important, thus making careful maintenance and handling of soft tissue attachments crucial to healing (Box 6-3).[14]

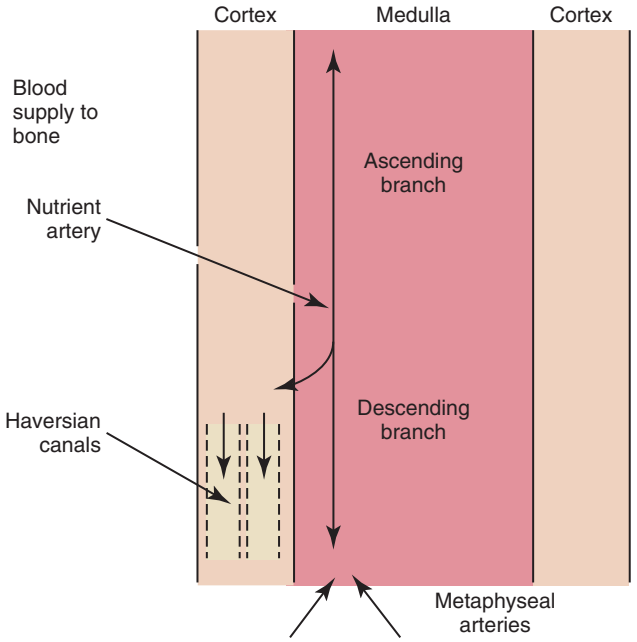


Figure 6-2 Blood supply to bone.

Primary Bone Healing

As discussed previously, bone is one of the few tissues that can undergo direct cellular regeneration to restore 100% of the original biomechanical properties. This can occur through two major types of healing, primary or secondary. Primary, or direct, bone healing occurs when there is a minimal fracture gap with rigid stability, and it proceeds by either contact or gap healing. Contact healing involves the direct formation of bone across a fracture line less than 0.1 mm wide by creating resorption cavities, or "cutting cones," through the Haversian canals parallel to the long axis of the bone (Figure 6-3).[9,10,15,16] Osteoclasts lead the front edge of the resorption cavity across the fracture line and remove bone and calcified matrix in preparation for osteoblastic activity immediately following. Finally, nutritional support is supplied by capillary loops following the advancing osteoblasts.[9,15,16] This progressive Haversian remodeling continues across the fracture line until the surrounding bone has been remodeled into new Haversian systems.

When the fracture line is greater than 0.1 mm but less than 0.5 mm, primary bone union can still occur through gap healing.[10,16] This distance cannot be directly crossed by a resorption cavity. As a result, lamellar bone produced from cells lining the medullary cavity and periosteum first fills the void in a transverse direction. Subsequently, resorption cavities cross the newly formed lamellar bone and again create new Haversian systems in the proper longitudinal direction.[10,16] In fracture repair, both gap and contact healing may occur in different portions of the same fracture line.[16] With primary bone healing, there is little radiographic callus formation, and the fracture line gradually disappears. Although direct healing entails direct formation of lamellar bone and remodeling of Haversian systems without intermediate fibrous or cartilaginous tissue, it does not reduce the time for fracture healing, and weeks to months may still be required before complete union.[15] In fact, early stability is generally decreased when compared with secondary healing, typically requiring longer periods before removal of stabilizing implants.[17] Radiographic evaluation is generally performed at 4- to

Box 6-3 Healing Timeline: Bone

Inflammation

Primary and secondary bone healing follow similar timelines. Immediate to 72 hr (hematoma and inflammation, secondary bone healing)

Repair

Radiographic healing (secondary): appearance of callus at 10-12 days, disappearance of fracture line within 30 days.[85] Disappearance of fracture line radiographically as early as 30 days with secondary bone healing. Primary bone healing takes longer.

Remodeling/Maturation

Woven bone development over mineralized callus. Complete remodeling of the callus 90 days after fracture repair for a simple fracture.[85] Primary bone healing (direct union, no callus formation) produces less early stability than secondary, and requires implants to be in place for a longer time than with secondary healing.[17]

Healing/Strength Characteristics

Regaining 100% tensile strength is possible because of regeneration. Healing and strength are faster with secondary bone healing and callous formation compared with primary bone healing. Rate of healing depends on presence of interfragmentary strain (faster at less than 2%), animal age, nature and location of the fracture (faster in sites with abundant cancellous bone and highly vascularized marrow), presence or absence of concurrent disease and surgical fixation method.[85] *Clinical union* defined as ability to withstand load so that fixation can be removed. In general, bone achieves clinical union according to the following timelines[17]:

- Puppies under 3 months: 2-4 wk
- Puppies 3-6 months: 4-12 wk
- Puppies 6-12 months: 5-8 wk to 3-5 mo
- Adult dogs (1 year of age or older): 7-12 wk to 5-12 mo

Rate is faster for IM pins and external skeletal fixation than for bone plating. Destabilize external fixators at approximately 4-6 wk in dogs.[20, 21]

Implications for Rehabilitation

Rehabilitation goals throughout healing have been suggested.[86] During inflammatory phase (immediately up to 2-3 days after injury), decrease pain, minimize soft tissue inflammation, maintain joint health/Range of motion (ROM) and establish safe, controlled mobility, depending on fracture stability. During repair phase (3 days to 4-6 wk after injury), small, gradually increasing cyclic loading to increase micromotion and improve callus formation with secondary healing; continued maintenance of joint health through ROM, active or passive.

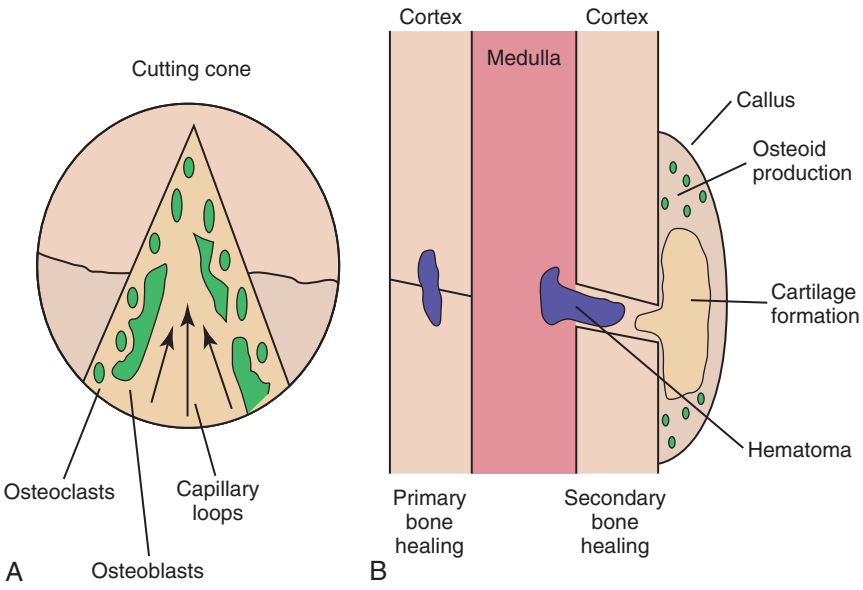


Figure 6-3 Bone healing. **A,** Primary bone healing with the formation of cutting cones. **B,** Secondary bone healing.

6-week intervals until the fracture line has resolved. The goal is to achieve recovery of function and early weight-bearing with implants employing interfragmentary compression so rehabilitation can begin at an early stage.

Secondary Bone Healing

Secondary, or indirect, bone healing occurs when there is less rigidity or absence of anatomic reduction such that the fracture gap is greater than 0.5 mm and is characterized by callus formation (see Figure 6-3).[16,18] The degree of callus formation is related to the amount of instability at the fracture site. In general, secondary bone healing proceeds through the same phases as basic wound healing.[9,10,19]

Similar to basic wound healing, hemostasis is the initial response at the time of injury, initiating the inflammatory phase. Immediate hemorrhage and hematoma formation

occur within the fracture gap in addition to the surrounding soft tissues that may be damaged. Destruction of the Haversian canals at the site of injury causes osteocyte death and necrosis of bone. The process proceeds with the migration of neutrophils followed by macrophages into the injured site for removal of necrotic debris. In addition, biochemical mediators such as bone morphogenetic proteins, platelet-derived growth factor, osteonectin, prostaglandins, and osteocalcin enter the fracture site to contribute to bone healing.[10] As the inflammatory response previously described subsides (24 to 72 hours), the repair process begins.[9,10,19]

The reparative phase begins with the surrounding tissue providing mesenchymal cells, which enter the fracture site to form osteoblasts, chondroblasts, and fibroblasts. During the initial 3 weeks, a fibrous and cartilaginous "soft" callus is formed by the deposition of types I, II, III, V, IX, and X collagen synthesized by mesenchymal cells. Types III and V collagen initially appear during the inflammatory phase, whereas types II and IX collagen peak during the cartilaginous aspect of the reparative phase 1 to 2 weeks after injury. Collagen type I is initially present at low levels in the first 2 weeks but becomes the predominant type by day 14.[8,13] The function of the soft callus is to unite the fracture ends and decrease interfragmentary motion and strain.[16] The fibrocartilage deposited is gradually transformed to bone by the process of endochondral ossification similar to that which occurs during physeal growth.[8,19] Chondrocytes progress to a hypertrophied stage followed by mineralization of the surrounding matrix. Invasion by capillaries brings cells that resorb the mineralized cartilage matrix, produce osteoid, and deposit bone. This transition from soft cartilaginous callus to hard bony callus is directly related to the blood supply available and the interfragmentary strain at the fracture gap. With high interfragmentary strain or poor blood supply, activity of osteoblasts and osteoid production is limited, and the callus remains as fibrocartilage.[10,16]

The remodeling or maturation phase proceeds following the transition from soft to hard callus. The fracture gap is bridged and stabilized with endosteal and periosteal callus consisting primarily of woven bone that must be remodeled to lamellar bone according to weight-bearing forces as directed by Wolff's law. In addition, the normal blood supply is reestablished. As the endosteal callus is gradually remodeled, the medullary cavity and its blood supply are restored. Similarly, as cortical bone is restored, the Haversian system is re-created and the normal centrifugal vascular pattern can return with concurrent resorption of the temporary extraosseous supply.[18,19]

Factors Affecting Healing

Biomechanical factors at the fracture site are important to healing. Wolff's law has a tremendous effect on bone deposition and resorption. The stress placed on the bone stimulates healing as long as the micromotion created does not exceed the acceptable interfragmentary strain for osteoid deposition.[18] If the mechanical load exceeds the strength of the reparative tissues, inhibition of bone healing or re-fracture occurs. Thus it is important that no or minimal motion between fracture fragments occurs early in the healing phase to allow adequate establishment of the extraosseous blood supply. After the vascular supply is established and callus formation has occurred, then controlled axial micromotion can stimulate appropriate bone remodeling. As such, destabilization of external fixator frames has been recommended at approximately 4 to 6 weeks following frame application to "dynamize" the fracture line.[20,21]

Electrical fields have been demonstrated in normal and injured bone and are related to either mechanical forces or bioelectric potentials.[22] Strain-related potentials occur secondary to mechanical loading of the bone and are related to piezoelectric activity resulting from collagen deformation and fluid flowing through space in the extracellular matrix.[10] Bioelectric potentials are generated by cellular functions associated with metabolic processes in the cell.[22] These electrical fields can be stimulated exogenously and have been demonstrated to favorably affect bone remodeling. Electrical stimulation can be provided by invasive or noninvasive methods. Invasive stimulation includes direct surgical implantation of a cathode at the site of stimulation and the anode placed in the surrounding soft tissue. Noninvasive stimulation can be accomplished by capacitive or inductive coupling of current to the appropriate site. With capacitive coupling, the electrodes are placed on the skin on opposite sides of the stimulation site, and electrical fields between 1 and 10 mV/cm are generated. Inductive coupling uses external electromagnetic coils to establish an electromagnetic field of time-varying or pulsed nature to create a secondary electrical field in the bone.[22] Currently, the mechanism of electrical stimulation of bone healing is not completely understood, but its use has been advocated with nonunion fractures.[23,24]

Alterations in endocrine balance can influence bone healing as well. Hormones such as parathormone, calcitonin, vitamin D, and thyroid may affect bone resorption and deposition.[10] Corticosteroids and diabetes mellitus can have inhibitory effects on bone healing.[10,25] In addition, there are numerous other miscellaneous factors that must be considered with respect to fracture healing, including patient age, fracture configuration, fracture location, and whether the site was grafted.[10,16] Young patients heal faster than geriatric individuals. The fracture configuration is also important because highly comminuted fractures or those with significant bone loss require more time to achieve bony union. Furthermore, fractures closer to the metaphysis in locations of high cancellous bone, or those that have been grafted, may have more rapid healing potential.

Efforts to enhance fracture healing continue along many pathways. The ability to stimulate bone healing with the use of growth factors is a future possibility, and research continues in the field of electrical stimulation. In addition, research has demonstrated potential advantages to using ultrasound and extracorporeal shockwave technology to enhance fracture healing.[24,26]

Muscle Healing

Muscle Structure

Skeletal muscle is composed of aggregations of muscle cells termed *fibers* (*myofibers*), consisting of multiple nuclei within each fiber (Figure 6-4). Each individual muscle belly is composed of numerous fibers bound together by connective tissue into multiple diffuse bundles (fascicles). A dense fibrous connective tissue sheath termed the *epimysium* surrounds the entire muscle. The *perimysium* is the connective tissue support surrounding each fiber bundle within the muscle belly and the *endomysium* represents sheets of connective tissue surrounding each independent muscle fiber.[27,28] Capillaries, nerves, and lymphatics traverse to the muscle fibers through the surrounding connective tissue sheaths, eventually reaching the endomysium, where a rich capillary network highly sensitive to sympathetic vasomotor tone is established around the individual myofibers.[29]

Each muscle fiber has an extensive subcellular structure. Myofibrils within the muscle fiber are composed of both thick (myosin) and thin (actin) filaments, troponin, and tropomyosin, which function to provide the contractile properties of the muscle fiber.[27,29] The myofibrils are surrounded closely by the sarcoplasmic reticulum, which plays a role in storage and release of calcium for muscle function.[29] Variations in the composition of the myofibers and sarcoplasmic reticulum result in different types of fibers (slow or fast twitch) for various muscular functions and fatigue resistance.

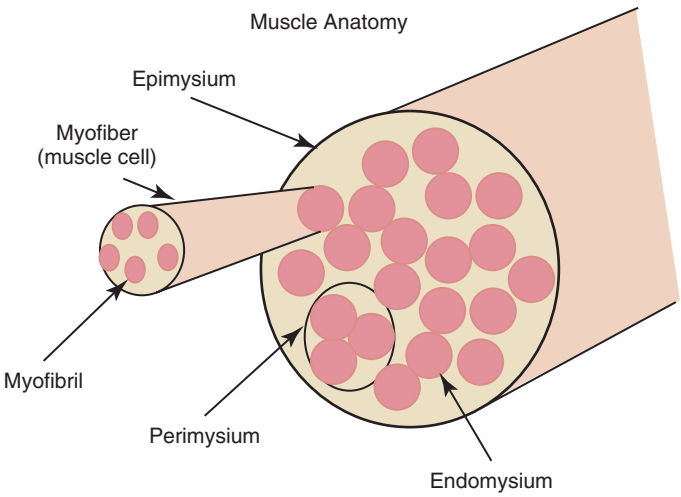


Figure 6-4 Anatomy of muscle connective tissue.

Muscle Injury and Healing

Injury to muscle occurs as a result of lacerations, contusions, ruptures, ischemia, and strains. A *strain* is described as an injury to the muscle or tendon with a tendency to occur near the myotendinous junction. Strains are described as grade 1 through 4, with grade 1 strains being disruption of a few muscle fibers and grade 4 being complete rupture of the muscle belly.[30] Often the cause of nonpenetrating injuries is forceful contraction of the muscle belly with concurrent passive extension.[28] Damage to the muscle results in tearing of the muscle fibers and disruption of the vascular or connective tissue support. The problem may be acute or chronic, and the severity may vary from mild injury to complete rupture.

Muscle healing progresses in the same basic pattern as general wound healing (Box 6-4). An initial period of hemostasis and inflammation precedes repair. Following

Box 6-4 Healing Timeline: Skeletal Muscle

Inflammation

Immediate to 72 hr (inflammation)

Repair

3 days (type III collagen) for scaffold for myofibers; normal collagen ratio > 6 wk

Remodeling/Maturation

Ratio of functional muscle fiber to scar tissue depends greatly on size of gap at site of injury

Healing/Strength Characteristics

Normal tissue regeneration is required for return to full function; occurs in minimum 4-6 wk.[28] Complete muscle lacerations near midbelly in rabbits showed final return to 50% of tension-producing ability and 80% of normal contraction shortening at 12 wk.[71] Partial tears have an improved outcome for tensile strength; return to 60% of tensile strength and normal contractile ability achieved with partial lacerations at 12 wk.[71]

Implications for Rehabilitation

Decreases in muscle contraction strength directly related to degree of injury and ingrowth of fibrous tissue. Rehabilitation depends on extent of injury (i.e., degree of strain, rupture). In mild strains, brief (3-5 day) immobilization followed by active muscle contractions in mobilization demonstrates better penetration of muscle fibers through the scar tissue and better muscle strength and size.[72] For muscle tears, immobilize for a minimum of 2-3 wk to protect repair, promote new collagen, and sustain remobilization stresses.[33,34] For rehabilitation of complete rupture, moderate activity can begin at 4-8 wk. At 8-12 wk, unrestricted activity may be allowed.[73]

hematoma formation, the edema and ischemia can result in necrosis of adjacent ruptured muscle fibers.[31] The initial inflammatory phase continues with neutrophil and macrophage removal of necrotic debris during the first 24 to 72 hours.[28,30] A distinct relationship between macrophage penetration and myofiber regeneration has been suspected; however, the essential component is reestablishment of the vascular supply to the injured fibers.[29] The repair phase follows and is a competitive process involving either regeneration of functional myofibers or production of fibrous scar tissue, depending on the severity of the injury and the size of muscle gap.[30] The repair by scar tissue production is not as desirable because of the higher chance of recurrent injury and a decrease in muscle contractile strength of approximately 50%.[30,32]

As in general wound healing, the repair phase continues with production of extracellular matrix by fibroblasts including proteoglycans, fibronectin, and collagen. Type III collagen appears within the injured site in the first 3 days and generally precedes the thicker type I collagen fiber production.[33] This extracellular matrix provides a scaffold for myofiber regeneration across the injury site.[30] Myoblastic stem cells from the surrounding wound environment (satellite cells) and mononuclear leukocytes migrate to the viable ends of myofibers and align across the gap, providing a route for myotubules to cross the injured site and repair the myofiber.[30] The alternative form of healing occurs when there is an inadequate source of myoblasts, inadequate vascularization, inadequate innervation, or excessive stress across the injured site creating a significant gap.[28,30] In this situation, excessive collagen deposition and fibroblastic activity lead to formation of fibrous tissue within the injured site. The result is a barrier to myofibers crossing the injury site and inadequate return to normal muscle function.

Timing of appropriate stress and motion at the injury site is important to the type of healing that occurs. For optimal muscle fiber healing and return to maximal function, mobility across the injury site should not begin until healing is well under way, such as in the reparative or early remodeling phases. During the reparative and remodeling phases, the muscle fibers can be aligned parallel to the line of action if appropriate stresses are allowed.[30] If motion is excessive, gap widening occurs and the muscle heals with more fibrous tissue.

Prolonged immobilization allows penetration of regenerating myofibers and decreased fibrous tissue formation; however, the muscle fibers tend to orient inappropriately to lines of stress and tension. This decreases the tensile strength of the muscle fibers, and if the fibrous tissue elongates during the remodeling phase, significant loss of muscle power can occur.[28,30] An initial immobilization period of 3 to 5 days followed by controlled mobilization accelerates the appearance of type I collagen fiber production and subsequently increases tensile strength.[33,34] Considering the normal timing associated with wound healing, recommendations have been made for 4 to 6 weeks of protected activity before increased activity.[28] Adequate approximation of the myofiber ends and controlled mobilization are extremely important to adequate long-term muscle healing and function.

Tendon Healing

Tendon Structure

Tendons, similar to ligaments, are composed primarily of type I collagen (>95%) organized into parallel bundles with surrounding extracellular matrix. Fibroblasts or tenocytes are the predominant cellular component of the tissue and function in repair and maintenance of the structure. The individual collagen bundles are surrounded by a loose connective tissue termed *endotenon*. The epitenon covers the entire group of bundles, and the paratenon covers and separates different tendon bellies, providing the potential for smooth gliding function.[28,35] Tendons are classified as either *vascular* (lacking synovial sheaths) or *avascular* (encased within a synovial lining). Tendons within synovial sheaths lack the typical paratenon. The blood supply to the tendon arises from three primary sources: the musculotendinous junction, the osseous point of insertion, and adjacent muscle and paratenon.[28]

Tendon Healing

The process of tendon healing follows the general course of wound healing as previously discussed with initial fibroplasia and collagen deposition followed by maturation and reorientation of the collagen fibrils (Box 6-5). The primary variable in tendon healing is whether the tendon is a vascular paratenon-covered tendon or a synovial-sheathed tendon. Paratenon-covered tendons rely less heavily on the intrinsic blood supply, as fibroblasts and capillaries invade the injured tendon ends from the surrounding tissue. This establishes the "one wound–one scar" theory.[35] With synovial-lined tendons, a longer period is required to achieve adequate tensile strength and healing because healing depends on the tendon's intrinsic blood supply or inflammatory repair tissue infiltration. Thus immobilization is extremely important in the initial period to resist gap formation at the repair site and allow adequate healing.[30,36,37]

Early descriptions of tendon healing involved an extrinsic process in which an influx of cells from the synovial sheath, paratenon, or epitenon allowed phagocytosis, fibroplasia, and subsequent collagen deposition.[28,38,39] The inflammatory stage is seen within the first 3 days and is quickly followed by randomly oriented collagen deposition by the fifth day. The reparative phase follows with progressively increasing collagen content through the first 4 weeks. Similar to muscle healing, the initial collagen is

Box 6-5 Healing Timeline: Tendon

Inflammation

Immediate to 72 hr

Repair

Day 5: random collagen orientation, progressively increasing for 4 wk; collagen production peaks 5-12 days after injury, markedly decreased by day 60

Remodeling/Maturation

Takes a minimum 28 days for collagen fibers to align according to stress; tendons appear normal by 112 days

Healing/Strength Characteristics

At 6 wk, 56% normal tensile strength occurs, 79% at 1 year and beyond.[36] Surgical repair of fresh gap required for appropriate organization of scar at 6 wk. Tendons with gaps > 3 mm have lower tensile strength at 6 wk compared to those with smaller gaps.[74] Synovial (avascular) tendons require longer healing times than vascular tendons.

Implications for Rehabilitation

Post-repair support for larger tendons may be needed for up to 6 wk. Method and extent varies depending on surgeon preference, location of the injury and patient behavior.[34] Prolonged immobilization may be detrimental. Complete immobilization for longer than 21 days results in reduced vascularization at the wound site.[75] With full or partial transection and surgical repair, passive mobilization exercises appear to be optimal to begin at day 5 following surgery.[76] Early, controlled passive mobilization continuing for 21 days appears to augment intrinsic healing, increase tensile strength and reduce adhesion formation.[41-46] One study showed active mobilization (cage activity) was not detrimental at 3 wk after injury for repaired partial flexor tendon lacerations up to 60%.[77] Conservative exercise with light weightbearing may begin at 6 wk, because having 25-50% of normal tensile strength for tendons has been considered adequate for withstanding normal muscle force. Minimum 12 wk for less restrained, more active loading.[78, 79] Light exercise to maintain ROM and stimulate healing callus cells. Some weightbearing is required for maturation of callus. Sufficient tensile strength return for normal activity load bearing may take months.

generally type III, which is replaced with the biomechanically superior type I collagen as repair and remodeling progress.[8,40] Research has demonstrated that healing of tendon ends by collagen deposition requires a minimum of 28 days for fibers to align parallel to lines of stress. In addition, collagen bundles are distinguishable from normal tendons until 112 days after injury.[38] Evaluation of healing of canine triceps tenotomies (a vascular tendon lacking a synovial sheath) demonstrated only 56% of breaking strength at 6 weeks and only 79% tensile strength 1 year following injury.[36]

More recently, evidence has been presented of the potential for intrinsic repair controlled predominately by the epitenon and endotenon.[41-44] This repair mechanism occurs primarily in synovial sheathed tendons with minimal damage to the synovial lining. If there is severe damage to the synovial sheath, the extrinsic mechanism overwhelms the capacity for intrinsic healing. The intrinsic pattern of cellular proliferation and sequencing is similar to extrinsic healing; however, there is a balance between epitenon and endotenon contributions and there is minimal adhesion formation.[40] The intrinsic healing process can be stimulated by early controlled mobilization, decreasing the risk of adhesion formation and providing greater tensile strength as a result of proper alignment of collagen fibers along the lines of stress.[41-46] Early controlled passive motion beginning within 21 days of repair appears to be ideal; however, the duration and magnitude of motion for optimal healing remain unknown. In addition, patient compliance in veterinary medicine is a problem and must be considered to ensure a good outcome.[30,42]

Tendon Repair

The goal of treatment is to minimize adhesion formation and return to maximum function. The appropriate treatment is surgical apposition of the transected or injured tendon ends. Numerous suture patterns have been designed to maximize tensile strength and attempt to minimize gap formation at the injury site. Commonly used techniques include the locking-loop and three-loop pulley patterns.[35,47-52] Adherence to several fundamental concepts is required. Meticulous hemostasis is necessary to minimize hematoma formation within the injury site and minimize adhesion formation. In addition, atraumatic technique and minimal handling of the tendon ends is important. Suture materials should be monofilament to minimize reactivity and nonabsorbable to allow time for the return of adequate tensile strength. Monofilament suture also allows efficient gliding through the tissue and even distribution of weightbearing forces and tendon tension.[28,35]

Ligament Healing

Ligament Structure

Ligaments are dense connective tissue structures consisting of fibroblasts, water, collagen, proteoglycans, fibronectin, and elastin that connect two or more bones.[11,53] They tend to widen at insertion points to blend with the periosteum and generally provide support to the articulating surfaces. Ligaments are primarily composed of long parallel or spiral collagenous fibers that are strategically arranged in multiple directions to restrain the joint from excessive excursions secondary to both normal and abnormal motions.[8,11] Histologically, the fibroblasts have elongated basophilic nuclei lying between collagen bundles.[53] The cellular and collagenous densities vary with each ligament. Collagen makes up 70% to 80% of ligament dry weight

composition, with the majority being type I fibers and approximately 10% being type III fibers.[53] The collagen fibers are generally crimped or buckled and can be stretched out with stress to provide elasticity and the potential to withstand rapid load application.[53]

Ligament Healing

Currently, a great deal of information remains unanswered regarding timing of ligamentous healing, especially with respect to postoperative mobilization techniques. This is because ligaments heal differently depending on the location. For example, the healing potential of the medial collateral ligament of the stifle is very good,[53,54] but the cranial cruciate ligament, which has received the most investigation, demonstrates virtually no healing response following injury.[55] However, it appears that ligaments generally heal according to the same basic pattern of inflammation, repair, and remodeling (Box 6-6).[55,56]

Within hours of injury, the defect is filled with an organized hematoma and the surrounding tissue becomes edematous from perivascular leakage of fluid. This inflammatory stage is characterized by an influx of inflammatory cells as in general wound healing. Monocytes and macrophages are found in the wound by 24 hours and respond by cleaning up the site and transitioning to the next phase. This stage lasts for approximately 48 to 72 hours.[55]

The reparative phase begins 2 to 3 days after injury and persists for approximately 6 weeks.[55] Both cellular and matrix production predominate, with granulation tissue and vascular ingrowth filling the defect between friable ligament ends. The scar in the first few weeks is highly cellular with fibroblasts actively synthesizing primarily types I and III collagen and other extracellular matrix components.[55] During this aspect of ligament healing, the scar cross-sectional area reaches a maximum.[30] Type I collagen predominates and tensile strength of the ligamentous repair tissue increases as time progresses.

The final stage of remodeling and maturation progresses as with normal wound healing and may take more than 12 months to complete. At that point, ligament strength is only 50% to 70% of the original tensile strength.[55] Part of the lost strength may also be a result of bone loss at the ligament insertion sites. In general, healing of the midsubstance of the ligament progresses more rapidly than the insertion sites because bone turnover and recovery are slower. Fibroblast and macrophage numbers decrease, and the collagen fibers become more densely packed and appropriately aligned. Factors that are important to appropriate repair of tissue include ligament end apposition, nutritional status, endocrine imbalances, severity of injury, blood supply, and mechanical stresses placed across the healing tissue.[30,55] As with muscle healing, allowing the ligament to heal across a large gap results in excessive scar tissue formation that obstructs histologically normal ligament from bridging the defect.[30]

Box 6-6 Healing Timeline: Ligament (Sprain)

Inflammation
Immediate to 72 hr

Repair
2-3 days to 6 wk

Remodeling/Maturation
N/A

Healing/Strength Characteristics
Occurs in several days to more than 1 yr, depending on grade (I-III). At 1 yr, 50-70% tensile strength occurs.[65] Healing properties and rate of healing depend on which ligaments are involved.[80,81] Transected canine stifle medial collateral ligament (MCL) strength 52% of controls after 14 wk of healing, reduced to 14% of controls when cranial cruciate ligament (CCL) also transected. Transected canine CCL weakness persists after 4 yr of healing (hence other methods of surgical repair needed). Lengthening may occur because of gap formation.

Implications for Rehabilitation
Time to begin rehabilitation varies widely depending on the ligament involved. When in doubt, parameters for tendon healing may generally be followed. Tensile strength reduction may be slowed or decreased with joint motion during immobilization and stress reduction. No change at 6 wk and 67% reduction at 12 wk in repositioned canine CCL in one study.[82] Low-duration (30 min) and high frequency (6 days/wk) exercise appears to have greatest benefit to ligament strength from one study in horses.[83] Sprain-avulsion fractures at ligament-bone interface have best prognosis for return to normal tensile strength; fractures heal within 2-4 mo, which restores ligament integrity.[84]

Articular Cartilage Healing

Articular Cartilage Structure and Function

Diarthrodial (synovial) joints consist of two or more hyaline cartilage surfaces that are tightly adhered to the underlying cortical end plates of apposing bones. Hyaline cartilage is designed to provide a smooth, low-friction, gliding surface for joint movement and to transmit weight-bearing forces to the underlying bone structure.[11,57-60] Cartilage is primarily avascular and without lymphatics. It is approximately 70% to 80% water by weight with the remainder composed of chondrocytes, type II collagen (90% to 95% of the total cartilage collagen), and proteoglycan aggregates.[11,57-61] Collagen provides tensile strength to the joint surface, which is divided into three zones. The most superficial layer, the tangential zone, has collagen fibers aligned parallel to the joint surface, creating primary

resistance to shear and tensile stresses placed on the cartilage surface. Deeper within the cartilage, the collagen fibers become oriented in a more oblique fashion and form the intermediate zone. The final, and largest, zone is the radial zone in which the collagen fibers are oriented in a perpendicular columnar arrangement and become embedded into the subchondral bone.[57-61]

Chondrocytes compose less than 10% of the total cartilage volume.[61] The chondrocytes are oriented similar to collagen except for an additional zone of calcification present deep within the cartilage to provide transition to the subchondral bone. Surrounding each chondrocyte is the cartilage matrix separated into three regions classified as pericellular, territorial, and interterritorial. The function of the chondrocyte early in life is primarily cell proliferation and matrix synthesis. As maturation proceeds, these processes slow, with cellular density decreasing and interterritorial matrix size increasing.[57-61]

Proteoglycan aggregates provide the compressive strength to cartilage and progressively increase in quantity from the intermediate to the radial zones of the cartilage.[58] The primary constituents of the proteoglycan aggregate are a series of glycosaminoglycans attached to a core protein, which in turn are linked to a backbone chain of hyaluronic acid. The glycosaminoglycans are composed of chondroitin-6 sulfate, keratan sulfate, and some chondroitin-4 sulfate. These highly sulfated and carboxylated structures carry negative charges that strongly repel each other, resulting in stiffly extended aggregates. In addition, they are highly hydrophilic. The resulting osmotic swelling pressure, along with the fixed anionic charges, produces dramatic compressive resistance. These products are synthesized by chondrocytes and provide supportive structure to the collagen fiber network.[56-61]

A capsule surrounds the joint and is created by an outer fibrous layer and an inner synovial membrane. The outer fibrous layer, continuous with the periosteum, is composed primarily of collagen and helps provide mechanical support. The inner synovial membrane is a thin layer of synovial cells closely associated with a subsynovial vascular plexus.[11,57-59] The cellular component of the synovial membrane is divided into type A synoviocytes, which are primarily responsible for phagocytic activity, and type B synoviocytes, which are responsible for hyaluronic acid production.[57,58] Synovial fluid is a plasma ultrafiltrate from the subsynovial plexus, which allows molecules less than 12,000 daltons to permeate into the joint. To this ultrafiltrate, the type B synoviocytes add hyaluronic acid.[57,58] Synovial fluid provides joint lubrication and cartilage nutrition, which occur through two separate processes.[11,57,58] Boundary lubrication is associated with the synovial membrane, the periphery of the cartilage surface, and hyaluronic acid. Hydrostatic lubrication is a process created by the weight-bearing force on the cartilage surface, which pushes water to the surface during weight bearing. Weight bearing and joint motion also improve the diffusion of nutrients to the chondrocytes and the removal of waste metabolites.

Cartilage Healing

Injury to articular cartilage and the subsequent healing response are directly related to the type of trauma incurred. Similar to the basic pattern of general wound healing, the ideal response includes initial necrosis and inflammation, followed by vascular ingrowth and the reparative phase (Box 6-7). The limiting factor for the repair of articular cartilage is its avascular nature. As a result, injury to the articular surface results in two separate responses based on whether the injury is limited to the cartilage surface or extends into the subchondral bone.[62,63]

Box 6-7 Tissue Healing: Articular Cartilage

Partial Thickness

Inflammation

No inflammatory response—avascular

Repair

Mitotic activity ceases 1 wk after injury

Remodeling/Maturation

Local chondrocyte activity does not adequately fill the defect at 1 yr because of lack of inflammatory healing response

Healing/Strength Characteristics

N/A

Implications for Rehabilitation

N/A

Full Thickness (to subchondral bone)

Inflammation

Immediate to 3-5 days (inflammatory response from marrow cells)

Repair

5-7 days to 2 mo

Remodeling/Maturation

Occurs in 2-6 mo; by 6 mo repair tissue changed significantly from normal hyaline cartilage. Repaired tissue is fibrous and inferior to hyaline cartilage. At 1 yr, 20% is type II collagen.[63]

Healing/Strength Characteristics

Defect repair depends on patient age, size, and location of the defect. Regardless, complete restoration of hyaline articular cartilage is very rare.[87]

Implications for Rehabilitation

Continuous passive range of motion (PROM) after injury appears to improve healing with mainly hyaline cartilage observed in 60% of defects (adolescent) and 44% of defects (adult) in rabbits undergoing continuous PROM, compared to 10% or fewer in controls (immobilization in a cast or free-range in cages).[87] Less profound results occur in defects larger than 3 mm in diameter.

Injury limited to the cartilage surface results in a number of secondary responses. A traumatic laceration perpendicular to the joint surface (without invasion below the calcified cartilage layer) results in localized death of the chondrocytes and subsequent loss of matrix support. Consequently, a matrix defect occurs that is characterized by a lack of vascular infiltration and no inflammatory response. As a result, there is no fibroblastic response, and the local chondrocytes must proliferate and fill the defect with new matrix.[62,63] The chondrocytes respond with an initial increase in mitotic activity adjacent to the defect.[64] However, the chondrocytes may not completely fill the defect and a lesion often remains. With a lesion parallel to the cartilage surface, a similar response ensues with initial limited cellular proliferation and increased proteoglycan and matrix synthesis to fill the injured site. The primary problem is the limited durability of the repair process and the failure to completely replace the cartilage matrix. If the defect is large, then degenerative joint disease may ensue. It appears that if the defect is small, such as in a surgical laceration, it will generally be limited in progression and not proceed to significant degenerative change within the joint.[62,63,65]

Full-thickness injury to the level of the subchondral bone allows access of marrow stem cells and vascular capillaries to the cartilage defect. As a result, the injury can respond similar to general wound repair with an initial debridement and inflammatory phase. Within 48 hours a fibrin clot forms, providing a scaffold for migration of fibroblasts.[63,65] Fibroblasts and collagen replace the clot in 5 to 7 days and metaplasia to fibrocartilage begins. Matrix formation occurs with a proteoglycan-rich environment, and defect repair takes approximately 2 months to complete.[63,65] Wound healing continues with remodeling and maturation. At 6 months, the glycosaminoglycan concentration decreases significantly and tends to be rich in dermatan sulfate, which is smaller and less wear resistant than other glycosaminoglycans. Type I collagen predominates in the early stages but slowly decreases with time (40% at 8 weeks, 20% at 1 year).[63] The repair tissue remains more fibrous and is biomechanically inferior to the normal surrounding hyaline cartilage with a gross distinction permanently remaining.[62,63,65]

Because of the poor repair potential of articular cartilage, research has focused on methods of facilitating repair of hyaline cartilage. Surgical debridement continues to play an important role in treatment of articular surface defects. Arthroscopic shaving of partial-thickness defects essentially relieves clinical signs associated with defects; however, it does not appear to dramatically alter healing potential.[62,63] Recent advances have led to the concept of perforation or abrasion of the calcified cartilage layer and invasion to the subchondral region to elicit vascular invasion and a local repair response.[63,66] Another important approach to repair of articular defects is the concept of early continuous passive motion postoperatively, because prolonged immobilization impairs the biomechanical and biochemical healing capacity of articular cartilage.[62,63,67] Additional research includes the potential for electrical stimulation, cartilage grafting, and the effects of growth factors on cartilage healing.[63]

Advances for the Future

Wound healing remains an area with tremendous research potential. An exciting area of interest includes the potential for modulation of wound healing with the use of various growth factors. Growth factors are low-molecular-weight polypeptide molecules that are located throughout the body and have a powerful influence on cellular activity. Factors such as transforming growth factor alpha and beta, platelet-derived growth factor, fibroblast growth factor, epidermal growth factor, and insulin-like growth factor modulate cellular activity by binding to receptors on appropriate target cells, resulting in stimulation of specific cellular activities. These substances may have various effects, including chemotaxis, mitogenesis, cellular activation, and stimulation of all aspects of wound healing, with their exogenous use intended to expedite the repair of injured tissues.[68-70] Recently, the potential for stem cell application to healing tissues has generated a great deal of interest. In addition to the altered cellular environment generated by the cells, the potential for stem cells to grow and differentiate into cells specific to a particular tissue type is exciting.

Summary

Wound healing in most tissues involves an initial process of hemostasis followed by inflammation. This allows for debridement of the injured region followed by chemotaxis of the appropriate cells for production of repair tissue. In general, the repair process involves production of collagen, bone, or myofibrils. Maturation occurs over time, allowing for appropriate alignment of collagen and tissue for resistance of tensile forces. The initiation of physical rehabilitation in the postoperative period is important, and the techniques should be used to augment the maturation phase to maximize early return of tensile strength and function. The time of initiation of various therapeutic modalities is important, and knowledge of the basic patterns of wound healing is imperative.

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7 Responses of Musculoskeletal Tissues to Disuse and Remobilization

Darryl L. Millis

Patients afflicted with neurologic, orthopedic, metabolic, and other diseases frequently have reduced weight bearing and use of limbs. In addition, some conditions may be treated with splints, casts, or other coaptation devices, resulting in decreased limb use. The tissues most affected by disuse and immobilization are cartilage, joint capsule, muscle, ligament, tendon, and bone. Knowledge of how these tissues respond to disuse and immobilization, the types of deleterious tissue changes, and the time frame over which these changes occur, are important in understanding the need for physical rehabilitation. For example, stress deprivation of joints results in proliferation of connective tissue within the joint space, adhesions between synovial folds, adherence of connective tissue to cartilage surfaces, atrophy of cartilage, reduced proteoglycan (PG) content, ulceration at points of cartilage to cartilage contact, disorganization of cellular and fibrillar ligament alignment, reduced collagen mass, increased ligament compliance, weakened ligament insertion sites, reduced load-to-failure and energy-absorbing capacity of the bone-ligament-bone complex, and osteoporosis of the involved extremity.[1]

Rehabilitation of patients with acute or chronic neurologic or orthopedic conditions involves the application of controlled challenges to tissues to improve strength, condition, and function. It is important to understand how to safely remobilize tissues after injury and a period of immobilization. Rehabilitation must sufficiently challenge tissues to enhance and positively influence their recovery and healing. However, if tissues are overchallenged, they may be damaged, ultimately delaying recovery or cause additional injury. There must be a balance between the simultaneous demands for protection against undue stress to facilitate healing, and the need for stress to attenuate atrophy of musculoskeletal tissues.[2] A review of these concepts forms the basis for a rational approach to developing rehabilitation programs.

Cartilage

Disorders of articular cartilage and joints are some of the most common and debilitating diseases encountered in veterinary practice. Understanding the normal structure and function of articular cartilage is a prerequisite to understanding the pathologic processes. The mechanical properties of articular cartilage arise from the complex structure and interactions of its biochemical constituents. The viscoelastic properties of cartilage, primarily resulting from fluid flow through the solid matrix, explain deformational responses observed during loading. For example, cartilage that is loaded rapidly is stiffer than the same cartilage that is loaded slowly. Degenerative processes include the breakdown of normal constituents of cartilage, especially the collagen network and PGs, which affects the mechanics of fluid flow through cartilage. Factors contributing to such breakdown include direct trauma, obesity, immobilization, and excessive repetitive loading of the cartilage. Training activities, without traumatic injury, do not appear to be a risk factor for developing osteoarthritis (OA) in normal joints, but such activity may be harmful to an abnormal joint.[3]

Normal Articular Cartilage

Articular cartilage covers the ends of long bones and is composed of chondrocytes, extracellular matrix, and water. Articular cartilage is avascular, aneural, and alymphatic. Synovial fluid is present in joints and provides a near-frictionless, wear-resistant, weight-bearing layer in contact with articular surfaces. Articular cartilage dissipates contact stresses during loading, distributing compressive and shearing forces, which are transmitted through subchondral bone to the stiffer cortical bone.

Chondrocytes are metabolically active and make up less than 5% of the cartilage volume. Metabolism of the cells is modulated by their location within the cartilage, by aging, and by biomechanical stresses. Chondrocytes produce and maintain the extracellular matrix and pericellular environment. The substrates for these processes come mainly from the synovial fluid. Weight bearing results in a synovial fluid "pumping" mechanism that facilitates the transfer of nutrients and waste exchange by diffusion from the cartilage surface.

The extracellular matrix is composed of collagen, PGs, and water. The orientation of collagen and PGs functions to distribute forces over the subchondral bone and provides a smooth surface that allows movement of joints

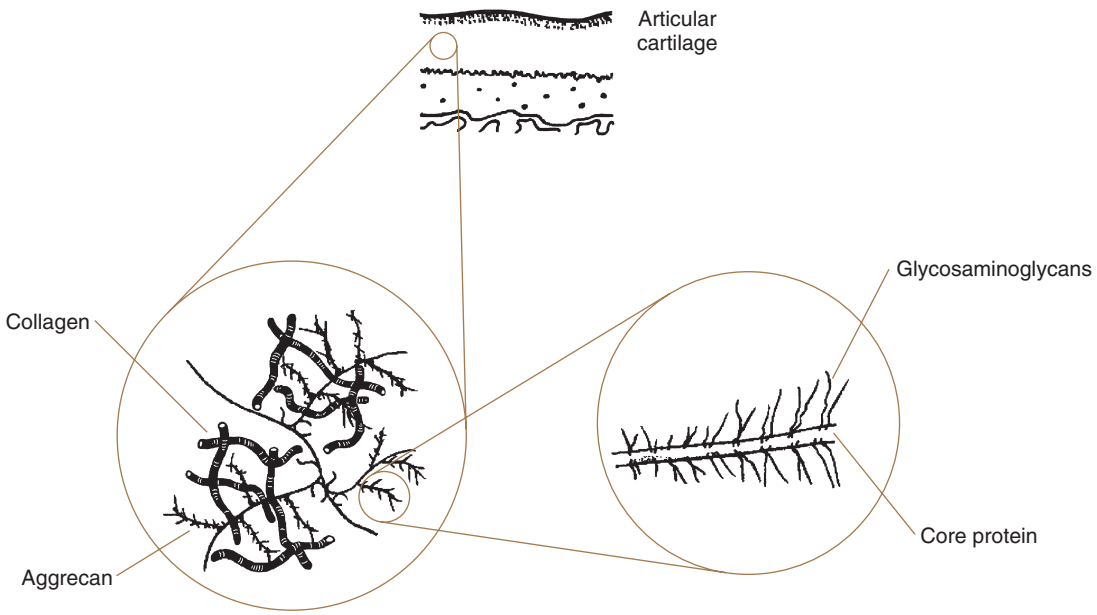


Figure 7-1 Diagram of articular cartilage, including collagen, proteoglycans, and water.

(Figure 7-1).[3] Collagen provides tensile strength to the cartilage, and structural support for the extracellular matrix. Type II collagen is the primary form in articular cartilage and is unique to cartilage. Type IX collagen is also unique to articular cartilage and links type II fibrils together, limits the separation of collagen fibrils, limits fibril diameter, and binds PGs to collagen. Processes resulting in articular cartilage loss may cause collagen degradation, either enzymatically, mechanically, or both. Type IX collagen is especially susceptible to enzymatic degradation.

PGs make up most of the extracellular matrix that is not collagen, and compose 22% to 38% of the dry weight of articular cartilage. PGs are highly hydrophilic, which results in water retention, creating swelling pressure and turgidity that is essential to articular cartilage function. A PG monomer consists of a core protein with one or more types of glycosaminoglycan (GAG) chains attached. The core protein has a hyaluronan binding region, GAG binding regions, and a carboxy-terminal end. A link protein helps to bind the PG chain to hyaluronan (Figure 7-2). Hyaluronan is a nonsulfated GAG located in the extracellular matrix and forms the backbone to which the PG monomers are covalently bound. *Aggrecan* refers to a PG monomer that combines with hyaluronan.

GAGs are structures with varying lengths of repeating disaccharides. Chondroitin sulfate and keratan sulfate are the major GAGs, composing almost 90% of total articular cartilage GAGs. GAGs are negatively charged, causing them to repel one another when attached to a core protein and occupy a large area.

Water composes 65-80% of total cartilage weight.[3] Although PGs are highly hydrophilic, enclosure in the collagen meshwork limits their ability to expand. This keeps cartilage turgid, resisting deformation. Water moves slowly within the cartilage matrix during loading as a result of resistance of flow through the collagen meshwork and the hydrophilic nature of the PGs. Water is forced out of articular cartilage during loading and some water weeps onto the articular surface, allowing hydrostatic lubrication. When the load ceases, water is reabsorbed. With rapid loading, cartilage is stiffer because water distributes more slowly, resulting in less compression of the cartilage. Cartilage is more compliant with slow loading because there is additional time for fluid movement to the cartilage surface.

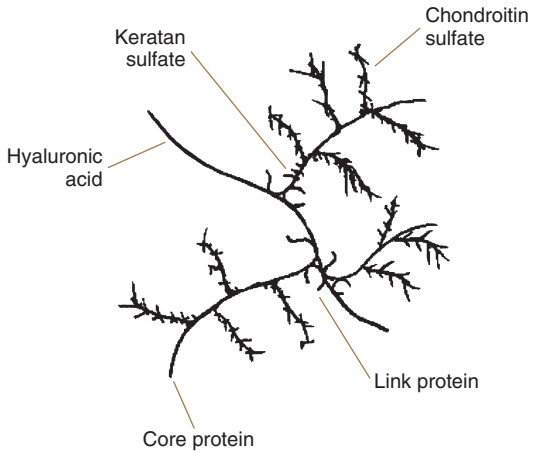


Figure 7-2 Proteoglycan monomer consisting of a core protein, glycosaminoglycans, hyaluronan backbone, and link protein.

Histologically, cartilage may be divided into various zones based on their depth within the cartilage and their characteristics. These zones include a relatively thin superficial layer; a middle layer with the cells arranged in vertical columns; and a tidemark, which is the upper limit of the zone of calcified cartilage, which in turn interdigitates with the subchondral bone plate.

The subchondral bone is the thin layer of bone beneath the calcified cartilage layer. It is 10 times more deformable than cortical bone and is important in distributing forces during loading. Its relatively high compliance helps to diminish the peak load placed on the cartilage, decreasing damage to the articular surface. The subchondral bone becomes stiffer with OA. As the bone becomes more dense and sclerotic, the cartilage and bone do not deform normally, and the chondrocytes and matrix are more likely to be damaged with loading.

Cartilage Changes Caused by Disuse and Immobilization

Clinicians who attempt to restore joint function should understand the effects of immobilization on articular cartilage (Box 7-1). Disuse of cartilage may result in atrophic or degenerative changes, and these changes are more marked and appear sooner in areas of contact.[4] Immobilization leads to a decrease in matrix and cellular components, disorganization of cartilage, and potential irreversible damage if immobilization is prolonged.[5] In addition to a reduction in synovial fluid production with immobilization, nutrition to cartilage is diminished as a result of reduced synovial pumping and nutrient diffusion. The age of the dog, as well as the form of immobilization used, may affect results. Several models of joint immobilization and disuse have been studied, including casting with the limb in flexion or extension, external skeletal fixation, and non–weight-bearing models (such as sling application). The changes in mechanical behavior and biochemical composition of articular cartilage appear to be less dramatic in limbs immobilized in a sling than in limbs immobilized in a cast. Adult greyhound dogs had stifles placed in a sling at 90 degrees of flexion for 4 or 8 weeks, which allowed some limited motion of the stifle but no weight bearing. There were no changes in the compressive properties of cartilage, PG content, collagen content, or cartilage thickness.[6] This suggests that immobilization with a sling may be less deleterious to articular cartilage as compared with other models of joint disuse in which cartilage changes are both progressive and degenerative.[6] Immobilization with a joint in flexion does not generally lead to arthritic changes in the short term, but cartilage atrophy may occur. Immobilization of a joint in extension results in increased muscle contraction against the immobilization device and changes in articular cartilage similar to those seen in OA.

The cartilage of limbs of young dogs immobilized in flexion for 3-11 weeks was grossly normal with no osteophytes.[7,8] However, there was a 13% to 60% early and progressive reduction of PG content following immobilization, with the depletion being most severe in the superficial zones.[4,8-10] Immobilization for as little as 6 days may result in a 40% reduction in cartilage PG synthesis, and up to 60% for joints immobilized for 8 weeks.[4] In addition, there is a 10-20% increase in water content. The accelerated PG loss appears to be caused by a combination of decreased synthesis and increased proteolysis of PGs.[11] The loss of PGs is somewhat selective, with preferential loss of

Box 7-1 Summary of the Effects of Disuse and Immobilization on Articular Cartilage

- Atrophic or degenerative changes
  - Changes more severe and appear sooner in areas of contact
- Decrease in matrix and cellular components
- Disorganization of cartilage and potential irreversible damage if immobilization is prolonged
- Reduced synovial fluid production
- Diminished nutrition to cartilage because of reduced synovial pumping and nutrient diffusion
- Immobilization of joints in extension results in arthritic changes to cartilage
- Immobilization of stifle joints in flexion results in cartilage atrophy in young dogs, but less atrophy in older dogs
  - 3 to 11 weeks of immobilization in flexion results in 13% to 60% reduction of proteoglycan content in young dogs
  - 40% to 60% reduction in proteoglycan synthesis with immobilization in flexion
  - Cartilage thickness reduced 9% to 50%
  - Up to 30% decrease in chondrocytes
  - Atrophy of subchondral bone
  - Elevated MMP-2 and decreased TIMP with 4 weeks of immobilization
  - Up to 30% decreased stiffness of cartilage
- Active stifle joint motion may be more important than weight bearing in meniscal cartilage
  - Joint inflammation with reduced weight bearing following cranial cruciate ligament transection and stifle stabilization may result in decreased meniscal stiffness (30%)
  - Joint motion and weight bearing may improve regeneration of tissue and maintain articular cartilage following partial meniscectomy as compared with immobilization
- Some joint motion during immobilization (as opposed to rigid immobilization) reduces deleterious effects
- If joint instability exists, such as cranial cruciate ligament rupture, temporary immobilization in flexion (non–weight-bearing position) may result in less osteoarthritis until stabilization procedure performed

MMP-2, Metalloproteinase 2; *TIMP,* tissue inhibitor of metalloproteinases.

chondroitin-6-sulfate.[9] The levels of synovial fluid PG components, including link protein, keratan sulfate, and total sulfated GAG, were determined in dogs after a 3-week recovery period following either 4 or 8 weeks of immobilization with the stifle in flexion.[12,13] The concentrations of keratan sulfate and sulfated GAG in the synovial fluid lavages were increased after disuse and after disuse with recovery compared with controls, but the levels of link protein remained low. This pattern of catabolism differs from that found in OA, in which link protein is elevated in synovial fluid lavages, along with keratan sulfate and sulfated GAG.

After 3 weeks of immobilization, PG aggregation is poor. These changes are reversible, however, because PG aggregates were normal 2 weeks after removal of a cast that had been worn for 6 weeks.[4] The interaction of PGs with hyaluronic acid (HA) may be reduced in older dogs because of an abnormality in the HA binding region of the PG core protein,[4] but others have shown no abnormality in aggregation in young dogs.[9,11]

Young dogs subjected to 11 weeks of immobilization by splinting had a significant reduction in hyaluronan concentration in the tibial and femoral condyles, and the patellar surface of the femur, with a concurrent decrease in aggrecan, but the ratio of hyaluronan to aggregan was unchanged.[14] Young dogs may respond differently, because there was no change in hyaluronan content in skeletally mature dogs following 4 to 8 weeks of immobilization, as compared with an 80% decrease during early OA.[10] This difference supports the concept that the mechanism of cartilage degradation in disuse and OA is different, and immature dogs may respond differently than older dogs to immobilization. It may also help explain why the changes in cartilage following immobilization may be reversible, whereas the changes in osteoarthritic cartilage are not reversible.

Cartilage thickness may be reduced 30-50% with immobilization, but the location of thinning within the cartilage is not consistent. In mature dogs, thinning occurs primarily in the uncalcified cartilage, whereas in young dogs it tends to occur in the calcified cartilage under the tidemark.[4,8] Immobilization may result in 30% fewer chondrocytes per unit area in cartilage.[4] The subchondral bone also atrophies.

Sling immobilization of the stifle for 4 weeks results in elevation of gelatinase (neutral metalloproteinase 2 [MMP-2]) and almost complete suppression of tissue inhibitor of metalloproteinases (TIMP), especially in the superficial zone, as well as reduced protein synthesis by chondrocytes.[15] In contrast, collagenase activity is normal. The return of MMP-2 and TIMP levels to normal was observed with a 2-week remobilization period or treatment with insulin-like growth factor-1 (IGF-1) and pentosan polysulfate. This suggests that cartilage is remodeling, similar to bone remodeling in disuse atrophy, but the material properties of the articular surface remain relatively intact following a period of immobilization with a sling.

Contact forces between articulating surfaces appear to be necessary to maintain normal PG content of articular cartilage.[9] The greatest depletion of GAGs (64%) occurred at the cranial and caudal extremes of the femoral condyles in the stifle joint cartilage of young beagle dogs following cast immobilization for 11 weeks in 90 degrees of flexion. These were locations where the immobilized cartilage lost contact with the opposing cartilage.[9]

The biochemical changes that occur with immobilization also affect the biomechanical properties of articular cartilage. Splint immobilization of stifle joints of young dogs for 11 weeks caused significant softening of femoral and tibial cartilage with no visible changes of the cartilage surface.[16] The rate of deformation during loading increased 42% and the average thickness of the cartilage decreased 9% as compared with controls. Loading of cartilage may be necessary to maintain cartilage stiffness, because stiffness was maintained in the contact area between the patella and patellar surface of the femur as a result of sustained loading between the femur and the patella produced by the flexion of the knee joint, whereas cartilage softening was greatest in nonloaded areas. There is correlation between cartilage stiffness and PG concentration. Therefore loss of PGs during immobilization may also affect biomechanical properties. Immobilization of stifles with a cast caused significant softening of up to 30% in the lateral femoral and tibial cartilages.[17] The GAG content of the cartilage was slightly decreased after immobilization, especially in the superficial zone of cartilage. The changes in stiffness correlated with alterations in GAG content of the superficial and deep zones. This confirms the key role of PGs in the regulation of cartilage stiffness. There may also be changes in the compressive and shear stiffness of cartilage from nonimmobilized contralateral limbs, possibly as a result of increased loading.[18]

The changes that occur to articular cartilage during immobilization are probably caused by a combination of decreased joint motion and reduced loading. Although both are important in maintaining cartilage integrity, normal loading is particularly important with contraction of muscles that span the joint and stabilize the limb during weight bearing.[6] In a non–weight-bearing model that allowed joint motion of the stifle joint, cartilage thickness, PG content, and PG synthesis were reduced, whereas water content was increased.[19] These changes were similar to those that occur with immobilization in a flexed position and suggest that weight bearing is necessary to prevent the bulk of changes.

Some joint motion during immobilization is beneficial. Stifles immobilized for 6 weeks with a cast, allowing 8 degrees to 15 degrees of motion, had less PG loss and a smaller decrease in PG synthesis than occurred with more rigid fixation. The limited motion provided by casting was

Box 7-2 Summary of Effects of Immobilizing Stifle Joints in Flexion on Articular Cartilage

- PG content reduced 25-60%
- Cartilage thickness reduced 30-50%
- Cells per unit area 30% fewer
- Water content increased 10-20%
- Net PG synthesis decreased 40-60%
- Atrophic subchondral bone

PG, Proteoglycan.

protective.[11] Elimination of motion by rigid fixation induces even more pronounced atrophic changes to articular cartilage. To study the effects of immobilization on the ultrastructure and surface contour of articular cartilage, knee joints of adult dogs were immobilized with circular external fixation for two to four weeks.[20] The degree of cartilage changes increased with the length of immobilization, and consisted of destruction of the superficial cartilage layer, cleft formation in the surface, and erosion of the articular cartilage. In another study, knees of mature dogs were immobilized for 6 weeks with external fixators. There was a 7% increase in water content compared with normal knee cartilage, and a 28% decrease in GAG content, which was greater than if some motion was allowed.[11] More importantly, GAG content did not return to normal after a 1 week remobilization period as it did in dogs that were not rigidly immobilized. Spinal cord injury, which may result in reduced mobility and decreased joint motion, may also result in cartilage thinning.[21]

Immobilization of the stifle in flexion may be beneficial when joint instability exists, such as occurs with a ruptured cranial cruciate ligament (CCL), because bearing weight on an unstable stifle results in OA. Dogs undergoing transection of the CCL had their stifle joints maintained in flexion or were permitted ad libitum ambulation for 12 weeks.[22] Dogs allowed to bear weight had osteophytes, fibrillation, and decreased cartilage PG content, although cartilage thickness was normal. PG synthesis was 80% greater than that in cartilage from the contralateral knee. In contrast, osteophytes were not seen when the limb was immobilized in flexion immediately after transection of the ligament, and the articular surface remained intact. The cartilage became atrophic, however, and PG content and PG synthesis were decreased compared with cartilage from the contralateral knee. Knee cartilage from immobilized limbs after cruciate ligament transection, therefore, resembled that from dogs whose limbs were immobilized without ligament transection (Box 7-2).

The effect of position of the limb during immobilization on blood flow to the joint must also be considered. For example, forced abduction of experimentally induced hip dysplasia with secondary OA results in significant reduction of blood flow, whereas immobilization in flexion results in the highest blood flow to the femoral head.[23]

Box 7-3 Considerations for Remobilization of Cartilage after Immobilization

- The length of immobilization, condition of cartilage, and the length and magnitude of weight bearing after immobilization affect cartilage recovery.
- After a 6-week immobilization period, 3 weeks of free, low-intensity activity results in normal cartilage.
- Longer periods of immobilization require longer recovery times.
  - Immobilization longer than 15 weeks may not result in complete recovery, even with 50 weeks of remobilization, in young dogs.
- Vigorous exercise after immobilization may be deleterious to cartilage.
  - Jogging young dogs 9.5 km/d at 5 km/h after immobilization for 3 weeks resulted in continued decreases in cartilage thickness (20%) and PG content (35%), even though PG synthesis increased (16%).

PG, Proteoglycan.

The Effects of Remobilization on Cartilage

The response of cartilage to remobilization depends on the biomechanical demand that the joint is exposed to, the condition of the cartilage, and the length of the immobilization and remobilization period (Box 7-3). If the joint is subjected to high stresses and repeated loading immediately after immobilization, cartilage may not be able to resist the stresses and may become damaged because of injury to the softened matrix. The duration and the degree of load bearing after immobilization are factors that determine the cartilage response. Also, the ability to restore biomechanical properties may depend on an intact collagen network.

The atrophic changes of cartilage that occur with a limb casted in flexion are somewhat reversible. Remobilization of a normal limb casted in flexion for 6 weeks by allowing ambulation for 3 weeks after cast removal resulted in the cartilage becoming normal.[4]

Longer periods of immobilization in young dogs may result in more long-standing effects, even with a gentle remobilization period. Skeletally immature female beagle dogs were immobilized in a cast for 11 weeks in 90-degree flexion followed by remobilization for 15 weeks.[9] After remobilization, PG content remained an average 18% lower than controls, with content at the minimum contact sites 33% lower. GAG concentration was restored in the more loaded regions of the patellofemoral region and tibial condyles, but remained lower than the control values in the

less loaded peripheral regions of the femoral condyles.[24] Uncalcified cartilage thickness of the femoral condyles was 15% less than in the controls, and the thickness of the calcified zone was reduced in most regions of the remobilized stifles. The thinness of the calcified zone may be due to advanced bone growth from the subchondral bone during remobilization. Softening of the articular cartilage during immobilization may expose the calcified zone to increased mechanical loading and result in mineralization and thickening of subchondral bone. The changes induced by unloading are reversible to a great extent, but full restoration of articular cartilage may not occur at all sites following 15 weeks of remobilization. Immobilization of the skeletally immature joint therefore may affect the development of articular cartilage in such a way that very slow recovery or permanent alterations are induced.

Longer periods of remobilization may be more beneficial. Hindlimbs of immature dogs were immobilized for 11 weeks and then remobilized for 50 weeks.[17,25,26] Immediately after the immobilization period, cartilage GAG concentration was reduced similar to that found in other studies. The amount of collagen crosslinks was also reduced during immobilization. After 50 weeks of remobilization, GAG concentration was restored at most sites, but remained 9-28% lower than controls in the patella, medial femoral condyle, tibial condyles, and the proximal femoropatellar surface. Collagen crosslinks were restored to control levels, indicating that collagen is resistant to reduced joint loading and slow remobilization. Although the articular cartilage of immobilized limbs became similar to control cartilage following long periods of remobilization, full restoration of articular cartilage GAG concentration was not obtained in all sites, even after remobilization for 50 weeks. This suggests that joint immobilization of young dogs can cause long-lasting articular cartilage PG alterations.

Because GAGs exert a strong influence on the biomechanical properties of cartilage, the biochemical changes that occur in cartilage during remobilization may also affect the biomechanical properties. The restoration of the biomechanical properties of articular cartilage was studied after 15 weeks of remobilization of the knee joint in mature beagles previously immobilized with a cast for 11 weeks.[27] Remobilization improved the decreased stiffness and cartilage thickness in femoral, tibial, and patellar cartilage created by immobilization, and approached values of nonimmobilized controls. However, stiffness of the femoral condylar cartilage remained lower and cartilage permeability was higher compared with the controls. A study with the same period of immobilization but with a remobilization period of 50 weeks resulted in restoration of the biomechanical properties of cartilage in the lateral condyle of tibia, but not in the lateral condyle of femur, where stiffness remained 15% below the control level following remobilization.[17] Similar to biochemical changes, articular cartilage biomechanical properties return toward normal during remobilization, although the recovery is not complete in all parts of the knee even after 50 weeks of remobilization. Immobilization of joints of young dogs may cause long-term, if not permanent, alterations of cartilage biomechanical properties, similar to changes observed in biochemical components of articular cartilage. This may predispose joints to degenerative changes later in life.

The atrophic changes seen in cartilage following immobilization are less reversible following immobilization with a rigid external fixator. However, in one study, 2 to 4 weeks of remobilization following 2 to 4 weeks of immobilization with an Ilizarov external fixator demonstrated that the longer the period of remobilization, the greater the reparative processes.[20]

In contrast to the improvements seen with mild remobilization strategies, vigorous exercise after a period of immobilization may have deleterious effects on cartilage.[28] Dogs that ran daily (9.5 km/d, 5 km/h) on a treadmill for 3 weeks after cast removal had continued decreases in cartilage thickness (20%) and PG content (35%), even though net PG synthesis was 16% greater than in cartilage from contralateral nonimmobilized stifles. Therefore vigorous loading of the joint following a period of immobilization may prevent reversal of cartilage damage and ultimately be detrimental to cartilage. It appears that the atrophic changes are more reversible with gentle spontaneous reloading.

Meniscal Cartilage Changes

Various experimental models for producing atrophy of meniscal tissue have yielded contradictory results. These models have included denervation of the stifle, immobilization (both internal and external), and disarticulation. Active motion of the stifle may play a more important role than weight bearing in preventing meniscal atrophy. In one study, internal skeletal fixation, which did not allow motion of the stifle joint, was applied for 12 weeks. There was significant atrophy of the lateral meniscus, despite the opportunity for weightbearing.[29] Another study evaluated the effect of active joint motion on the maintenance of meniscus mass in a unilateral ankle disarticulation non–weight-bearing model of disuse. This model maintained active knee motion without weight bearing. Despite the loss of collagen and calcium mass in the femur and tibia because of decreased weight bearing, there was no loss of meniscal mass.[30]

Although meniscal mass may be maintained when motion is allowed in stifles with limited weight bearing, there may be profound changes in biomechanical and biochemical properties. Meniscal cartilage stiffness decreased by 30% 8 weeks following transection of the CCL and immediate stabilization of the stifle in one study, despite the normal appearance of the meniscus.[31] Although weight

bearing and joint motion were not restricted following surgery, limb use was reduced and it is possible that a combination of inflammation and decreased limb use after surgery resulted in decreased stiffness. Chronic stifle instability may result in further deterioration of the meniscus because clinical cases of cranial cruciate ligament rupture (CCLR) undergoing partial meniscectomy had a 60% decrease in meniscal stiffness.

The effect of early motion on healing meniscal repairs has been evaluated in humans at the time of anterior cruciate ligament (ACL) reconstruction. In one study, 80% of patients undergoing immediate postoperative range of motion (ROM) and early partial weight bearing had complete meniscal healing with no deleterious effects of immediate knee motion on meniscal repairs.[32] Although meniscal repair is not generally performed in dogs, studies of healing medial meniscal incisions demonstrated that dogs with immediate limb mobilization had greater collagen content in the healing meniscus than those that were immobilized in a cast.[33] There were no significant differences in collagen between the repaired menisci with mobilization and the control menisci. Prolonged immobilization resulted in decreased collagen formation in healing tissue. The vascular response during meniscal healing is also affected by immobilization.[34] In a study of rabbits with meniscal injury, meniscal blood flow was increased fivefold in animals in which the stifle was allowed motion, but this increase was prevented by immobilization 4 weeks after injury. Furthermore, healing was diminished in immobilized knees.

Partial or complete meniscectomy is more commonly used to treat meniscal damage in dogs. In general, meniscal removal results in degeneration of articular cartilage, and cartilage stiffness is reduced.[35] Less severe changes occur with partial meniscal resection, but the changes are greater on the tibial surface as compared with the femoral surface.[36,37] Early joint motion may be beneficial to these patients. In studies of bilateral medial meniscectomy in dogs, one group was allowed immediate joint movement, and another group underwent knee joint immobilization with an external skeletal fixator (ESF) for 5 weeks immediately following meniscectomy. After removal of the fixation, the animals were allowed free movement for 21 weeks.[38,39] The normal menisci and the regenerated tissue at the meniscectomy sites were examined for collagen content, collagen assembly, and GAG distribution. Although the collagen content of the regenerated tissue was not different from normal menisci, collagen fiber development and GAGs were not normal. Movement following meniscectomy is likely beneficial to matrix formation in the regenerating tissue. Regarding articular cartilage, twice as much PG was present in femoral condyles of meniscectomized animals encouraged to bear weight on joints immediately after surgery compared with controls or joints immobilized after surgery.

Adaptations of Cartilage to Increased Mechanical Stress

Many reports have suggested an association between the biochemical and biomechanical properties of cartilage and the mechanical stress to which it is subjected. For example, the increased weight bearing in the limb opposite a limb casted in flexion resulted in a 19% increase in uncalcified cartilage thickness and a 25-35% increase in GAG concentration in the intermediate, deep, and calcified zones of the femoral condyles.[8] It appears that increased weight bearing augments local PG content of the articular cartilage matrix. In fact, PG content and cartilage thickness are generally greater in central weight-bearing areas than in peripheral areas, and contact forces help maintain normal cartilage matrix.[8] In general, chondroitin sulfate predominates in pressure-bearing regions, whereas dermatan sulfate predominates in tension-transmitting regions of connective tissues.[40]

The influence of static and intermittent stress on articular cartilage metabolism was examined in vitro using full-thickness plugs of cartilage from femoral condyles of normal adult dogs.[40] When cartilage was exposed to a static stress equivalent to three times body weight, or to cyclic stresses at a duty cycle of 60 seconds on–60 seconds off, net GAG synthesis was suppressed to 30-60% of that in controls. Protein synthesis also decreased, and water content increased. In contrast, when a duty cycle of 4 seconds on–11 seconds off was used, GAG synthesis was increased by 34%, but protein synthesis or water content was not affected. The changes in GAG synthesis do not appear to be due to changes in diffusion of nutrients during loading. These results suggest that regular, intermittent stresses, such as those obtained with walking, are important to maintain normal articular cartilage.

Continuous strong compression or heavy-impact loading to joints may cause deterioration of articular cartilage.[41] Blunt trauma applied to cartilage results in changes in the zone of calcified cartilage, with an increase in cellular clones, bony bar formation in the calcified cartilage, vascular invasion, and decreased PG content, thickness of the calcified cartilage, and the number of chondrocytes.[42,43] Blunt trauma to articular cartilage causes significant alterations in the deeper layers of cartilage, without disruption of the articular surface.

Effects of Training on Cartilage

The adaptation that occurs in functioning joints is known as *Leed's hypothesis,* which states that cartilage becomes conditioned to transmit, without sustaining damage, the stresses to which it is most regularly subjected.[27] Exercise places biomechanical and physiologic demands on articular cartilage.[44] Mild to moderate levels of running in dogs may stimulate adaptation, but strenuous levels can cause detrimental articular cartilage changes (Box 7-4). There

Box 7-4 Summary of Effects of Increased Mechanical Stress and Training on Cartilage

- Increased weight bearing of limb contralateral to a limb maintained in a sling may result in up to 19% increase in uncalcified cartilage thickness and 25-35% increase in PG concentration.
- Blunt trauma to cartilage may cause changes in the zone of calcified cartilage (deeper zone) without disruption of the articular cartilage.
- Mild to moderate training (running 4 to 20 km/d for 15 weeks) has no apparent deleterious effects on cartilage, but increases cartilage stiffness (6%), cartilage thickness (11-23%), and PG content (28%).
- Severe training (young dogs running 40 km/d for 1 year, or older dogs running on a treadmill at 9.6-12.8 km/h for 1 h/d for 8 months) may have adverse effects on cartilage.
  - Decreased cartilage stiffness (12-14% in some parts of the joint)
  - Decreased PG content in some parts of the joint superficial cartilage
  - Irreversible changes in collagen of cartilage
  - Increased matrix metalloproteinases
  - Potential cartilage injury, indicated by biomarkers, in the first 2 to 6 weeks of intense training, followed by repair
- Lifelong, low-impact training apparently has no deleterious effects on articular cartilage.

PG, Proteoglycan.

may be activity thresholds below and above which articular cartilage is maintained or destroyed. Between these thresholds, cartilage seems to adapt to the loads placed on it.

Intermittent compression tends to stimulate chondrocyte biosynthesis. Joint motion without compression results in articular cartilage thinning, and static loading causes decreased chondrocyte biosynthesis.[44] Cartilage subjected to high stress has higher PG content and is stiffer than cartilage exposed to low stress levels. Most studies of moderate running indicate no injury to articular cartilage, assuming there are no abnormal biomechanical stresses acting on the joints.

Mild to Moderate Training

Young beagle dogs running 4 km/d, at a speed of 4 km/h at a 15-degree incline on a treadmill for 15 weeks had no macroscopic damage to the cartilage surface and a 6% increase in mean cartilage stiffness, with most of the increase occurring on the patellar surface of the femur and the tibial condyles.[45] Cartilage thickness also increased by a mean of 11%, with increases of 19-23% in the lateral condyle and patella surface of the femur. In addition, there was a 28% increase in GAG content of the femoral condyles, located mainly in the intermediate and deep zones.[46]

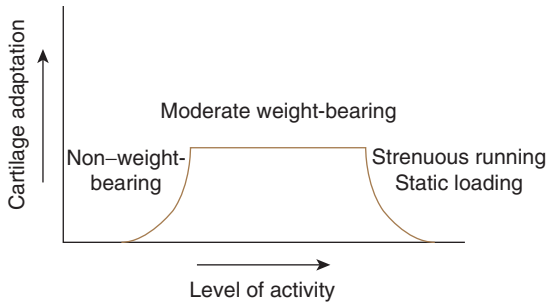


Figure 7-3 Adaptive responses of articular cartilage to activity. (Based on a diagram from Hallett and Andrish.[44])

This degree of exercise had a mildly anabolic effect on properties of cartilage. Running 20 km/d on a treadmill for 15 weeks did not further improve mechanical properties (Figure 7-3).[47]

Severe Training

Skeletally immature dogs subjected to 15 weeks of running exercise at a rate of 40 km/d had no change in cartilage total hyaluronan or PGs.[14] However, running 20 km/d for nearly 1 year resulted in a 6% reduction in thickness of the uncalcified cartilage of the medial femoral condyle, with an 11% reduction in GAG content.[48] Although tibial cartilage was stiffer, femoral condyle stiffness was unchanged with this degree of training.[47]

In similar studies of running 40 km/d, the effects of training for 1 year on young canine articular cartilage were investigated and found that the cartilage response to running training was site dependent.[49,50] Cartilage stiffness decreased 12-14% in the lateral, but not in the medial condyles of the femur and tibia, perhaps because of excessive impact loading and damage to the lateral region.[49] GAG content was depleted in the superficial weight-bearing areas of the joint, including the lateral femoral condyle.[50] There were slight increases in the thickness of the uncalcified and calcified cartilage, and the subchondral bone.[51] There also appeared to be reorientation of the superficial zone collagen network.[52] Although no overt cartilage damage was seen, softening of the cartilage may jeopardize the ability of cartilage to maintain its normal structural and functional properties over time. The response to long-term exercise may be different in other joints. Although there was a decrease in GAG content and cartilage thickness in the head of the humerus following training, cartilage stiffness was not affected.[49,50] The articular cartilage of the femoral head appears to adapt very well to long-term distance training.[53]

Strenuous training protocols in older dogs may result in deleterious changes to cartilage. Running aged dogs on a treadmill at 9.6-12.8 km/h for 1 h/d, 6 d/week for 8 months led to matrix degradation in the femoral head. PG content was decreased, and there was irreversible destruction of

collagen fibrils, with erosion and fibrillation of the cartilage surface. Older dogs may be more vulnerable to mechanical stress changes than younger animals.[54]

A study attempted to use magnetic resonance imaging (MRI) of cartilage and serum and synovial fluid markers of cartilage damage to determine if training injury to cartilage could be detected and monitored in dogs training at different intensities.[55] Three groups were evaluated: (1) a training group that ran on a slope of 10 degrees at a speed of 200 m/min, (2) an intensified training group that ran on a slope of 10 degrees at a speed of 300 m/min, and (3) a control group that was allowed unrestricted cage activity. Dogs in the training and intensified groups were trained daily, running for an hour in the morning and in the afternoon for 10 weeks. Changes in the MRI examination suggestive of early injury of articular cartilage were noted in the training and intensified groups as early as 2 weeks of training. Damage was most severe at 4 to 6 weeks, and then gradually improved. There were no differences between the two training groups. Elevations of cartilage oligomeric matrix protein, matrix metalloproteinases 1 and 3, and tissue inhibitor of matrix metalloproteinase 1 in serum and synovial fluid were seen in both training groups. Histologic examinations at the end of training demonstrated cartilage damage and repair in the stifle joints in the training and intensified groups; furthermore, the intensified group indicated more remodeling of articular cartilage. The authors concluded that high-intensity training may induce injury, followed by a repair process. In addition, changes in biomarkers reflected pathologic changes in the cartilage better than MRI.

Lifelong Training

One study evaluated the effect of lifelong low-impact exercise on articular cartilage.[56] Dogs were exercised on a treadmill at 3 km/h for 75 min, 5 d/wk for 527 weeks while wearing jackets so that the total weight carried was 130% of body weight. There were no ligament, meniscal, or cartilage injuries, or osteophytes found on necropsy. Biochemical, cartilage thickness, and biomechanical properties were not affected by training. These results suggest that a lifetime of relatively low-impact weight-bearing exercise in dogs with normal joints does not cause alterations in the structure and mechanical properties of articular cartilage that might lead to joint degeneration.[56]

Effects of Exercise on Cartilage during Acute Inflammation

Excessive exercise during periods of acute joint inflammation may exacerbate synovitis. Passively exercising the stifle joints of dogs in a model of acute inflammation for 5 seconds every 15 minutes for 4 hours resulted in a twofold increase in the synovial fluid leukocyte count, whereas exercise for 5 minutes every 15 minutes for 4 hours produced a ninefold increase in inflamed joints.[57] Forms of acute inflammatory arthritis may require periods of physical inactivity for recovery. Observations of humans with rheumatoid arthritis suggest that excessive activity may increase the severity of the disease.[58] Immobilization was chondroprotective to guinea pig stifles following an intraarticular injection of iodoacetate. Animals that were not immobilized had decreased GAG content and a 10-20% reduction in the number of chondrocytes 1 week after injection. Three weeks after injection, cell death and loss of GAG progressed, and surface fibrillation and osteophytes developed. Although the loss of GAGs was not altered by immobilization after iodoacetate injection, the depletion of chondrocytes was reduced. Furthermore, neither osteophytes nor fibrillation developed.[59] Therefore immobilization may be protective against exacerbation of synovitis during acute bouts of inflammatory joint disease.

However, isometric exercise does not appear to increase joint inflammation.[60] The effects of passive range of motion and isometric exercises on experimental inflammatory arthritis in a rabbit model were assessed. Passive range of motion significantly increased synovial leukocyte counts over control values of the nonexercised joints, but isometric exercises across a joint with acute synovitis did not affect synovial leukocyte count. Another factor to consider is the increased synovial cell count in joint motion as compared to the overall deleterious effects of immobilization on articular cartilage. A study of acute crystal-induced joint inflammation in rabbit knees compared the effects of joint movement with immobilization.[61] Over 42 days, immobilized knees had greater cartilage histologic abnormalities and loss of GAGs compared with exercised joints. Overall, joint motion enhanced the synovitis, but joint immobilization had more adverse effects on articular cartilage. Another study of a rabbit antigen-induced synovitis model had similar findings, with less articular cartilage damage at the end of the 6-week study in those rabbits that had early continuous passive range of motion as compared to those with immobilized knees.[62] Therefore, it seems prudent to allow active motion of inflamed joints, with reasonable caution. Aquatic exercise may be appropriate to reduce weight-bearing stresses on articular cartilage while allowing active joint motion.

Abnormal Biomechanical Stresses

Although moderate training programs appear to have no detrimental effects on articular cartilage in animals with normal joints, training program may be deleterious to articular cartilage in dogs with abnormal biomechanical stresses acting on joints. Transection of the CCL is a common model to produce OA. OA occurs as a result of joint instability during weight bearing. Joint immobilization may be somewhat protective against OA development. In a CCL transection model, the stifle joint of one group was immobilized in a cast for 12 weeks, and the other group had no immobilization for 12 weeks.[22] Stifles of

dogs with CCL transection without immobilization had changes typical of OA, including increased water content and PG synthesis, and decreased PG content, but the cartilage thickness was normal. Osteophytes were not seen with CCL transection and immobilization, but the cartilage was atrophic (40% thinner), water content was increased, and PG content and synthesis were diminished. These data suggest that OA develops with cruciate ligament transection as a result of mechanical instability. If the knee is immobilized after cruciate transection so that loading is reduced, OA does not develop in the short term.

Effects of Medications on Immobilized Cartilage

Medications are commonly prescribed for dogs with joint injuries. Many of these drugs may have deleterious effects on articular cartilage metabolism, especially if the joint is immobilized. For example, fluoroquinolones, such as enrofloxacin and ciprofloxacin, may be toxic to chondrocytes, resulting in cellular changes that alter cell adhesion.[63] Knowledge of these effects may result in more effective rehabilitation strategies to minimize damage to cartilage. Alternatively, some drugs may provide beneficial effects to immobilized cartilage.

Nonsteroidal antiinflammatory drugs (NSAIDs), such as aspirin, are commonly used in the management of OA. Aspirin may suppress PG synthesis in normal canine knee cartilage, as well as in early OA cartilage, such as that following CCL transection.[64,68] Aspirin may affect GAG synthesis by inhibiting uridine diphosphate-glucose dehydrogenase, an enzyme important in the synthesis of chondroitin sulfate.[64] Uptake of acetylsalicylic acid increases by 35% in osteoarthritic cartilage, suggesting that the drug permeates it more readily than in normal cartilage. The lower PG content of OA cartilage may be more important than fibrillation or surface disruption in rendering it vulnerable to aspirin.[69] In addition, aspirin suppresses GAG synthesis in unloaded cartilage to a much greater extent *in vitro* than it does in loaded cartilage.[70,71] Aspirin also markedly reduces prostaglandin E_2 synthesis by the cartilage, and drug concentrations in cartilage from loaded zones are lower than those in cartilage from unloaded sites.

Aspirin administration aggravates the changes that occur in cartilage during limb immobilization. In one study, a high dose of aspirin (40 mg/kg tid) was administered daily for 6 weeks while one hindlimb was immobilized in a cast.[69] Whereas the cartilage of contralateral nonimmobilized limbs in dogs receiving aspirin had normal GAG content, the decreases in GAG content and net PG synthesis were significantly greater in immobilized stifles receiving aspirin as compared with immobilized stifles alone. Aspirin also apparently causes synthesis of type I rather than type II collagen by atrophic articular cartilage during immobilization.[70] Although these results indicate that aspirin has an adverse effect on articular cartilage of immobilized joints, aspirin administration did not preclude reversal of these changes if the dog was allowed to walk freely for 3 weeks after cast removal.[69] However, if dogs ran daily on a treadmill for 3 weeks after cast removal, the decrease in GAG content of immobilized knees persisted and actually worsened in dogs receiving aspirin.

Although high doses of aspirin may have detrimental effects on cartilage, other NSAIDs apparently have little or no negative effects at doses that achieve expected levels in synovial fluid. In fact some NSAIDs may be somewhat chondroprotective, including carprofen,[71,72] deracoxib,[73,74] and etodolac.[75,76]

Treatment with corticosteroids may also have detrimental effects on normal cartilage and cartilage of immobilized limbs. Prednisone given to normal dogs with immobilized stifle joints resulted in a 58% loss of GAG content in one study.[77] In nonimmobilized joints of dogs receiving prednisone, GAG content decreased 11-31%, compared with controls.

The use of HA in dogs may have some benefit to articular cartilage following immobilization. In one study, stifles were immobilized for 4 weeks using transarticular external skeletal fixation.[78] Treated dogs received HA. The PG content was reduced after immobilization in all dogs. Remobilization of the stifles was associated with damage to the surface and tangential layers of cartilage. However, remobilization with HA improved PG content and reduced structural damage. In another study, casts were placed on the hindlimbs of dogs for 92 days. Beginning on day 56, dogs received intraarticular injections of HA. Injections were repeated at 4-day intervals until the end of the study. Although femoral condylar articular cartilage had decreased PG content in all cast groups, the decrease was less in the HA-treated group. HA may inhibit inflammatory mediators, such as tumor necrosis factor-alpha (TNF-α) and stromelysin.[79] Other studies have indicated that HA may not be beneficial for treatment of OA, however.[80]

Joint Capsule

Changes in Joint Capsule with Immobilization

Joint capsules are also affected by immobilization and remobilization. The functional and structural consequences of immobilization and remobilization of the glenohumeral joint after 12 weeks of immobilization were studied in 10 beagle dogs.[81] One forelimb was immobilized in a cast. The cast was removed and remobilization was allowed for 4, 8, and 12 weeks. After 12 weeks of immobilization, the passive ROM was markedly impaired, intraarticular pressure was increased during movement, and the filling volume of the joint cavity was reduced. There was synovial lining hyperplasia and vascular proliferation in the wall of the joint capsule, but there was no

increase of fibrous collagen in the capsular wall. Both the functional and structural changes were unaltered after 4 weeks of remobilization, but after 8 weeks they began to reverse, and they returned to normal levels after 12 weeks. Both functional and structural changes after 12 weeks of immobilization of a normal glenohumeral joint are reversible by remobilization. The collagen composition of the capsule seems unrelated to the degree of capsular contraction that occurs during 12 weeks of immobilization.

Similar changes were observed in the carpus and elbow in dogs in which normal forelimbs were immobilized for 16 weeks.[82] All dogs had a 20-30% decrease in ROM in the elbow and carpal joints, which returned to normal within 6 weeks after remobilization. In addition, all dogs were significantly lame immediately following immobilization, but returned to soundness within 6 weeks after remobilization began. Limbs casted for 32 weeks had severe joint stiffness and muscle atrophy, and recovery of normal limb use was delayed by several weeks.[83] Deficits were also present after only 12 weeks of immobilization, but full function resumed much sooner.

It is helpful to know which tissues contribute to joint contractures following prolonged immobilization. One study evaluated contractures in a rat model by immobilizing one knee in flexion.[84] After 8 weeks, the animals were allowed to resume unrestricted activity for 4 weeks. Interestingly, 88% of the contractures remained in the immobilized group after dividing skin and muscle, suggesting an important contribution of the caudal knee capsule in limiting knee mobility. In fact, another model in rats confirmed the role of the joint capsule in contracture during knee joint immobilization.[85] Joint contracture rapidly progressed after joint immobilization until 8 weeks and advanced more slowly after 8 weeks, suggesting that joint contracture develops early in immobilization and progresses over time. After releasing the caudal capsule, the immobilized group regained knee extension.

The contribution of collagen content as a specific cause of joint capsule contracture is somewhat controversial. Type I collagen composes approximately 85% of the collagen content of the joint capsule, and provides mechanical strength, contributes to tissue elasticity, and is produced in sites undergoing fibrosis.[86] Collagen type III may be present in tissues requiring high levels of mechanical compliance. It is a constituent of normal synovial tissue, and is also present in inflamed and proliferative rheumatoid synovium. Some studies have shown an increase in joint capsule collagen content, especially with concurrent joint injury,[87-89] whereas others have shown no change or even a decrease in collagen content with joint immobilization. In a rat knee immobilization model, mRNA of type I collagen decreased rapidly after immobilization, but the immunoreactivity of the capsule was not changed in the immobilized or the control groups at any time points.[90] In another study, the collagen of control and immobilized periarticular connective tissue was type I collagen, whereas type III collagen, which occurs in response to inflammation, was not present.[91] The periarticular connective tissue contracture process is apparently different from the intraarticular process in which fibrofatty connective tissue and type III collagen proliferation are prominent. A later study confirmed that types I and III collagen were not increased in the joint capsule following immobilization of rat knees, suggesting that another process, such as synovial or capsular adhesions, was responsible for joint contracture.[89] In addition to potential changes in collagen content, GAG and water content decrease in the joint capsule and synovium with immobilization.[91] This may reduce the plasticity and lubrication efficiency of these tissues.

Changes of the joint capsule undergoing contracture may also be related to growth factors. Joint capsule may produce transforming growth factor–beta1 (TGF-β_1) and connective tissue growth factor (CTGF) after immobilization, and these growth factors may play a role in causing and maintaining capsular changes. Collagen synthesis and myoblast induction are related to TGF-β, and CTGF is regulated by TGF-β.[92,93] CTGF also induces type I collagen formation. In a flexion knee immobilization model in rats, TGF-β_1, CTGF, and fibrotic change were greater in the caudal capsule and may have resulted in limited motion in extension.[92] Another study of joint injury followed by immobilization indicated similar increases in mRNA and protein levels for collagen types I and III, matrix metalloproteinases 1 and 13, and TGF-β_1 were significantly greater in immobilized capsules compared with controls, whereas tissue inhibitor of matrix metalloproteinases were significantly less in experimental capsules.[93] It may be possible to prevent joint contractures by somehow blocking these growth factors. In another study, although both TGF-β_1 and CTGF were increased in the joint capsule after immobilization, collagen type I was not increased.[93]

The specific cause of joint capsule contracture may be associated with the proliferation of myofibroblasts in the joint capsule following immobilization. In one study rabbit knee joints were rigidly immobilized in maximum flexion.[93] After 8 weeks groups were allowed periods of remobilization for 0, 8, 16, or 32 weeks. The average flexion contracture of knees was greater in the remobilization groups as compared with nonimmobilized controls, especially during early mobilization. Myofibroblast numbers in the posterior joint capsules were elevated four- to fivefold in the knees with contractures when compared with the contralateral knees, suggesting that these cells may be involved with joint contracture. A follow-up study indicated that these changes occur relatively soon after immobilization, and the changes persist for some time.[93] Differentiated myofibroblasts are characterized by the expression of alpha–smooth muscle actin (α-SMA), a protein associated with the contractile properties of this cell.[94]

Mast cells and neuropeptide nerve fiber numbers may also be involved in joint capsule contracture because elevated numbers of these cells have been implicated in other fibrotic and contracture conditions. In a posttraumatic contracture model in rabbits, the knee joint was immobilized.[95] Rabbits were sacrificed 4 or 40 weeks after surgery (including 32 weeks of remobilization). Myofibroblasts, mast cells, and nerve fibers were four to five times greater in the contracture capsules when compared with the control capsules at all time points, suggesting myofibroblasts, mast cells, or neuropeptide influences may underlie some of the pathologic changes in the joint capsule following trauma. In addition, the neuropeptides substance P and calcitonin gene–related peptide can cause mast cell degranulation, and this may contribute to neurogenic inflammation, pain, and swelling.[96]

Remobilization of Joint Capsule Immobilization

The form of joint mobilization may be important in restoring joint motion after a period of rehabilitation. The effect of low torque–long duration stretching to increase the ROM and to restore the mechanical properties of contracted joints was studied in rat knees immobilized for 40 days.[97] After remobilization, the contracted knees were placed in one of four treatment groups treated with repeated stretches of diverse torques and duration: stretching with low torque (0.02 Nm) and long duration (40 minutes), high torque (0.045 Nm) and short duration (20 minutes), high torque and long duration, and low torque and short duration. Animals received three sessions a week for 4 weeks, for a total of 12 treatment sessions. Low-torque and long-duration repeated stretching resulted in greater restoration of ROM with more normal mechanical properties compared with high-torque and short-duration stretching.

Heat may be useful to improve motion in joints that have contracture after immobilization. Infrared and ultrasonic heat therapies were compared in a rat knee flexion contracture model.[98] Six treatment sessions of stretching with infrared or ultrasound over a 2-week period were more effective than stretching without heat, but there was no significant differences between the two heat treatment groups.

Regular therapeutic exercise is also important to recover joint motion after immobilization. Joint ROM was reduced by 46% in a 2-week ankle joint immobilization model in rats.[99] Immobilization was followed by free cage activity and free remobilization or treadmill running once a week, three times a week, or six times a week. Ankle joint motion was improved in all three treadmill exercise groups in comparison with the free remobilization group, with running three and six times a week being more beneficial for recovery of joint contracture compared with no running or once-a-week running (Box 7-5).

Box 7-5 Summary of Effects of Disuse and Remobilization on Joint Capsule

- Immobilization may result in reduced (20-30%) passive range of motion, lameness, muscle atrophy, increased intraarticular pressure during joint motion, reduced filling volume of the joint, synovial hyperplasia, and vascular proliferation of the joint capsule.
- Improvement may occur 4 to 8 weeks after remobilization.
- Contracture of joints during immobilization occurs relatively rapidly in the first 4 to 8 weeks and involves the flexor side of the joint capsule.
- The role of collagen in joint capsule contracture is controversial.
- TGF-β_1, CTGF, and matrix metalloproteinases may be involved in joint contracture.
- Myofibroblasts may be the cells that result in joint capsule contracture (may increase four- to five-fold)
  - Changes occur relatively soon after immobilization
  - α-SMA, a protein associated with the contractile properties of this cell, is expressed.
- Mast cells and neuropeptide fibers are also increased four- to five-fold with joint capsule contracture.
- Low torque, long duration stretching over time may be more effective in improving joint capsule contracture.
  - Stretching with heat application may be more effective.
- Frequent, regular therapeutic exercises are more effective in improving joint mobility than free activity.
- The effects of immobilization on joint capsule may be improved by the use of intraarticular sodium hyaluronate, cyclooxygenase-2 inhibitors (reduces the expression of α-SMA), and medications that inhibit mast cell degranulation.

α-SMA, Alpha–smooth muscle actin; *CTGF,* connective tissue growth factor; *TGF,* transforming growth factor.

Medications for Joint Capsule Immobilization

Although evidence exists for improving joint capsule stiffness using physical modalities, little is known about pharmacologic intervention to improve joint capsule changes as a result of immobilization. However, intraarticular sodium hyaluronate may be of benefit. Rabbit knee joints were immobilized in one study.[100] A 1% solution of sodium HA was administered intraarticularly at a dosage of 0.1 mL/kg body weight twice weekly. Changes in joint contracture, articular cartilage, and synovial membrane were compared with a saline control group during a 6-week immobilization period. In all groups, joint contracture and articular cartilage degeneration progressed, but were inhibited by HA. In addition, the effects on decreasing joint contracture and articular cartilage degeneration were more potent with higher-molecular-weight HA. Inhibition of cyclooxygenase-2 (COX-2) with NSAIDs may downregulate the expression of α-SMA, a protein associated with the contractile function of myofibroblasts.[93]

Models of joint contracture have shown an increase in mast cells in fibrotic tissue, suggesting that mast cells may be important in joint capsule stiffness. Mast cells synthesize and secrete proteases, growth factors, and cytokines, many of which have mitogenic effects on fibroblasts and myofibroblasts.[101] Medications that inhibit mast cell activation, such as ketotifen fumarate, have antiallergic and antihistamine properties. In particular, ketotifen noncompetitively blocks the binding of histamine to its H1 receptor and inhibits calcium-dependent vesicle degranulation of activated mast cells (mast cell stabilization).[102] In a rabbit posttraumatic joint contracture model, animals treated with ketotifen had reductions in the severity of joint contractures and the degree of joint capsule myofibroblast and mast cell hyperplasia.[102] However, contractures in the ketotifen-treated groups were not completely reversed as compared with nonoperative controls. Substance P, a neuropeptide that is an important mediator in the transmission of pain, is released into joints and is an important proinflammatory agent. Substance P is also a potent mast cell activator and is secreted in response to pain and injury. One study has documented elevated substance P in nerve fibers in contracted joint capsules of humans and rabbits. Increased expression of substance P has also been documented in other models of fibrosis, suggesting that this may be another target to modulate capsular stiffness.[95] Due consideration for the potential for delayed wound healing, including bone healing, should be made if considering treatments that inhibit mast cell function.

Muscle

Canine Muscle Fiber Types

Several types of muscle fibers exist, which perform somewhat specialized functions. Traditionally, they have been classified as two general types: type I (slow twitch) and type II (fast twitch) fibers (Table 7-1). Type I fibers are more adapted for oxidative metabolism and functions, such as maintaining posture, whereas type II fibers are associated with glycolytic metabolism and the generation of power and speed during muscle contraction. Fiber types previously identified in immature and mature canine skeletal muscles include types I, IIA, and IIC, with the latter representing less than 10% of the total fiber population.[103-105] Type IIC fibers likely represent a transitional fiber between types I and II and are precursors to type IIA and IIB fibers in neonatal animals. Type IIA fibers have more oxidative capacity, but they still function in the generation of speed and power. Type IIB fibers have less oxidative capacity. The presence of true type IIB fibers has been debated in the dog, and type IIDog fibers, which are peculiar to the dog, or type IIX fibers have been suggested as a fiber type that is similar to but does not correspond exactly with classical type IIB fibers of other species.[105,106] True type IIB fibers in the dog may only be present in the laryngeal muscles. The interpretation of fiber type composition depends on the staining and incubation methods used.[105] Type IIB or type IIDog fibers are not found in all muscles

Table 7-1 Canine Muscle Fiber Types

SLOW TWITCH OXIDATIVE FIBERS	
Type I	Greatest oxidative capacity
FAST TWITCH GLYCOLYTIC FIBERS	
Type II	
Type IIA	Greater oxidative power than IIB, but function in generation of speed and power
Type IIB	Classic fiber type in other animals, least oxidative. True type IIB fibers may not exist in dogs except in the laryngeal muscles.
Type IIC	May be transitional fiber between types I and II, precursors to type IIA and IIB in neonatal animals
Type IIDog	System that recognizes this as a peculiar fiber type to dogs, similar to type IIB in other species
Type IIX	System that recognizes this as a fiber type in dogs similar to, but not exactly corresponding with, type IIB fibers in other species. Rapidly contracting, greatest cross-ssectional area, lowest concentration of capillaries and muscle nuclei. Still has high oxidative capacity compared to other species (intermediate between types I and IIA of other species), but is the least oxidative muscle fiber type in dogs.
HYBRID FIBERS	
Type I+IIA	Second greatest oxidative capacity
Type IIAX	Intermediate between fiber types IIA and IIX

and were biologically significant only in the semitendinosus muscle in one study.[104] The metabolic potential of these fibers is fairly similar to that of IIA fibers, but significantly different to that of IIB fibers in other mammals, suggesting that they may be designed to play a different role during locomotion. All canine muscle fibers have an abundant capillary supply and moderate to high oxidative capacity, which may be related to the extraordinary athletic capability of dogs.

Recent methods of assessing muscle fiber types have incorporated immunohistochemical analysis of myosin heavy chain content of muscle fiber types rather than relying strictly on enzymatic staining. This technology has suggested that there are several muscle fiber types in dogs, with some of these being hybrid muscle fiber types.[107] It is believed that these hybrid fibers allow fine-tuning activity to allow for different contraction forces, contraction velocities, and resistance to fatigue. The main fiber type isoforms are type I, IIA, and IIX, which have been identified in previous studies. Hybrid fiber types include type I + IIA and IIAX. Small amounts of type IIB fibers have been found in laryngeal muscles. Type IIX fibers are rapidly contracting fibers and have the greatest cross-sectional area and lowest concentration of capillaries and muscle nuclei. Nevertheless, type IIX fibers are more oxidative than other species, and it is this unusually high oxidative capacity that is probably responsible for the high endurance ability of dogs. Oxidative capacity decreases significantly in the order I > I + IIA > IIA > IIAX > IIX. Unlike other species, IIX fibers appear to be intermediate between type I and IIA fibers regarding their contractile, metabolic, and morphologic features.[107]

One study of the semitendinosus muscle found that hound-type dogs had the largest muscle fibers, but the fiber-type percentages were similar compared with mixed-breed and beagle dogs with similar activity levels.[108] In general, there are no differences between the left and right limb muscles, but there may be significant differences in the percentages of muscle fiber types between individuals of the same breed for the same muscle.[109] Other studies have shown that greyhounds have greater fiber size and a higher percentage of type II fibers than mixed-breed dogs, presumably because of the demand for intense physical activity.[110] However, there were no differences in fiber types in trained versus untrained Greyhounds, indicating that the distribution of fiber types may depend more on breed and genetics than level of training for this breed.

The size of muscles may be genetically related to a negative regulator of skeletal muscle growth, myostatin, a member of the TGF-β family of growth and differentiation factors.[111] Decreased expression of myostatin as a result of genetic mutation results in larger muscle mass in knockout mice and double-muscled cattle. Recently, a mutation in the myostatin gene was found in some whippet dogs.[112] Dogs with this mutation, known as "bully" whippets, are more muscular and faster in racing events. However, some dogs with this trait have muscle cramping in the shoulder and thigh and do not conform to the American Kennel Club breed standards; approximately 50% of affected dogs have an overbite.[112] The role of myostatin in resistance training and subsequent detraining has been studied in men.[113] After 30 or 90 days of resistance training, followed by 3 to 90 days of detraining, myostatin RNA levels in muscle biopsies significantly increased. In addition, type II muscle fibers began to atrophy after 3 days, with half of the acquired fiber area lost after 10 days of detraining. Myostatin may play a role in muscle atrophy with immobilization. In addition, there is a different amount of myostatin in different muscles. In people, the greatest amount of myostatin is found in quadriceps muscle fibers containing 100% type IIB fibers, whereas the soleus muscle does not have any appreciable type IIB muscle fibers or myostatin mRNA.[114]

Different muscle groups have variable muscle fiber type patterns that may be due to function and use (Table 7-2).[110,115] In fact, muscle fiber type may change if muscle

Table 7-2 Muscle Fiber Types in Various Muscles of Adult, Mixed-Breed Dogs

Muscle	%	Muscle	Fiber	Types	
	I	I+IIA	IIA	IIAX	IIX
Semitendinosus	11.6	5.8	36.1	38.4	8.1
Latissimus dorsi	18.1	2.3	31.4	32.9	15.2
Gluteus medius	19.9	3.8	48.1	22.9	5.3
Triceps brachii	27.1	4.2	48.3	20.3	0
Longissimus lumborum	38.4	5.1	46.5	10.1	0
Rectus femoris	48.1	2.6	32.5	16.9	0
Vastus intermedius	65.3	1.0	31.6	2.0	0
All muscles	27.1	3.1	37.1	24.8	7.9

Adapted from Acevedo LM, Rivero JL: New insights into skeletal muscle fibre types in the dog with particular focus towards hybrid myosin phenotypes. *Cell Tissue Res* 323:283-303, 2006.

function is altered by transposing muscles. In one study, the fibularis longus tendon was severed and surgically transferred to the tendon of the cranial tibial muscle, a functionally different muscle.[115] Later muscle biopsies demonstrated an increase in type I and type IIB fibers, which was similar to the fiber composition of the cranial tibial muscle. These findings indicate that muscle fiber remodeling may occur and suggests that changes in muscle fiber type are manifestations of functional adaptation.

Muscle biopsy technique may affect the results of muscle fiber typing.[109,116] For example, the percentage of type I fibers in the cranial tibial and semitendinosus muscles progressively increases from the superficial and middle regions to the deepest portion. Although the distance of the section from the origin of the muscle does not significantly affect the mean percentage of fiber types, the variation in fiber types varies less deeper within the muscle, so a needle biopsy taken from deep within muscle should provide a more consistent and reliable estimate of fiber type proportion than a superficial specimen.

Factors Affecting Muscle Contraction

Skeletal Muscle Blood Flow

The ability of muscles to contract depends on blood flow to the muscle, primarily because of the oxygen delivered to the working muscle from the red blood cells. Small decreases in blood flow are associated with reduced contraction strength. When muscles contract, muscle blood flow typically increases, depending on the metabolic rate that is established by the contraction pattern and frequency. The ability of canine oxidative skeletal muscle to maintain isometric force is limited by blood flow.[117] This limitation implies a mismatching of blood flow and the metabolic rate. The blood flow past a muscle cell is determined by the number of capillaries per fiber, the vascular conductance, the capillaries perfused, and vascular autoregulation. The resulting mismatching of flow and metabolism accelerates the fatigue process.

The frequency of muscle contraction and tension development affects blood flow during muscle contractions.[118] The magnitude of the blood flow response to maximal exercise in different muscles is extremely varied. For example, skeletal muscle blood flow increased progressively up to maximal oxygen uptake in the gracilis, semitendinosus, and semimembranosus muscles of dogs in one study.[119] However, the blood flow response to maximal exercise leveled off during submaximal exercise in the gastrocnemius muscle. In addition, there are species differences in skeletal muscle blood flow.[120] Muscle blood flow can reach 300-400 mL/100 g of tissue/min in dogs, ponies, and rats, whereas it reaches only 75 mL/100 g of tissue/min in a 75-kg man. The role of the diffusion gradient of oxygen from the blood to muscle cells is also important in exercising muscle.[121]

Muscle capillarity decreases in aged dogs.[122] Endurance training, however, enhances capillarity, and old rats and humans may attain levels of capillarity comparable to their active young counterparts, even when performing considerably less exercise.[123] In addition, there were no age-related effects on blood flow to the triceps, deltoideus, flexor carpi ulnaris, superficial digital flexor, gastrocnemius, gracilis, semimembranosus, and semitendinosus muscles of dogs during maximal exercise on a treadmill.[123]

Skeletal Muscle Oxidative Capacity

The ability of muscles to function depends on oxygen delivery and utilization by muscles. Oxygen uptake by the gastrocnemius muscle group of dogs appears to be linearly related to external work rate and to the load against which the muscles shorten.[123] The major contribution to oxygen uptake during contraction appears to be the number of contracting muscle fibers. Oxygen uptake of muscle also increases with stimulation frequency, reaching a peak at five twitches per second.[124] Further increases in stimulation frequency result in lower oxygen uptake, perhaps secondary to mechanical restraints imposed by contraction duty cycle and vascular compression. However, muscle contractions may have only a small direct effect on muscle blood flow. Their main effect may be to reduce venous pressures.[125] The limiting factors for skeletal muscle oxygen uptake vary according to the intensity of muscle contractions.[126] During transitions from rest to contractions of dog gastrocnemius muscles with 70% of peak oxygen uptake, oxygen delivery to muscle, intramuscular blood flow, and peripheral oxygen diffusion are not limiting factors for skeletal muscle oxygen uptake kinetics. An increase in capillary density (reflecting a reduced diffusion distance caused by reduced fiber size) also may not increase the muscle oxygen diffusing capacity, suggesting that diffusion distance is not a primary determinant of muscle oxygen diffusing capacity.[127] Therefore oxygen uptake may be mainly determined by intrinsic skeletal muscle oxidative metabolism.

Maximum oxygen uptake is limited by oxygen delivery as a result of a limited and uneven distribution of muscle blood flow. When oxygen delivery is reduced, hypoxia results in reduction of muscle performance. Endurance training results in an increased number of capillaries in muscle over time. Hypoxia enhances angiogenesis in canine skeletal muscle after endurance exercise, whereas lactic acid appears to inhibit vascular endothelial cells.[128] Increased oxygen delivery to muscle may decrease fatigue. Metabolic rates can be increased by increasing oxygen supply.[121] During high metabolic rate isotonic tetanic contractions, muscle fatigue was diminished by polycythemia, but the effect appeared to be transient.[129] Norepinephrine can produce large increases in muscle recovery oxygen consumption, whereas β-blockers, such as propranolol, reduce the rate of oxygen uptake during exercise and postexercise recovery.[130] Catecholamines may make a significant contribution to postexercise recovery oxygen consumption.

Oxidative capacity may decline in many muscles of sedentary individuals. Endurance training can greatly improve endurance in old age with an increase in muscle oxidative capacity.[131] With endurance training, old individuals may attain levels of muscle oxidative capacity similar to those in identically training young individuals.

Skeletal Muscle Carbohydrate Metabolism

Muscle fatigue may be the result of several factors, with the relative contribution of each dependent on the condition and nature of the exercise, including the intensity, duration, muscle mass, and energy substrate. Fatigue may occur as the result of accumulation of hydrogen ions, inorganic phosphate, substrate depletion, and alterations in calcium ion function.[132] Adequate cellular oxygen content and maintenance of adenosine triphosphate (ATP) levels are critical to avoid fatigue.

An increase in muscle oxidative capacity contributes to reduced glycogen depletion. Muscle glucose transport is enhanced after a bout of exercise, regardless of age.[131] Muscle glycogen depletion, creatine phosphate depletion, and lactate accumulation during contractile activity are exaggerated in older animals, apparently secondary to reduced muscle oxidative capacity and blood flow. Resting muscle glycogen concentration is diminished in older humans, probably in part because of a more sedentary lifestyle. Although several months of endurance training raise muscle glycogen concentration in older people, levels still remain below those of younger individuals.

Studies of the role of intracellular oxygen on the efflux of lactate from skeletal muscle during graded exercise from submaximal to maximal effort indicate that a constant intracellular oxygen content is unrelated to increasing lactate efflux.[121] Furthermore, muscle tension development is reduced by lactate.[132] This effect of the lactate ion appears to be independent of pH.

Aging

Muscle mass typically declines in old age, secondary to muscle fiber atrophy.[131] The loss of strength in old age is predominantly accounted for by reduced muscle mass. Although there are no apparent effects of aging on muscle fiber type percentages in dogs, there is a 25% reduction of cross-sectional area of type II fibers in aged dogs.[122] The cross-sectional area of type I fibers is relatively unchanged, however, and may actually increase in some muscles. The mechanism for age-related atrophy is unknown, but may be related to a loss in the number of alpha motoneurons.[131]

Heart Disease and Skeletal Muscle

Patients with chronic heart failure may be limited by exertional fatigue. Changes in skeletal muscle may contribute to this fatigue, because biochemical and histologic abnormalities of skeletal muscle may develop in patients with chronic heart failure. Dogs with chronic heart failure have changes in skeletal muscle fiber type, fiber size, and fiber ultrastructural properties.[133,134] Skeletal muscle weights, muscle fiber area, and the percentage of skeletal muscle type I fibers may be reduced, whereas the percentage of skeletal muscle type II fibers may be increased with heart failure. There are apparently no ultrastructural abnormalities, no differences in developed tension per gram muscle during stimulation, maximal developed tension, muscle fatigability, or preferential atrophy or hypertrophy of either muscle fiber type in skeletal muscle of dogs with heart failure. Although skeletal muscle atrophy occurs with heart failure, the remaining muscle appears to exhibit normal performance and metabolism. These changes may be the result of adaptations of skeletal muscle to anaerobic metabolism.

Response of Muscle to Disuse and Immobilization

The muscles most vulnerable to disuse atrophy are the postural muscles that contain a relatively large proportion of type I (slow-twitch) muscle fibers, extensor muscles, and muscles that cross a single joint (Box 7-6). Conversely, those least susceptible to atrophy are those not used as postural muscles (antagonist muscles), flexor muscles, muscles that cross multiple joints, and muscles that are predominantly composed of type II (fast-twitch) muscle fibers.[58,135] Immobilization reduces the chronic load on these muscles (Figure 7-4). Muscle strength decreases rapidly during the first week of immobilization, with further losses occurring more gradually over time.[136] Loss of muscle force production is not entirely explained by muscle atrophy alone. There may be up to a 50% reduction in peak force, even when the muscle mass has been normalized to constant muscle mass. This suggests that a cellular component may be involved, such as an alteration in sarcoplasmic reticulum function. In addition, there is decreased mitochondrial function and reduced protein synthesis. Interestingly, there is often an increase in capillary density and sometimes blood flow to muscles undergoing atrophy.[127,137,138] Removal of weight-bearing activity has less effect on type II muscle fibers because these do not perform a major function in maintaining posture. In fact, with reduced stress, there may be an increase in type II muscle fibers. This results in increased maximum muscle velocity, not only in type II muscle fibers, but also in type I muscle fibers. These changes help attenuate a decline in power output as a result of atrophy.

The length at which the muscle is immobilized also affects the degree of atrophy, with muscles immobilized in a shortened position atrophying to a greater degree, with reduced force generating capacity and oxidative activity. When a muscle is immobilized in a shortened position, the number of sarcomeres decreases, leading to reduced muscle fiber length. If the muscle is immobilized in a

Box 7-6 Summary of the Effects of Disuse and Immobilization on Skeletal Muscle

- Postural type I muscle fibers, extensor muscles, and muscles that cross a single joint are at greatest risk for atrophy.
- Immobilization with the muscle in a shortened position results in greater atrophy.
- Muscle strength decreases rapidly during the first week of immobilization, with further losses occurring more gradually.
- Loss of muscle force production not entirely explained by muscle atrophy alone.
- Alteration in sarcoplasmic reticulum function occurs.
- Sarcoplasmic reticulum calcium uptake is decreased.
- Mitochondrial function is decreased.
- Protein synthesis is reduced.
- Oxidative and glycolytic enzyme activities are reduced and are associated with reduced capacity for energy production.
- Responsiveness to insulin is reduced, which affects ability of muscle to take up glucose.
- Ca-ATPase activity is reduced following immobilization.
- Muscle atrophy is an imbalance of protein formation and degradation, being primarily a decrease in muscle protein synthesis.
- Muscle RNA synthesis is decreased up to 50%.
- Ubiquitin-proteasome pathway of intracellular protein degradation accounts for majority of nonlysosomal protein degradation and is a complex multicomponent system that selectively targets proteins for destruction.
- Pathway uses ubiquitin, a protein that covalently binds to targeted proteins and acts as a destruction signal for the proteins.
- Ubiquitin-tagged proteins degraded by a large protease complex, the 26S proteasome.
- Responsible for liberation of amino acids via cleavage of proteins such as myosin.
- Ubiquitin ligases MuRF-1 and MAFbx increase more significantly.
- Inflammatory process may be present during atrophy.
- mRNA upregulation of TNF-α, IL-1, IL-6, and leukocyte marker CD45 occurs.
- TNF-α initiates transcription of NF-κB.
- Some NF-κB mediators (p50 and Bcl-3) increase and are associated with disuse muscle atrophy, suggesting an alternate, noninflammatory NF-κB.
- Inhibition of NF-κB alone does not prevent muscle atrophy.
- NF-κB activates MuRF-1.
- Some protein kinases activated by TNF-α or ROS, such as hydrogen peroxide, may be associated with oxidative stress and the generation of ROS, which also activate NF-κB and FOXO.
- The IGF-1/PI3K/Akt pathway is an important intracellular regulator of muscle hypertrophy.
- IGF-1 stimulates activation of PI3K.
- P13K creates a cell membrane binding site for a serine/threonine kinase.
- Akt (also referred to as *protein kinase B*) activates rapamycin (mTOR) kinase.
- mTOR increases protein synthesis by activating p70S6 kinase and eukaryotic translation factor 4E binding protein 1 (4E-BP-1), regulatory proteins involved in protein synthesis.
- IGF-1/PI3K/Akt pathway suppresses expression of ubiquitin ligases that are important in signaling muscle atrophy.
- The IGF-1/PI3K/Akt pathway also activates FOXO proteins, transcription factors that regulate target genes for metabolism, apoptosis, and cell cycles.
- Akt phosphorylates FOXO proteins.
- This prevents translocation to the cell nucleus, inhibiting transcription functions.
- Blockade of FOXO by the IGF-1/PI3K/Akt pathway is important to prevent muscle atrophy.

Ca-ATPase, Calcium adenosine triphosphatase; *FOXO,* forkhead box O; *IGF,* insulinlike growth factor; *IL,* interleukin; *MAFbx,* muscle atrophy F-box; *mTOR,* mammalian target of rapamycin; *MuRF-1,* muscle ring finger 1; *NF-κB,* nuclear factor kappa B; *PI3K,* phosphatidyl inositol-3-OH kinase; *ROS,* reactive oxygen species; *TNF,* tumor necrosis factor.

stretched position, the fibers become lengthened because of the addition of sarcomeres.[136,137] Connective tissue is increased in atrophic muscles, which may result in increased muscle stiffness.

Models of reduced limb usage include hindlimb immobilization (HI), hindlimb suspension (HS), denervation, and spaceflight. There is greater muscle atrophy with HS than HI because HI muscles are able to contract against the immobilizing material. In contrast, HS muscles contract, but there is no load to contract against.

The change in muscle fiber size and fiber percentage was studied in three heads of the dog quadriceps following 10 weeks of rigid immobilization with an ESF.[135] Muscle fiber atrophy was greatest in the vastus medialis and least in the rectus femoris. The atrophic response for type I fibers was, in order from most to least atrophied, vastus medialis, vastus lateralis, and rectus femoris; for type II fibers, atrophy of the vastus medialis was equal to vastus lateralis, and both atrophied more than the rectus femoris. In fact, vastus medialis type I and II muscle fiber areas were only approximately one third of normal following immobilization. Tetanic torque also declined by approximately 60%, and muscle fiber diameter correlated strongly with torque.[139] The rectus femoris acts as a knee extensor and hip flexor and crosses two joints, which makes it less susceptible to atrophic changes because the hip joint was not immobilized in this model. The changes in the vastus medialis suggest that this muscle performs a greater postural function than the vastus lateralis. A similar study with 10 weeks of immobilization indicated that there was a significant decrease in both type I and type II fiber area, and muscle fiber areas recovered to only approximately 70% of control values following 4 weeks of remobilization.[140] Tetanic torque improves

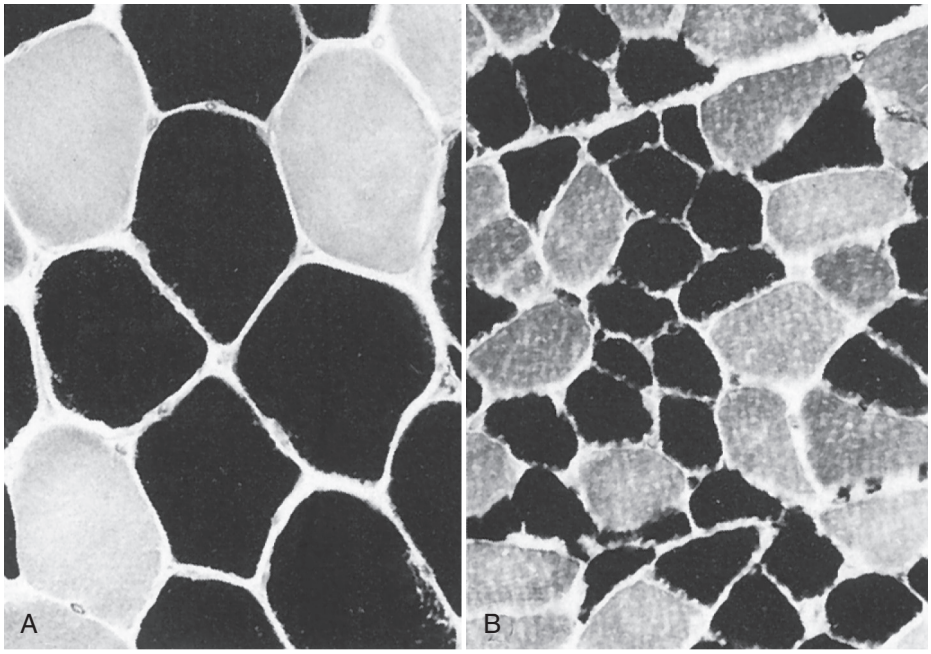


Figure 7-4 Photomicrographs of biceps femoris muscle. **A,** Normal muscle. **B,** Same muscle following 60 days of disuse. There is general atrophy of both types I (light stained) and II (dark stained) muscle fibers. (Figure 9-9 from Carlton WW, McGavin MD: *Thompson's special veterinary pathology,* St Louis, 1995, Mosby.)

during remobilization, but remains below preimmobilized levels.[139]

In general the muscles most vulnerable to immobilization-induced atrophy are those that cross a single joint and have a large proportion of type I muscle fibers, such as the vastus medialis and vastus intermedius.[135] The next most susceptible muscles are antigravity muscles that cross multiple joints and have primarily type I muscle fibers. Examples of these muscles are the gastrocnemius and rectus femoris muscles. Muscles least susceptible to atrophy following immobilization are those that are intermittently activated and have predominantly type II muscle fibers, including the cranial tibial, long digital extensor, and biceps femoris muscles.

Biochemical changes also occur in muscles with immobilization. The effect of immobilization on muscle carbohydrate metabolism was investigated in dogs.[141] Total carbohydrate and glycogen content of skeletal muscle fell during immobilization. The glycogen-degrading enzyme phosphorylase was activated 1 week after immobilization. Thereafter, enzyme activity decreased and remained significantly lower. In contrast, muscle glucose and lactate concentrations were unchanged. Oxidative and glycolytic enzyme activities may be reduced and are associated with reduced capacity for energy production. Immobilization may also result in reduced glutathione content.[142] Reduced muscle activity may reduce the responsiveness of muscle to insulin, which affects the ability of muscle to take up glucose. There is also increased total muscle calcium concentration, decreased sarcoplasmic reticulum calcium uptake, and reduced Ca-ATPase activity following immobilization.

The blood supply to the myotendinous junction is also affected by immobilization. In one study of rats, the vascular density of the myotendinous junction was decreased by 30% after 3 weeks of immobilization.[143] Vascular density returned to normal after 8 weeks of cage remobilization. Progressively increasing running resulted in greater vascular density. The effects of immobilization on peak oxygen uptake and effective oxygen diffusive conductance in skeletal muscle were studied in dogs.[144] Dogs were cage-confined for 8 weeks, with immobilization of a hindlimb for the last 3 weeks. Gastrocnemius muscles were electrically stimulated to elicit peak oxygen uptake at three levels of arterial oxygenation. Immobilization was sufficient to reduce muscle mass by 31% and citrate synthase activity by 68%; however, it had no effect on peak oxygen uptake or oxygen diffusive conductance. Immobilization also apparently does not affect muscle oxygen diffusing capacity.[127]

Other species have similar changes in muscle with immobilization. Immobilization of a hindlimb of sheep for 9 weeks resulted in an 8% reduction in thigh circumference and a slight decrease in the area of type I fibers.[145] In addition, Na^+-K^+ pump concentration and citrate synthase activity were reduced, which may contribute to fatigue during activity. A modified rat tail suspension model, which allows the rear limbs to remain mobile without any weight bearing, was used to determine the atrophic response of muscle with unloading.[146] Maximal muscle atrophy occurred within 14 to 30 days. The gastrocnemius was less

severely affected by suspension than by other immobilization techniques, suggesting that muscle atrophy in the suspension model is different from immobilization atrophy. One significant response was the occurrence of decreased blood flow, which may indicate that hypodynamic nonimmobilized muscle has altered functional demands. Similar changes occur in humans. Patients immobilized with a cast after tibial fractures or ligamentous injuries had decreased limb circumference immediately after removal of the cast.[147] These differences persisted even after 81 days of remobilization.

Prolonged bed rest in people causes changes in muscle mass.[148] In humans, 4 to 5 weeks of bed rest results in a 10% loss of muscle mass in the lower limbs and 4 months of bed rest results in 20-30% loss of muscle mass. In people, 10 minutes of standing per day during bed rest attenuates muscle atrophy by 25%. If some resistance exercise is added, muscle atrophy is attenuated by up to 50%. In addition, there is an apparent shift in muscle fiber type with bed rest. After 35 days of bed rest in one study, there was a slow to fast shift in muscle phenotype.[149] Consistently, fiber type distribution was shifted toward type II and IIAX fibers. Muscle fiber atrophy was variable among fiber types. Myosin concentration was significantly lower in type I and type IIA muscle fibers after bed rest than before bed rest, whereas the myosin/actin ratio did not vary, suggesting a disproportionate loss of myosin compared with fiber cross-sectional area and a proportional loss of myosin and actin.

Spaceflight may also result in significant loss of muscle mass because of the loss of weight bearing and loading.[148] Muscle volumes of the calf, thigh, and lower back were determined before and after an 8-day space shuttle mission. The soleus-gastrocnemius (−6.3%), anterior calf (−3.9%), hamstrings (−8.3%), quadriceps (−6%), and intrinsic back (−10.3%) muscles were decreased 24 hours after landing compared with baseline. After 2 weeks, the hamstrings and intrinsic lower back muscles were still below baseline.

Reflex inhibition of muscles, a situation in which sensory stimuli impede the voluntary activation of muscle, may occur with joint injury and result in muscle atrophy, and loss of strength and activation of the muscle. Reflex inhibition may be measured directly by electromyography, or the sequelae of reflex inhibition may be measured, such as thigh circumference measurement and muscle biopsy.[150] The most frequently cited causes of muscle reflex inhibition in joint injury are pain, joint effusion, and joint immobilization. Finally, there may be selectivity of affected muscles and muscle fibers with reflex inhibition following joint injury. In light of these findings, several suggestions have been offered for preventing reflex inhibition that can be applied to rehabilitate the most affected muscle group, the quadriceps femoris. These include cryotherapy, transcutaneous electrical nerve stimulation, electromyostimulation, traditional exercise training, joint mobilization, rest, and proper positioning of the limb in rest and exercise.

Mechanisms of Muscle Atrophy

It is believed that muscle atrophy occurs as a result of an imbalance of protein formation and degradation, and although recent research has begun to unravel how this imbalance occurs, the exact mechanisms are unknown. Most studies suggest that decreased muscle protein synthesis is the prime cause of muscle atrophy, although there is also transient muscle protein degradation in early muscle atrophy. In people, muscle protein synthesis decreases in the first 10 days of immobilization, and then the rate of muscle synthesis appears to stabilize. RNA synthesis also decreases with immobilization.[138] Quadriceps muscle protein turnover was assessed in men immediately after the end of 37 days of leg immobilization in a plaster cast after tibial fracture.[151] Quadriceps muscle protein synthetic rate was 0.046% per hour in the contralateral leg, but was only 0.034% per hour in the immobilized leg. Muscle RNA activity in the immobilized leg was 50% that of the nonimmobilized leg. Immobilization was associated with significant atrophy of type I muscle fibers, but no significant change occurred in type II fiber diameter. Mean quadriceps fiber volume was smaller in the injured leg by 10.6% and the difference in muscle thigh volume was 8.3%. Some studies have shown little evidence of increases in mRNA for catabolic enzymes or increases in enzyme activity during this period,[152] although others suggest that such increases occur very quickly after immobilization or spinal cord transection.[153] However, it appears most likely that the process of muscle atrophy during immobilization is primarily a result of decreased protein synthesis, with continued normal muscle breakdown.[154]

Recently, attention has focused on the role of the ubiquitin-proteasome pathway of intracellular protein degradation. It is believed that this system accounts for the majority of nonlysosomal protein degradation and is a complex multicomponent system that selectively targets proteins for destruction.[155] The pathway uses ubiquitin, a protein that covalently binds to targeted proteins and acts as a destruction signal for the proteins. Three distinct conjugating enzymes are involved in this process: an E1 ubiquitin-activating enzyme, an E2 ubiquitin-conjugating enzyme, and an E3 ubiquitin-ligating enzyme.[156] The ubiquitin-tagged proteins are then degraded by a large protease complex, the 26S proteasome. This pathway is responsible for the actual liberation of amino acids via cleavage of long-lived proteins, such as myosin. Two muscle-specific ubiquitin ligases, muscle ring finger 1 (MuRF-1) and muscle atrophy F-box (MAFbx), increase more significantly than other components of this proteolytic pathway in mice.[114] A study of the molecular aspects of skeletal muscle atrophy demonstrated the importance of these ligases in a mouse model.[157] In this model, significant

skeletal muscle atrophy occurred, with the myofiber size of the cranial tibial muscle reduced by 36% compared with the untreated contralateral limb within a few days. MAFbx and MuRF-1 mRNAs were significantly upregulated by two- and sixfold, respectively. Interestingly, an early inflammatory process was present during atrophy in this study, characterized by the mRNA upregulation of TNFα, interleukin (IL) 1, and IL-6 (2-, 2.4-, and 3.4-fold, respectively), along with upregulation of the leukocyte marker CD45 (6.1-fold). Muscle rapidly recovered with remobilization, with increased levels of uncoupling protein-3 and peroxisome proliferator-activated receptor gamma coactivator-1 alpha mRNA, important components of oxidative muscle metabolism. Studies of rats in a hindlimb suspension model have also shown increased function of the ubiquitin-proteasome pathway, with increased ubiquitin mRNA levels, conjugating enzymes, and proteasome subunits, indicating the potential importance of this pathway of muscle degradation. Manipulation of this system of muscle degradation may alter the process of muscle atrophy and preserve muscle mass during periods of immobilization. However, knock-out of the MuRF1and MAFbx genes does not completely prevent atrophy, suggesting that other ligases are involved.[114]

The IGF/phosphatidyl-inositol-3-OH kinase (PI3K)/Akt pathway is an important intracellular regulator of muscle hypertrophy. IGF-1 stimulates the activation of PI3K, which then creates a cell membrane binding site for a serine/threonine kinase, Akt (also referred to as *protein kinase B*). Akt activates mammalian target of rapamycin (mTOR) kinase, which increases protein synthesis by activation of p70S6 kinase and eukaryotic translation factor 4E binding protein 1 (4E-BP-1), regulatory proteins involved in protein synthesis. The IGF-1/PI3K/Akt pathway also suppresses the expression of ubiquitin ligases, which are important in signaling muscle atrophy.[114] The IGF-1/PI3K/Akt pathway activation also activates forkhead box O (FOXO) proteins, transcription factors that regulate target genes for metabolism, apoptosis, and cell cycles. Akt phosphorylates FOXO proteins, which prevents translocation to the cell nucleus, effectively inhibiting their transcription functions. Thus blockade of FOXO by the IGF-1/PI3K/Akt pathway is important in helping to prevent muscle atrophy and may be an approach to help prevent muscle atrophy.

Chronic inflammation may also induce protein loss and muscle atrophy through the action of inflammatory mediators on the nuclear factor kappa B (NF-κB) pathway.[114] TNF-α is one inflammatory mediator that initiates transcription of NF-κB. Although disuse atrophy does not result in increased TNF-α, certain NF-κB mediators (p50 and Bcl-3) increase and are associated with disuse muscle atrophy, suggesting an alternate, noninflammatory NF-κB pathway. Furthermore, several genes associated with p50 and Bcl-3 and muscle atrophy have been identified in a mouse model of disuse atrophy.[158] Although NF-κB activity inhibition attenuates cancer cachexia, its inhibition is not sufficient to prevent disuse muscle atrophy. Another potential mechanism of signaling muscle atrophy is the activation of MuRF-1 by NF-κB. Mitogen-activated protein kinases may also be involved in muscle atrophy, especially when activated by TNF-α or reactive oxygen species (ROS), such as hydrogen peroxide. Immobilization of limbs is associated with oxidative stress and the generation of ROS, which may also activate the NF-κB and FOXO systems.[114] Inhibition of both NF-κB and FOXO systems can reduce muscle atrophy as a result of immobilization. In one study of rat limb immobilization, control treatment resulted in a 47% decrease in muscle fiber cross-sectional area.[159] Fibers from immobilized muscles that had inhibition of FOXO induction had a 76% inhibition of atrophy. Inhibition of both FOXO and NF-κB had even greater inhibition (up to 95%) of muscle atrophy. These findings suggest that the combined effects of inhibiting immobilization-induced NF-κB and FOXO transcriptional activity has an additive effect on preventing immobilization-induced atrophy. The mechanisms of muscle atrophy may be more complex in people than in small laboratory animals.[160] For example, anabolic resistance may partially explain, in part, reduced muscle protein synthesis of atrophied muscle in the postprandial state, even with high levels of amino acid present. This anabolic resistance during immobilization could have implications on nutritional interventions.

Myostatin may also play a role in disuse atrophy of muscle because unloading in a rat hindlimb unloading model resulted in an upregulation of myostatin and activin IIB, the myostatin receptor.[161,162] However, the role of myostatin in muscle atrophy remains unclear because the soleus muscle, which contains no myostatin, undergoes the greatest atrophy with unloading. It may be that myostatin inhibits hypertrophy rather than induces atrophy.[114] Another study evaluated antibody-directed myostatin inhibition in mice during unilateral hindlimb casting.[163] Although hindlimb casting reduced muscle mass, fiber size, and function of soleus and extensor digitorum longus muscles in control animals, antibodies directed against myostatin attenuated the loss of muscle mass, fiber size, and function with greater effectiveness when given early in immobilization.

Muscle use and disuse apparently govern the number of acetylcholine receptors that are regulated through a feedback mechanism. Immobilization of skeletal muscle results in disuse atrophy and resistance to nondepolarizing muscle relaxants. Dogs undergoing cast immobilization of a hindlimb for 3 weeks had resistance to metocurine, a muscle relaxant, by the fourth day of casting.[164] This resistance persisted for 2 weeks after cast removal, but was normal 6 weeks after cast removal. These responses suggest that there may be upregulation of muscle acetylcholine receptors as a result of immobilization.[165] These changes

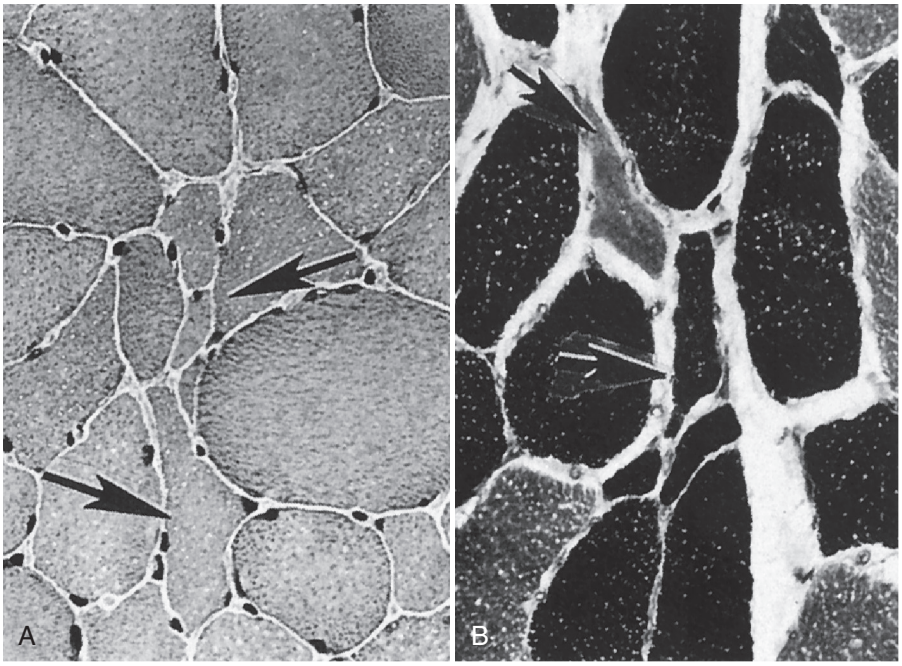


Figure 7-5 Neurogenic muscle atrophy. **A,** Early denervation atrophy as indicated by small angular fibers (*arrows*). **B,** Atrophic angular fibers of both type I (light) and type II (dark) muscle fibers. (From Carlton WW, McGavin MD: *Thompson's special veterinary pathology,* St Louis, 1995, Mosby.)

may also occur in patients with muscle weakness who are in the intensive care unit. A canine intensive care model that involved 3 weeks of anesthesia with pentobarbital and positive-pressure ventilation indicated that the absence of muscle tone and reflex responsiveness for 3 weeks was also associated with exaggerated resistance to neuromuscular blockade.[166]

Neurogenic Muscle Atrophy

Muscles undergo denervation atrophy with damage to the spinal motoneurons or to the motor nerves in the ventral roots.[167] It has been well documented that there is an early rapid loss, followed by stabilization, of muscle mass with neurogenic muscle atrophy (Figure 7-5).[167,168] On the other hand, disuse atrophy occurs with damage to the central nervous system. Neural activity is generally reduced with spinal cord lesions, but varies with the type of lesion and the level of spasticity.

Structural and biochemical changes occur with neurogenic atrophy, including changes in glycolytic and oxidative metabolic enzymes. Functional recovery following nerve injury and repair is directly related to the degree of muscle atrophy that takes place during the period of nerve regeneration and the ability of surviving motoneurons to sprout and reinnervate the denervated muscle fibers.[167,169] For injuries with 85% of the motor supply to a partially denervated muscle intact, all denervated muscle fibers are reinnervated and the partially denervated muscle recovers completely. Conversely, lesions with injury to 85% of the motoneurons result in inadequate sprouting and incomplete recovery. Motoneurons may increase the number of muscle fibers they normally supply by fivefold with sprouting. However, the distance over which sprouting occurs is limited and with extensive denervation, nerve sprouts may not grow far enough to reinnervate muscle. The extent of muscle atrophy is related to a number of factors, including the distance through which the nerve must regenerate; the age of the patient; and the type of nerve injury and other associated tendon, soft tissue, and bony damage. In animals with spinal cord injuries, the capacity of paralyzed muscles to sustain contractions is reduced, therefore reducing muscle endurance. Adequate nursing care of paraplegic or tetraplegic animals is necessary to prevent muscle atrophy from disuse.[170] Secondary conditions, such as disuse osteoporosis of bones and contracture of joints, must also be considered in dealing with patients having neurogenic muscle atrophy.

Computed tomography was used to evaluate neurogenic muscle atrophy in dogs with induced sciatic nerve injury.[171] The cross-sectional area of muscles decreased 1 to 2 weeks after denervation, and the differences were significant after 3 weeks. Additional decreases were minimal after 28 days, after which time there was significant infiltration of adipose tissue into the muscle. The cross-sectional areas of the biceps femoris, semimembranosus, and semitendinosus muscles decreased by approximately 30% by 60 days, with most of the changes occurring in the first 2 to 3 weeks. In another study of denervated latissimus dorsi muscle, there

was a 20% loss of lean muscle mass after 6 six weeks, 45% loss at 6 months, and 55% loss at 9 months.[168] Histologic examination found atrophy of individual muscle fibers. With denervation atrophy, there is preferential atrophy of type II muscle fibers which is the opposite of what is seen with immobilization of innervated muscles.[138]

Denervated dog gastrocnemius muscle has a progressive decrease in total protein content, alanine aminotransferase, aspartate aminotransferase, and glutamate dehydrogenase activity, and elevation in free amino acid, ammonia, urea, glutamine contents, and AMP deaminase activity levels following neurectomy.[172] Total protein content decreased 50% by 4 weeks after muscle denervation. Catabolism of muscle protein may result in increased metabolites, including ammonia, urea, and glutamine.

Serum carbohydrate metabolism was evaluated in denervation atrophied and denervation electrical-stimulated dogs.[173] Neuromuscular electrical stimulation was applied to the gastrocnemius muscles of the latter group for 30 min/d for 15 days, using biphasic pulses of 10 V, 100 ms duration and 2 Hz. Denervation atrophy may result in hyperglycemia possibly resulting from the lack of uptake by the muscle. Electrical stimulation applied to denervated muscle may reverse the hyperglycemia, indicating that muscular work may be important in modulating serum carbohydrate metabolism.

Continuous electrical stimulation of denervated muscles during the period of nerve regeneration appears to help maintain the integrity of the muscle fibers and their potential functional capacity.[169] In addition, electrical stimulation may be applied for extended periods with little evidence of discomfort. This modality may be useful in patients with the potential for partial or complete recovery from nerve injury.

Changes in Muscle with Aging

Changes in muscle as people and animals age include decreased muscle mass, fiber size, and fiber number. These changes, along with increased intramuscular fat and connective tissue, result in reduced ability to generate muscle force.[174,175]

Muscle satellite cells also decrease by the age of 70 in people.[176] In addition, the loss of muscle mass and muscle function may result from diminished regenerative capacity of muscle, increased preprogrammed cell death activity (apoptosis), anabolic resistance, protein degradation, and autophagocytosis occurring in skeletal muscle.[177] Although muscle mass declines with aging, the loss of muscle strength occurs at a greater rate than the loss of muscle mass in older people, suggesting that atrophy alone is not the cause of strength loss.

Neuromuscular changes may contribute to the loss of muscle strength, including reduced muscle activation and contractile rate.[178] In fact, there is a loss of spinal motor neurons with aging, which is paralleled by a reduction in muscle fiber number and size, resulting in reduced muscle function and strength.[179] Aging results in reduced maximal motoneuron firing frequency and muscle activation. There is some thought that muscle atrophy with aging results, in part, from neuropathic changes that lead to motoneuron death, which leads to a decrease in the number of muscle fibers. In addition, motor unit recruitment and firing frequency are reduced in older adults.[180,181]

Interestingly, unloading skeletal muscle in older animals leads to less disuse muscle atrophy compared with that of young animals, but skeletal muscle regrowth is slowed and less complete in older animals as compared with young during remobilization.[182] Similar findings occur in humans.[183] One reason for altered recovery of muscle mass in young versus older animals after limb immobilization may be related to differences in gene expression. In one study, HI of rats resulted in a 27-37% loss in soleus muscle mass. The atrophied soleus muscle of young but not old rats recovered to the same mass as before treatment.[184] The mRNA levels of muscle growth potentiating factors were differentially expressed between young and older animals, and may modulate muscle growth that could explain the failure of muscles of older animals to recover lost muscle mass. In addition to changes in muscle and the nervous system with aging, tendon changes also contribute to muscle weakness with aging. The interplay between these muscle and tendon changes has an effect on the length-force and force-velocity relationships of the muscle and is likely to affect the ROM, rate of force development, maximum force development, and speed of movement of the older individual.[185]

Recently, it has been shown that resistance training may attenuate the decline in muscle performance with aging. In people, strength training programs of 3 months' duration may increase muscle cross-sectional area by 5-17%, which is similar to changes in young adults.[186,187]

Exercise can induce the expansion of satellite cells in skeletal muscle of older people, as well as younger people.[176] In addition to muscle hypertrophy as a result of strength training in older people, there also appear to be significant changes in neuromuscular function, leading to improved function.[179]

Muscle Changes with Orthopedic Conditions

Hip Conditions

Hip conditions are common in dogs and may affect muscle mass. In some conditions, such as hip dysplasia, lameness and reluctance to exercise may result in pelvic limb muscle atrophy with compensatory shoulder muscle hypertrophy.[188] Excision of the femoral head and neck is sometimes performed as a treatment for severe hip dysplasia or fractures of the acetabulum. Although fewer dogs have obvious lameness or pain in the operated hip after recovery from surgery, muscle atrophy is noted in approximately 50% of dogs following femoral head and neck excision, even after

8 years.[189] Moderate or marked muscle atrophy is most common in larger breeds of dogs. Difficulty in jumping and in climbing stairs may be noted postoperatively in larger breeds of dog and may be related to the loss of power needed for jumping and hip extension following femoral head and neck excision. Legg-Calve-Perthes disease, or aseptic necrosis of the femoral head, is a condition affecting small breeds of dogs that is also treated with a femoral head and neck excision. In a clinical study of dogs with avascular necrosis of the femoral head, muscle atrophy was present in 25 of 35 dogs at the time of diagnosis as a result of pain and lameness.[190]

Cranial Cruciate Ligament Injury

Changes in muscle mass are common in dogs with CCLR and following surgical treatment of the injury. In an experimental study, dogs had a CCL transected, followed by immediate stabilization with an extracapsular procedure.[191] Results of this study revealed muscle atrophy of the surgical leg by 2 weeks, with muscle mass beginning to return between 4 and 8 weeks, but significant atrophy was still present 8 weeks after surgery. In addition, the contralateral nonsurgical limb underwent hypertrophy, possibly because of the increased loading on that limb during recovery.

A study of dogs with naturally occurring CCL deficiency evaluated patients before surgical treatment and 1.5, 7, and 13 months after surgery.[192] The degree of quadriceps muscle atrophy present prior to surgery correlated significantly with the degree of cartilage fibrillation, indicating a relationship with the severity of the condition. Although there was slightly greater muscle atrophy 6 weeks after surgery, muscle mass improved 7 and 13 months after surgery, but significant residual muscle atrophy remained in many dogs even after 1 year. A measure of quadriceps atrophy may be a useful tool for assessing long-term outcome.

A long-term outcome of thigh circumference of 29 clinical patients with CCLR and stabilization with tibial plateau leveling osteotomy (TPLO) was recently reported.[193] Standing mid-thigh circumference measurements were made in triplicate. Clinical lameness was graded blindly. Although the mean thigh circumference for the operated limb was 98.5% of the contralateral limb, a significant difference existed between the limbs. Time after surgery had no apparent effect on outcome. Four dogs (14%) exhibited some lameness in the TLPO limb during evaluation (one dog was 1 to 2 years postoperative and three dogs were 2 to 3 years postoperative). The source of the lameness was not identified, and there was no difference in thigh circumference between dogs with lameness and those without lameness.

Information regarding changes in specific muscles following CCLR and stifle stabilization surgery may be useful to target specific muscles for rehabilitation. Muscle atrophy was studied in a group of dogs undergoing surgical transection of a CCL and immediate stifle stabilization using a modified retinacular imbrication technique.[194] Thigh circumference and body composition measured by dual-energy x-ray absorptiometry (DEXA) were used to estimate limb muscle mass before surgery, and 2, 5, and 10 weeks after surgery. Actual mass of rear limb muscles was determined at 10 weeks. Muscle mass decreased with transection of the CCL, despite immediate stifle stabilization (Figure 7-6). The changes were evident by 2 weeks, and continued until 5 weeks, followed by a slight recovery at 10 weeks. The muscles most affected at 10 weeks were the quadriceps, biceps femoris, and semimembranosus, and these muscles weighed only 60% of the intact contralateral side (Figure 7-7). The muscles of the crus are similarly affected (Figure 7-8).

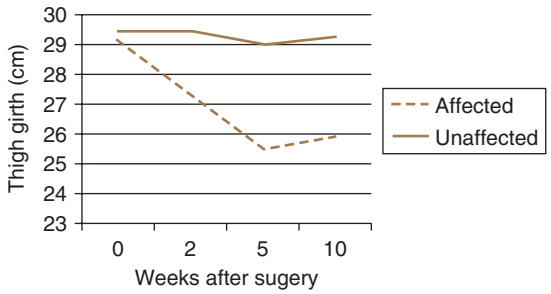


Figure 7-6 Rate of muscle atrophy following cranial cruciate ligament transection and immediate stifle stabilization with no postoperative rehabilitation.

Changes in thigh girth and DEXA lean tissue mass were similar over time and indicated significant muscle atrophy following cruciate transection and repair. The quadriceps and biceps femoris muscles are important in supporting weight during stance, and should be targeted during rehabilitation to improve muscle mass and strength. The time course of changes suggest that postoperative physical rehabilitation should continue for at least 5 weeks after surgery.

Another study evaluated morphometric characteristics of hindlimb muscles in Labrador retrievers with and without CCL deficiency and found that atrophy associated with CCL deficiency predominantly affected the quadriceps muscle.[195] The authors also suggested that dominance of the gastrocnemius muscle over active restraints to cranial tibial thrust might be associated with predisposition to CCL deficiency in Labrador retrievers. This dynamic imbalance between muscle groups of the rear limbs could serve as a basis for screening programs and preventive rehabilitation, especially considering that targeted training programs, such as plyometric training, may reduce the risk of injury 3.6 times in women.[196]

Muscle atrophy also occurs in people following surgery for torn ACLs. Thigh and calf girth measurements of involved and noninvolved extremities were determined prior to and following knee surgery for acute and chronic

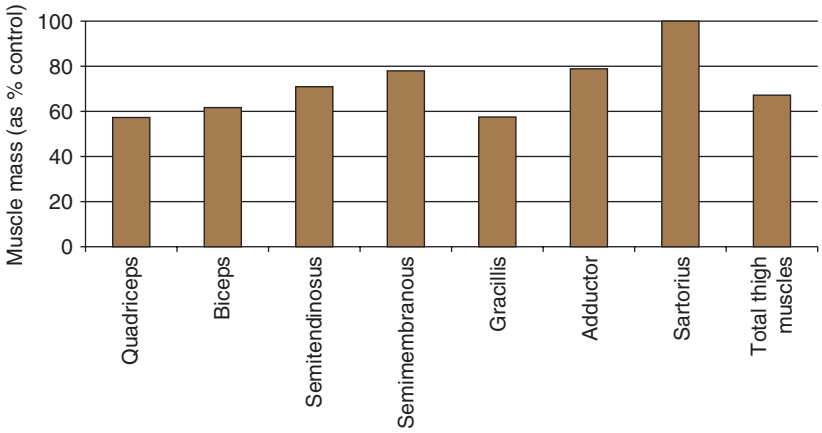


Figure 7-7 Mass of thigh muscle mass 10 weeks after cranial cruciate ligament transection and stifle stabilization with no rehabilitation. Values are percentage of the mass of the unaffected contralateral limb.

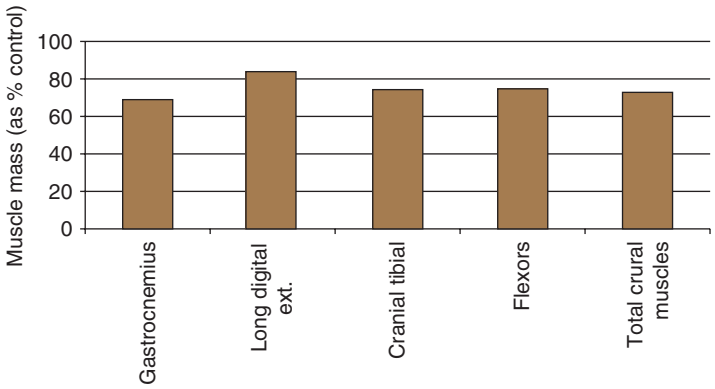


Figure 7-8 Mass of crus muscle mass 10 weeks after cranial cruciate ligament transection and stifle stabilization with no rehabilitation. Values are percentage of the mass of the unaffected contralateral limb.

knee injuries.[197] Although thigh and calf girth measurement differences existed between the involved and noninvolved extremities prior to and after surgery, the bulk of the girth measurement differences existed prior to surgery for both groups. The differences between involved and noninvolved limbs were in the 2-6% range, which were similar to differences observed in another study.[198] They concluded that altered gait and reflex inhibition contributed to muscle atrophy following injury and surgery. Muscular changes more than 5 years following ACL injury were evaluated to determine if there were differences between patients with ACL reconstruction and conservative treatment.[199] Thigh circumference was measured and the relationship of strength to electrical activity and muscle size was analyzed. Only minimal differences in thigh circumference were found between the uninjured and injured limb of both groups; however, isokinetic torque and electromyogram (EMG) values showed significant differences. These changes may be due to modified use of muscle fibers and altered joint receptor afferent signaling following injury.

These changes may result in chronic loss of quadriceps femoris strength in humans following ACL surgery despite aggressive rehabilitation. One study evaluated the correlation of thigh muscle size and strength with thigh circumference, muscle cross-sectional area by MRI, and isokinetic strength.[200] Patients were evaluated 48 months after surgery. There was a significant 1.8% decrease in thigh circumference, a 10% decrease in average quadriceps torque, and an 8.6% decrease in quadriceps cross-sectional area by MRI in the involved extremities compared with the uninvolved extremities. Persistent quadriceps weakness and decreased cross-sectional area 48 months postsurgery and rehabilitation suggest that some patients may not completely recover from surgery, despite aggressive therapy. Strength deficits may persist for long periods, even with less severe injuries, such as partial tears of the medial collateral ligament (MCL).[201] Muscle atrophy that occurs following cruciate injury and surgery may result in early muscle fatigue, which also affects EMG patterns of quadriceps and hamstring muscles.[202] As little as 10 minutes walking may affect the gait pattern, particularly the activity of the hamstring muscles. This suggests that endurance training, in addition to strength and stabilization training, should be emphasized during rehabilitation.

Various specific techniques can be used for strengthening of the hamstring and quadriceps muscles in people following surgery for ACL repair, with or without resistive equipment.[203] In addition, electrical stimulation may help prevent muscle wasting caused by immobilization. People are generally able to return to full activity and competitive

sports after 6 to 12 months. An intense rehabilitation program following TPLO surgery for stabilization of a CCLR may improve muscle mass significantly more than standard home care. In one study, dogs with cruciate-deficient limbs had significantly reduced thigh circumference before surgery as compared with values for the contralateral limb.[204] The rehabilitation group had significantly larger thigh circumference than the home-exercise group 6 weeks after TPLO, with no difference between the affected and nonaffected limbs.

Fractures

Muscle atrophy may occur following fractures, particularly those with severe comminution. A stable repair with anatomic reduction is vital to encourage early weight bearing, joint motion, and use of the limb. Distal femoral fractures treated with limb immobilization in extension for 3 to 7 weeks resulted in limb hyperextension, generalized muscle atrophy, abducted gait, and limited range of joint motion.[205] Lesions in muscle biopsies included fiber size variability, increased fibrosis, and focal necrosis. Histochemical and morphometric studies showed significant type I fiber atrophy in the vastus lateralis muscle, with a reduction of the number of type I fibers. Atrophic changes in the gastrocnemius and biceps femoris muscles were not significant. The atrophy seen in the vastus lateralis muscle may be a result of immobilizing the limb in extension, which results in shortening of this muscle group. Immobilization of a muscle in a shortened position results in preferential atrophy caused by a reduced number of sarcomeres in series and reduced protein synthesis. In contrast, passive stretch of muscles in a lengthened state promotes muscle growth and may explain the relative sparing to the biceps femoris and gastrocnemius muscles.

A follow-up experimental study evaluated the effect of splinting in extension for 2 weeks following trauma to the distal portion of the quadriceps femoris muscle in dogs.[206] Flexion of the stifle joint was limited after splinting. A reversible type I fiber atrophy of the vastus lateralis, biceps femoris, and gastrocnemius muscles occurred. Early type II fiber atrophy was seen in a few muscles. Multifocal fiber necrosis was the only irreversible change seen. Relative fiber percentages did not change appreciably during splinting or recovery.[206]

Arthritis

The relationship between arthritis and muscle mass is unknown in dogs, but we suspect that muscle mass and strength decline with the progression of OA and the subsequent reduction in activity. Muscle weakness may predispose to OA and its clinical signs in people.[207] Reduced quadriceps strength is one of the earliest signs in people with knee OA, and the degree of strength loss has association with the severity of OA.[178] Muscle weakness may lead to functional deficits in joint stability and reduced physical activity and possibly contribute to further joint deterioration. The ratio of quadriceps to hamstrings muscle strength is important in people for knee stability and for protection from excessive stress.[208] People with knee OA have weak knee flexor and extensor strength, with relatively greater weakness in the quadriceps muscles. The mean quadriceps to hamstring strength ratio of 1.4 is less than ratios reported for young healthy adults of 2. The low ratio might be explained by reflex inhibition resulting from pain associated with disease, because the quadriceps muscle can be selectively inhibited by pain and effusion in the joint, leading to decreased production of muscle tension. In fact, joint enlargement had a negative correlation with the ratio of quadriceps to hamstring muscle strength in one study.[209]

The severity of the arthritis may also affect muscle mass. The strength and bulk of the quadriceps muscle of children with juvenile chronic arthritis were studied over a 2-year period.[210] Children with high OA severity scores had the least strength and muscle bulk. As the arthritis progressed and the severity score worsened, there was a corresponding reduction in muscle strength and bulk. The presence and intensity of local arthritis may be one important factor affecting muscle function in dogs with chronic arthritis.

Patients with knee OA had significantly weaker quadriceps strength, larger voluntary muscle activation deficits, and smaller lean muscle cross-sectional area compared with contralateral, nondiseased limbs.[178] Muscle activation deficits accounted for most of the variance in quadriceps strength of the OA limb, whereas lean muscle cross-sectional area explained the majority of variance in strength of the nondiseased limb. These findings suggest that small changes in muscle activation may have a profound effect on quadriceps strength in OA limbs.

Knee joint pain as a result of inflammation may also result in muscle atrophy. In one study, complete Freund's adjuvant was injected in rat knee joints to induce inflammation and pain.[211] One week after injection, muscle atrophy occurred only on the injected side. Weight of the rectus femoris and soleus muscles was decreased by 20%. The reduction in cross-sectional areas was similar for fast and slow muscle fibers. The authors suggested that early treatment to inhibit joint pain and inflammation may prevent muscle atrophy.

Response of Muscle to Remobilization

Mechanical loading is essential for muscle regeneration (Box 7-7). Therefore following immobilization to protect healing muscle, early remobilization is required to induce correct growth and orientation of regenerated myofibers.[212] Fortunately, most of the changes that occur in skeletal muscle during immobilization are reversible. One rule of thumb regarding humans states that a remobilization period of twice the duration of the immobilization period is necessary for a return of limb circumference to normal values.[147]

Box 7-7 Considerations for Remobilization of Muscle after Immobilization

- Mechanical loading is essential for muscle regeneration.
- Regaining muscle mass during remobilization takes at least twice as long as the period of immobilization.
- There is decreased potential for muscle regeneration in older animals.
- Loss of muscle strength also occurs during immobilization.
  - Hindlimb immobilization for 10 weeks caused a 60% decline in tetanic torque that improved with 5 weeks of remobilization.
- Training, especially more intense training, results in more rapid muscle mass recovery.
  - Treadmill training and swimming are useful.
    - An adaptation period may be necessary in remobilized muscle to be able to withstand moderate training and avoid damage during early exercise.
    - Decline treadmill training may be more effective for some muscle groups.
    - Treadmill running three and six times per week is more beneficial for muscle recovery compared with no running or running once per week.
    - Resistance exercise stimulates muscle protein synthesis and activation of muscle satellite cells.
      - Eccentric muscle contractions causes greater satellite cell activation and muscle mass gains compared with concentric muscle contractions.
    - Satellite cell activation involves an initial inflammatory response.
      - Possible that NSAIDs may reduce postexercise muscle protein synthesis, but NSAIDs also decrease discomfort and muscle soreness.
    - Training before immobilization and remobilization has a positive effect on muscle mechanical behavior compared to being sedentary.
- Rapamycin (mTOR)-S6 kinase turns on muscle protein synthesis, and myostatin is rapidly suppressed with postimmobilization training.

mTOR, Mammalian target of rapamycin; *NSAID,* nonsteroidal antiinflammatory drug.

There appears to be an age-dependent decline in muscle regeneration potential, so remobilization and loading of muscle may be especially important in older animals.

The effect of remobilization following splinting the stifle joint of young dogs with and without muscle injury was studied.[206] A rear limb was splinted in extension for 3 weeks and the biceps femoris, vastus lateralis, and gastrocnemius muscles were studied after splint removal and following 6 weeks of free activity. A reversible type I fiber atrophy occurred in most splinted muscles. Relative fiber percentages did not change appreciably during splinting or recovery. Beagle dogs undergoing HI by external fixation for 10 weeks had a 60% decline in tetanic torque, which improved somewhat during a 5-week remobilization period.[139] The diameters of type 1 and type 2 fibers measured from the vastus lateralis muscle followed the same trend.

Similar improvements occur with remobilization in humans and other animals. Changes in limb circumference and passive resistance of the human knee caused by immobilization were studied during remobilization.[147] Patients immobilized with a long leg cast after tibial fractures or ligamentous injuries were evaluated immediately after cast removal and after 18, 36, and 81 days of remobilization. Although improved, differences in midthigh circumference were still present after 81 days of remobilization. During remobilization, the increased resistance to flexion found immediately after removal of the cast resolved. This may indicate a rapid readaptation of muscle length (shortened because of immobilization in a shortened position) to almost normal values. After cast immobilization for 3 weeks, rat limbs were remobilized by free cage activity or treadmill running.[213] Typical changes of muscle atrophy occurred during the immobilization period. Remobilization, especially treadmill running, resulted in return of muscle toward normal, particularly the fiber size, capillary number, and fiber type distribution. Another study of the same model that compared low and high intensity treadmill training after immobilization found that high-intensity activity was more effective in restoring muscles to normal than was low-intensity exercise.[214] Sheep hindlimbs were immobilized in a plaster splint for 9 weeks, followed by remobilization for 9 weeks.[145] There was a slight decrease in type I fiber area at 9 weeks, and a slight increase at 18 weeks. The Na(+)-K+ pump density and citrate synthase activity were reduced by 39% and 30%, respectively, after 9 weeks of immobilization. During remobilization both increased to the same level as in the control animals.

Physical condition prior to immobilization and remobilization may have an effect on muscle. One study evaluated training versus being sedentary before immobilization, and then rehabilitation. Rats were divided into seven groups: control (C); trained (T); sedentary (S); trained and immobilized (TI); sedentary and immobilized (SI); trained, immobilized, and rehabilitated (TIR); sedentary, immobilized, and rehabilitated (SIR). Swimming was used for training whereas sedentary animals were kept in cages of reduced size. Cast immobilization on a pelvic limb was used for limb immobilization, and rehabilitation was performed in water. Training improved all mechanical properties of muscle when compared with sedentarism. Training before immobilization and rehabilitation had a positive effect on muscle mechanical behavior compared with sedentarism.[215] Other studies have reported similar findings, and have suggested that training after immobilization should be carefully undertaken to avoid damage to muscle.[216] Remobilized muscle may not be able to withstand moderate training exercise before an adaptation period has occurred to avoid repeated damage during early

exercise. Another study also evaluated the effects of swimming training prior to and after a period of limb immobilization in rats. Treatment groups included C, 6 weeks under baseline conditions; T, swimming training for 6 weeks; TI, swimming training for 6 weeks and then immobilized for 1 week; and TIR, swimming training for 6 weeks, immobilized for 1 week, and then remobilized with swimming for 2 weeks. Regenerative characteristics of the soleus muscle were more significant in the groups submitted to swimming.[217]

Land-based remobilization may also be important in muscle recovery. A study evaluated different remobilization protocols in muscles following HS of rats. Treatment groups included controls, suspended, suspended followed by free activity, suspended followed by training on a declined treadmill, and suspended followed by training on a flat treadmill. The soleus and anterior tibialis muscles were evaluated. The suspended soleus muscle showed a significant increase in the proportion of intermediate and hybrid fibers and a decrease in the number of type I fibers. Some of these changes were reversible after remobilization. The three remobilization programs led to the recovery of both the proportional number of fibers and their size. The anterior tibialis muscle had a significant increase in the number and size of type I fibers and a cell size reduction of type IIB fibers after suspension, which recovered after training on a declined treadmill and free movement. Especially regarding the soleus, the findings indicated that training on a declined treadmill induced greater tissue restructuring after suspension.[218]

The frequency of training may also be important regarding the recovery of muscle mass after immobilization. Following 2 weeks of limb immobilization of rats, 6 weeks of exercise was instituted under different conditions: free cage activity and free remobilization, one time/week treadmill running, three times/week running, or six times/week running. Immobilization significantly reduced the cross-sectional area of soleus and gastrocnemius type I and type II muscle fibers. Immobilization also increased the ratio of type II to total fiber numbers in the muscles and induced pathologic changes in muscle fibers. Some of these changes were not corrected with free remobilization. Training one, three, and six times per week resulted in recovery toward normal levels. However, running three and six times per week was more beneficial for recovery of muscle atrophy compared with no running or running once per week.[99]

Skeletal muscle regeneration is mediated by muscle stem cells, or satellite cells. Injury or stress results in activation of satellite cells, with migration to these sites and differentiation into multinucleated myofibers. Satellite cells are activated if muscle contractions are of sufficient intensity and duration. However, there are significant species differences in the magnitude of change and time of muscle protein synthesis patterns. Resistance exercise stimulates muscle protein synthesis, and the acute increase in muscle protein synthesis with exercise is greater and longer lasting than changes after eating.[219] Eccentric muscle contractions appear to cause greater satellite cell activation and muscle mass gains as compared with concentric muscle contractions.[160] Nerve activity is also required to support the growth of regenerated myofibers and the development into various muscle fiber types.

Satellite cell activation also involves an initial inflammatory response.[212] Macrophages are involved in phagocytosis of necrotic debris, and they promote myogenic differentiation by activating satellite cells and causing differentiation. During eccentric exercise, sarcomere structure is damaged and inflammatory cytokines are produced, especially IL-6.[220] In addition, the NF-κB inflammatory signaling cascade is activated during early rehabilitation following immobilization.[221] Because inflammation and perhaps prostaglandins may stimulate satellite cell proliferation, it is possible that NSAIDs may reduce postexercise muscle protein synthesis.[160] However, NSAIDs also decrease the discomfort and inflammation of muscle soreness.

Myogenic regulatory factors are a group of transcription factors that induce the transcription of skeletal muscle proteins and coordinate satellite cell differentiation.[160] Although further research on the mechanisms of signaling muscle protein synthesis is needed, it is known that the rapamycin (mTOR)-S6 kinase pathway is involved with turning on muscle protein synthesis. During rehabilitation from immobilization-induced atrophy in humans, myostatin is rapidly suppressed and remains at a relatively low level throughout rehabilitation and muscle recovery, suggesting that myostatin also plays a key role in the regenerative process[160] (Box 7-7).

Adaptations of Muscle to Increased Mechanical Stress

Mechanical stress may be induced by endurance or strength training. Endurance training consists of repetitive, low-intensity contractions that result in changes in oxidative metabolism. Strength training is characterized by a limited number of contractions that occur against a high (often maximal) mechanical load.

Endurance Training

Endurance training does not affect muscle fiber number or cross-sectional area, but it alters metabolic responses. There is a twofold increase in oxidative capacity of all muscle fiber types with endurance training. There is an increase in muscle mitochondrial density, oxidative enzyme activity, muscle glycogen, and intracellular lipids.[146] Fatty acid use increases while reliance on carbohydrate metabolism is reduced. Muscles become more fatigue-resistant with training. Endurance training also elevates the antioxidant and detoxicant status of muscle.[142] For example, total

glutathione, glutathione peroxidase, glutathione reductase, and gamma-glutamyl transpeptidase increased in the leg muscles of beagle dogs treadmill trained for 40 km/day at 5.5-6.8 km/h, 15% upgrade, 5 days/wk for 55 weeks.

Training also results in augmentation of muscle capillarity by new capillary formation, which may increase the available surface area for oxygen diffusion and decrease the distance over which oxygen must diffuse to reach cells.[144] Training also appears to improve the functional blood-tissue gas exchange properties of muscle. The effects of exercise training on peak oxygen uptake and effective oxygen diffusive conductance in skeletal muscle were investigated in dogs exercised on a treadmill 1 h/day, 5 d/wk for 8 weeks.[144] Peak oxygen uptake by gastrocnemius muscles was 38%, 33%, and 19% greater and oxygen diffusive conductance was 71%, 75%, and 68% greater during normal levels of oxygen, moderate hypoxia, and severe hypoxia, respectively, in exercised dogs as compared with controls.

Endurance training can induce changes in muscle fiber types. The alterations initiated by long-distance running on muscle fiber type distribution was studied in young dogs run on a treadmill for 55 weeks, 5 days a week.[222] The daily running distance was gradually increased to 40 km and maintained at that level for the final 15 weeks. In the triceps brachii muscle, there was a shift from type II to type I fibers, which was also observed in the thoracic and cervical spinal muscles. In addition, there was also a shift toward a higher oxidative capacity of type II fibers. However, in the lumbar muscles, the percentage of type II muscle fibers increased significantly in the running group. Training can induce changes in fiber type composition not only in limb muscles but also in the stabilizing spinal muscles.

Although the ubiquitin-proteasome pathway has been implicated in the pathophysiologic findings of muscle atrophy, this system is also apparently involved in the response to training and conditioning. It is possible that increased proteolysis of muscle is required to repair exercise-induced damaged protein, or the general turnover of muscle proteins is increased. A field study of 44 English pointers and Labrador retrievers used for bird hunting and retrieving was conducted to examine changes in the ubiquitin-proteasome pathway. Muscle biopsy samples were collected from all dogs preseason and at peak season. There was pronounced up-regulation of ubiquitinated conjugates and the p31 subunit during the peak hunting period compared with the preseason period. The catalytic core of the proteasome (b–subunits) showed no apparent up-regulation with training, however. Increased training also resulted in increased muscle glycogen levels and citrate synthase. It appears that up-regulation of specific components of the ubiquitin-proteasome pathway was an indication that it plays a role in skeletal muscle turnover during training and conditioning.[223]

Although endurance training brings about changes in muscle metabolic properties, muscle mass may not be affected.[224] Young beagle dogs trained on a treadmill for 1 year, gradually increasing running to 40 km/day at a speed of 5.5-6.8 km/hour with a 15-degree inclination during the last 15 weeks, while control dogs were kept in their cages. There was no difference in mass of cranial tibial and semitendinosus muscles between trained and control dogs.

Strength Training

Strength training uses brief, maximal contractions and requires a relatively high rate of anaerobic energy production. In addition to increased enzyme activity for glycolysis and glycogenolysis, there is an increase in muscle strength and size. Muscle fiber cross-sectional area is directly related to the tension the muscle is capable of exerting.[225] Strength training by overloading muscle increases the cross-sectional area of all fiber types, especially the fast twitch fibers. New myofibrils are synthesized, and there may also be hyperplasia of muscle fibers. There is also improved synchronization of motor units.[146] This may help explain why the changes in muscle cross-sectional area are smaller than the changes in maximal voluntary force production.[226] Resistance training may especially result in increased cross-sectional area of type II fibers.[131]

The contraction time is increased with muscle hypertrophy because of the greater distance that must be traveled in the sarcoplasmic reticulum during contraction.[225] The increases in strength may not become apparent until the training program is well under way.[226] There is little effect on oxidative metabolism by muscle cells compared with endurance training. In fact, mitochondrial density may decline with strength training because of a dilutional effect by the larger quantity of myofibrils, although the absolute volume of mitochondria are unchanged.[226] In some instances, there may be a mild increase in oxidative capacity, suggested by the increase in type IIa muscle fibers with strength training seen in some studies.[227] In models of muscle hypertrophy, collagen content increases, with changes in collagen metabolism beginning as early as 3 days after increased loading.[58] Muscle fiber damage may also occur during vigorous strength training. The increased collagen content may prevent skeletal muscles from contracting against excessive loads. Lactate accumulation in overworked muscles may contribute to increased collagen synthesis by stimulating proline hydroxylase.

Compensatory hypertrophy of muscles may be induced by removal of one or more muscles of a synergistic group of muscles.[225] This results in overloading of the remaining muscle or muscles, with increased muscle weight and enlargement of remaining muscle fibers. In addition, there may be an increase in the number of fibers as a result of muscle fiber splitting. Weight training results in muscle fiber splitting of cats trained to lift weights.[225] The degree

of fiber splitting is related to the intensity of exercise, and all muscle fiber types may be affected.

Unfortunately, there is relatively little information regarding the effect of strength training on muscles in dogs, perhaps because of the difficulty in having them perform strength training activities and the difficulty in measuring changes in strength. A study of cats trained to move a bar and lift weights for a food reward resulted in muscle hypertrophy of 7% to 34% after 40 weeks.[228] A variety of studies have evaluated strength training in humans. Men participating in an exercise program designed to strengthen the quadriceps muscles had increased thigh girth, and muscle strength increased 40% with training.[229] In addition, endurance and time to exhaustion while cycling and running increased, indicating some benefit to endurance exercise. The effect of adding heavy-resistance training to increase leg-muscle strength was further studied in humans undergoing endurance training.[230] After 10 weeks, leg strength increased an average of 30%, but thigh girth, muscle fiber areas, and oxidative activities were unchanged. Short-term endurance was increased by 12%. Exercise to exhaustion also increased after strength training, indicating that certain types of endurance performance, particularly those requiring fast-twitch fiber recruitment, can be improved by strength-training supplementation.

Muscle strengthening may occur, even in older individuals. For example, one study of geriatric men compared the training effects of voluntary isometric contraction on the quadriceps femoris to determine if exercise changes age-related muscle weakness.[231] Maximal voluntary isometric contraction torque increased with even low training loads during 12 training sessions over 4 weeks. Altering dietary intake to achieve carbohydrate loading has been used to enhance muscle mass prior to competitions in humans, but one study indicated there was no advantage to carbohydrate loading to enhance muscle girth over weight-lifting alone.[232]

Although gender differences associated with strength training are unknown in dogs, female cats had greater muscle hypertrophy following weight training than male cats.[233] Gender differences also exist regarding skeletal muscle hypertrophy subsequent to heavy-resistance training in humans. Absolute changes in muscle mass are generally greater in men than women, but the percentage changes may not be significantly different.[234] For example, women completing a 20-week heavy-resistance weight training program for the lower extremity consisting of squats, vertical leg presses, leg extensions, and leg curls twice a week had a significant increase in maximal isotonic strength and hypertrophy of muscle fiber types I (15%), IIA (45%), and IIA + IIB (57%).[227] These data are similar to those in men and suggest considerable hypertrophy of muscle fiber types occurs in women if exercise intensity and duration are sufficient.[232]

In people, resistance training may, both alone and in combination with feeding, be a marked stimulator of human muscle protein synthesis and, in the absence of nutrition, sometimes muscle protein breakdown.[219] When feeding is superimposed on the postexercise stimulus, there is usually a net positive protein balance and protein synthesis occurs. The question of how much loading results in muscle gain often arises regarding the practice of lifting heavier loads to induce hypertrophy and strength. Acute studies in people indicate that increased muscle protein synthesis only occurs when intensities of load lifted exceed 60% of a single repetition maximum,[235] but at intensities beyond 60% and up to 90% of a single repetition maximum, there is a similar stimulation of muscle protein synthesis. Therefore it may be that resistance exercise at higher intensities has little additional value in stimulating muscle hypertrophy. However, low-intensity exercise (20% of single repetition maximum) with concurrent blood flow occlusion may stimulate muscle protein synthesis,[236] with an accompanying rise in S6K1 phosphorylation. Other studies have also shown that blood flow occlusion, when practiced chronically and with low lifting intensities (30% to 40% of single repetition maximum), can result in muscle hypertrophy and strength gains.[237] It is possible that venous occlusion results in local fatigue and the recruitment of type II muscle fibers, which are not normally recruited at low intensities. Recruitment of type II fibers results in activation of many signaling proteins, such as S6K1, ribosomal protein S6, extracellular signal–regulated kinase, and p38 mitogen-activated protein kinase, which are involved in regulating muscle protein synthesis.[238]

The Effects of Medications and Nutrition on Immobilized Muscles

Steroids

Naturally occurring or iatrogenic hyperadrenocorticism has been associated with myopathy in dogs.[239] Most dogs have muscle stiffness, proximal appendicular muscle enlargement, and myotonic discharges on electromyography. Muscle weakness and muscle atrophy may be present in some dogs. Histologic changes are characteristic of noninflammatory degenerative myopathy. Clinical signs of the myopathy may improve to varying degrees following treatment of hyperadrenocorticism. Steroids have a catabolic effect on muscle proteins, reduce protein synthesis, and alter the insulin responsiveness of muscle that is immobilized.[138] During treatment with corticosteroids or during catabolic conditions, glutamine synthetase increases and glutamine, the most abundant amino acid in muscle, is released at a high rate. On the other hand, endurance exercise reduces basal levels of glutamine synthetase.[240] In addition, there is increased susceptibility of type II fibers to atrophy, and type IIb fibers are more susceptible to atrophy than type IIa fibers.

Resistance exercise training may help attenuate the muscle loss associated with glucocorticoid administration by up to 40%.[240] Endurance training may also prevent glucocorticoid-induced muscle atrophy. Running attenuated muscle loss by 25-50% in rats receiving corticosteroids concurrently with the start of training. Prior endurance training does not appear to offer any advantage in preventing atrophy associated with corticosteroid administration compared with beginning training at the same time of steroid administration. Initiation of endurance exercise shortly after the onset of steroid treatment helps to prevent further muscle atrophy from that time forward, but does not prevent the atrophy that has already occurred. Atrophy sparing seems to occur to a greater extent in muscle types that are highly recruited during endurance training.

Androgens and synthetic anabolic steroids have been recommended to help attenuate loss of muscle mass because of their ability to increase protein synthesis and promote muscle growth.[240] Although the effects of anabolic steroids on athletes and athletic performance are controversial, they do not appear to enhance aerobic metabolism. Anabolic steroids do seem to be beneficial in patients in negative nitrogen balance, such as those patients who are undernourished. The influence of oral anabolic steroids on body mass index (BMI), lean body mass, anthropometric measures, and functional exercise capacity among humans with chronic obstructive pulmonary disease was evaluated.[241] A group receiving testosterone and daily oral stanozol for 27 weeks had increased weight, whereas the control group lost weight. Lean body mass, BMI, arm muscle circumference, and thigh circumference increased in the treated group, but there were no changes in endurance exercise capacity. Anecdotally, there may be some benefit to administration of anabolic steroids to dogs with chronic medical conditions that may result in muscle atrophy.

Antiinflammatory Drugs

The exact role of NSAIDs in the treatment of acute muscle injuries is unknown, but it has been suggested that these agents may be contraindicated, perhaps because NSAIDs may delay muscle regeneration following injury and inhibit exercise-induced satellite cell proliferation in humans. The cellular mechanisms by which NSAIDs may negatively affect muscle regeneration and inhibit the exercise-induced satellite cell proliferation are unknown. In one study, eight healthy male volunteers performed 200 maximal eccentric contractions with each leg.[242] Indomethacin, an NSAID, was infused into the vastus lateralis muscle of one leg to block local prostaglandin synthesis. Protein synthesis was determined from 24 to 28 hours following exercise. Myofibrillar and collagen protein synthesis were unaffected by local NSAID infusion. The expression of growth factors, such as TGF-β and IGF-1, and matrix-related genes increased after exercise and were unaffected by NSAIDs. Although NSAIDs seem to inhibit exercise-induced satellite cell proliferation, there appear to be only limited effects on gene expression and on postexercise protein synthesis. However, COX-2 specific inhibitors apparently have little effect on muscle protein synthesis or satellite cells in people.[243,244]

Muscle soreness may or may not be alleviated with NSAIDs or COX-2 specific inhibitors. The effects of two different NSAIDs and physiotherapy modalities on the healing of acute hamstring muscle tears of humans were studied.[245] Patients received meclofenamate, diclofenac, or a placebo. All patients received the same intensive physiotherapy treatment, including rest, ice, compression, elevation, therapeutic ultrasound, deep transverse friction massage, stretching, and exercise over a 7-day treatment period. Pain, swelling at the site of the muscle tear, and isokinetic muscle strength were improved in all groups, but there was no difference among groups. In fact, the pain score of the more severe injuries was significantly lower in the physiotherapy group than in those receiving NSAIDs and physiotherapy at day 7. Therefore there may not be an additive effect on the healing of acute muscle injuries when NSAIDs are added to standard physiotherapeutic modalities.

Growth Hormone

Growth hormone (GH) may help to improve muscle atrophy as a result of immobilization or denervation.[138] The actions of GH are mediated by complex interactions among several hormones, receptors, and binding proteins.[246] GH results in stimulation of protein synthesis. In addition, amino acid and glucose uptake are increased, and lipolysis is increased. Treatment with GH results in an increase in lean body mass in humans and rats, although strength is probably not improved. One of the primary effects of GH is the production of IGF-1 by the liver, which in turn also affects muscle tissue. IGF-1 may be a mediator of the response of GH because it also results in increased amino acid uptake and protein synthesis.[247] IGF-1 stimulates muscle satellite cells to enter the cell cycle and proliferate, with some differentiating and fusing with myofibers. However, most studies indicate that IGF-1 has minimal effect on the adaptation of muscle to changes in loading. The GH response to acute aerobic exercise may be augmented with repeated bouts of exercise.[248] During prolonged administration, resistance to the anabolic effects of GH may occur.

Administration of GH during periods of non–weight-bearing activity may not result in maintenance of muscle mass.[194,246] However, treatment during recovery may be beneficial. Beagle dogs receiving a GH secretagogue during a 10-week immobilization and 5-week remobilization period had a 60% decrease in tetanic torque during immobilization, which was similar to untreated dogs, but had a threefold increase in tetanic torque in the treated group compared with controls during remobilization.[139]

The diameters of type I and type II fibers measured from the vastus lateralis muscle followed the same trend. These data suggest that the GH secretagogue increased the size and strength of the quadriceps muscle during remobilization. GH may also attenuate the catabolic effects of corticosteroid treatment on muscle mass.[246]

Nutrition and Muscle

Amino acids, hormones, and physical activity appear to be the major short-term physiologic regulators of muscle mass, primarily through their actions on protein synthesis, whereas long-term regulation involves changes in gene expression. Amino acids are the main dietary components that stimulate muscle protein synthesis. In people, branched-chain amino acids, especially leucine, stimulate protein synthesis in a dose–dependent manner. The stimulatory effect of leucine on protein synthesis is mediated through upregulation of mRNA translation.[249] A number of mechanisms contribute to the effect of leucine on translation initiation. The increase in muscle protein synthesis may be mediated by a key component in the signaling pathway controlling these phosphorylation-induced mechanisms, a protein kinase called the *mammalian target of rapamycin* (mTOR). There is a limit, however, to the amount of muscle protein synthesis that can occur with a single meal. Leucine, either alone or with other essential amino acids, stimulates muscle protein synthesis for a short period of up to 2 hours. Muscle protein synthesis then returns to basal rates, possibly explaining why constant delivery of leucine does not maintain elevated muscle protein synthesis.[250] Increased dietary protein also induces enzymes of amino acid catabolism, particularly of the branched-chain and aromatic amino acids, and this may partially explain why muscle mass cannot be increased solely by eating more protein.[251] However, the effect of muscle protein breakdown by amino acid catabolism may be decreased when insulin release is stimulated, for example by glucose in a meal, because insulin apparently inhibits protein breakdown.

Branched-chain amino acid supplementation may have mild effects on muscle atrophy because of immobilization and help regulate protein turnover. In one study, short-term (6 days) HS of rats resulted in losses of myofibrillar proteins, total RNA, and rRNAs and pronounced atrophy of the soleus muscle.[252] Although the addition of branched-chain amino acids to the diet did not prevent muscle atrophy and had no apparent effect on regulators of proteasomal protein degradation, they reduced the loss of myofibrillar proteins and RNA, and attenuated several signal transduction proteins that act as regulators of protein synthesis and cell growth in muscle.

Leucine is especially important in the stimulation of muscle protein synthesis and can be a sole stimulus for muscle protein synthesis, but a full complement of amino acids are necessary for muscle synthesis.[219] Younger people seem to have greater ability to synthesize muscle protein than older people with the same level of amino acids available, suggesting a potential reduced signaling response for muscle synthesis in older people.

Providing appropriate nutritional components after exercise may affect muscle protein synthesis. When branched chain amino acids, including leucine, isoleucine, and valine were administered to people during and after one session of quadriceps muscle resistance exercise, an increase in mTOR, p70 S6 kinase, and S6 phosphorylation was found in the recovery period after exercise.[253] Exercise without branched-chain amino acids intake led to a partial phosphorylation of p70 S6 kinase without activating the enzyme. These results suggest that branched-chain amino acids administered after exercise may stimulate muscle protein synthesis.

Although insulin has little effect on protein synthesis in human muscle, it has an inhibitory effect on protein breakdown. In the immediate postexercise period there are marked increases in muscle protein breakdown.[254,255] When food containing protein and carbohydrate is given in the postexercise period, the increase in protein synthesis is exacerbated and the increase in the protein breakdown is blunted in an insulin-related effect, so there is a net increase in muscle protein synthesis.[256] Whereas exercise stimulates protein synthesis in muscle, disuse atrophy results in a marked decrease of muscle protein synthesis.[152]

Much discussion has occurred regarding protection of muscle from injury as a result of fatigue and excessive contraction in people. *N*-acetylcysteine has been suggested as a possible muscle protectant from overuse injury. Repeated electrical stimulation or eccentric exercise in which muscle lengthens while being loaded may cause injury.[257] Inflammation and muscle damage may result in delayed-onset muscle soreness.[258] Muscle-derived ROS may be involved in the development of muscle fatigue and may result in muscle damage as a result of excessive contractile activity.[259] The lipoxygenase pathway may promote the production of ROS. Although study results of other antioxidants to prevent postexercise muscle soreness have been mixed, *N*-acetylcysteine possesses antioxidant activity, increases glutathione synthesis, reduces ROS during muscle contractions, and scavenges hydrogen peroxide and hydroxyl free radicals. One study investigated the effects of pretreatment with *N*-acetylcysteine on fatiguing contractile activity by electrical stimulation of muscles.[259] Muscle fatigue was delayed, tetanic force was preserved, and plasma creatine kinase and lactate dehydrogenase activities were decreased in *N*-acetylcysteine–treated rats. The content of muscle-derived ROS in both gastrocnemius and soleus muscles was also decreased in *N*-acetylcysteine–treated rats, suggesting that pretreatment has a protective effect against injury induced by fatiguing contractile activity in skeletal muscle.

Skeletal muscle recovery from disuse atrophy requires the recruitment of insulin signaling for muscle growth, which is driven by protein synthesis. There is some belief that dietary fish oil may enhance insulin signaling and protein metabolism. One study was performed to evaluate whether dietary fish oil facilitates muscle recovery during remobilization after disuse atrophy.[260] Immobilization for 10 days, followed by 3 or 13 days of remobilization, was applied to the hindlimbs of rats fed corn oil (control group) or fish oil as a source of dietary fat. The immobilization-induced reductions in soleus muscle weight and myosin heavy-chain content were significantly restored by 3 days of remobilization in the group receiving corn oil. However, muscle recovery in the fish oil group did not significantly change until 13 days of remobilization. Throughout the remobilization period, levels of prostaglandin $F_2\alpha$ and COX-2 mRNA were significantly increased, although the increase of prostaglandin $F_2\alpha$ was less in the fish oil group until day 13. These findings suggest that dietary fish oil may inhibit early muscle recovery after disuse atrophy by suppressing signaling pathways and prostaglandin $F_2\alpha$ synthesis.

Recently, vitamin D receptors have been found in muscle tissue.[261] Vitamin D deficiency has been associated with muscle weakness and changes in EMGs, which improve with vitamin D supplementation. There may also be atrophy of type II muscle fibers with vitamin D deficiency. Other studies have shown a relationship between 25-hydroxycholecalciferol levels and various measures of muscle strength and function with aging. Vitamin D receptor expression decreases with age and is unaffected by vitamin D levels. Vitamin D may have a role in transcription and regulation of IGF binding protein, which binds IGF in tissues, including muscle. Vitamin D levels also have an effect on membrane calcium channels of muscle cells in numerous species.[262] Because calcium is critical for skeletal muscle function, vitamin D levels may have a significant effect on muscle function, performance, and injury risk.

Ligaments and Tendons

Ligament and Tendon Structure and Biomechanics

Ligaments and tendons consist primarily of type I collagen that forms parallel fibers. Ligaments extend between bones to stabilize joints, whereas tendons permit movement of bones by connecting muscles to bones.[58] Both are relatively pliant and flexible to allow natural movements of the bone to which they attach. They are also strong and relatively inextensible to offer resistance to applied forces, especially tensile forces. Ligament and tendon structure and chemical composition are very similar in humans and many animal species, including rats, rabbits, dogs, and monkeys. They are composed of dense connective tissue with primarily parallel fibers of collagen, with a small number of collagen fibers that run perpendicular to the predominant direction of stress. Cross-links that form between collagen chains are important to collagen tensile strength and resistance to proteases.[263] Reducible cross-links are found in newly formed collagen, while nonreducible cross-links are found in mature collagen and account for stronger and stiffer collagen. The concentration of cross-links and their ratio varies between species and location. For example, the rabbit CCL has three to five times more nonreducible cross-links than the patellar tendon.[263] Ligaments also have more cells per mass than tendons.[263]

Most ligaments do not function as a unit structure because at a particular joint position, one portion of a ligament may be taut while another part is relaxed.[264] This is relevant because certain joint positions may need to be avoided to reduce stress on healing ligaments. Ligament insertions may be direct or indirect.[264] With a direct insertion, collagen fibrils course directly into the bone, with some fibers blending with the periosteum. In contrast, indirect insertions have a zone of fibrocartilage and mineralized fibrocartilage between the ligament and cortical bone. These zones are important when considering ligament insertions and their responses to stress and rehabilitation from injury.

The biomechanical responses of ligaments and tendons are nonlinear because of progressive fiber recruitment and viscoelastic properties. The structural properties of the entire bone-ligament-bone complex that must be considered depend on the mechanical properties of the ligament, macroscopic and microscopic ligament geometry, the cross-sectional area and length of the ligament, and the material properties of the fibrous and ground substance constituents.

When initially loaded, ligaments and tendons stretch with a slight increase in load. On histologic examination, the fibers change from a relaxed wavy appearance to a parallel alignment as the fibers become oriented in the direction of loading. This region on a load-deformation curve is known as the *toe region* (Figure 7-9). As the load is increased, the curve becomes more linear, corresponding to full parallel orientation of the fibers. The linear portion of the curve also indicates elastic deformation, with return to normal structure when the load is released. If loading continues, the load-deformation curve exits the linear region and enters the region of plastic deformation. This point is termed the *yield point.* From a histologic perspective, some microfailure may occur even before the yield point is reached, resulting in damage to the ligament or tendon. After the yield point is reached, gross failure of the ligament occurs and permanent structural damage occurs and remains even if the load is released. When the ligament or tendon fails and can support only a negligible load, the ultimate failure point has been reached. It is important to

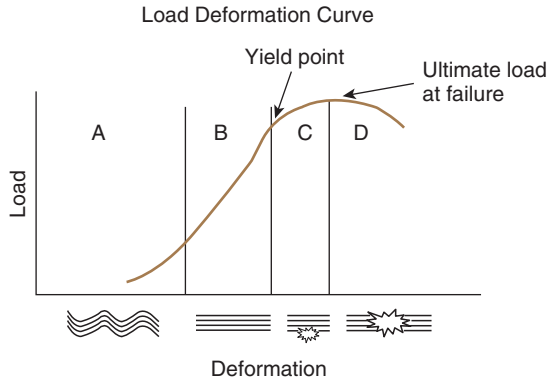


Figure 7-9 Load-deformation curve of ligament biomechanical testing. **A,** Toe region. Note the bottom portion of the diagram indicating the wavy appearance of the collagen fibers. **B,** Linear physiologic loading and zone of elastic deformation. The collagen fibers become taut. **C,** Early permanent damage and zone of plastic deformation. There is damage to some of the fibers. **D,** Ultimate load at failure. There is gross disruption of the ligament.

note that the ligament may retain continuity despite micro- and macrofailure.

The mechanical properties of ligaments and tendons are a reflection of their material characteristics, allowing for comparison between other ligaments and tendons. They also reflect the orientation and organization of the collagen fibers. High strain–rate techniques for studying knee ligament failure in people have replaced the previous low strain–rate methods and distinguish the failure mechanism of ligaments from that of bone. For example, anterior cruciate bone-ligament-bone preparations of primates failed at a higher load and absorbed more energy at a faster rate of deformation than at a slower rate.[265] Specimens failed by ligament disruption at faster rates, and by tibial avulsion fractures at slower rates. Ligament function is further defined by measuring the restraining force provided by specific ligaments. Advances in analytic techniques and models permit precise analysis of joint position, motion, and laxity.[266]

Recent studies have indicated that, in addition to tendon and ligament support, neuromuscular reflexes may play a large role in joint stability.[264] Impulses from mechanoreceptors in the joint capsule, menisci, and ligaments transmitted to the spinal cord result in instantaneous reflex regulation of muscles acting on a joint to provide coordinated joint function and stability. Canine lateral collateral ligaments have nerve fibers and Golgi-Mazzoni and Ruffini corpuscles, some of which resemble Golgi tendon organs.[267] Mechanoreceptors are more numerous at the ends than in the middle of ligaments, and they are present in both the subsynovial and the interfascicular connective tissue and are surrounded by vessel networks. The number of corpuscles may be related to the type of stress to which a ligament is subjected. ACL-deficient humans have disruption of an ACL-hamstring rapid reflex arc, which normally helps to limit anterior motion of the tibia.[264] Instead, a slower reflex originating from the knee joint capsule limits anterior tibial motion. Rehabilitation techniques to help restore ligament-muscle reflexes should be considered.

Knowledge of the blood supply to ligaments and tendons is necessary to understand how healing and rehabilitation may be affected. Blood flow to the proximal, middle, and distal parts of the CCL were 3, 2.2, and 3.5 mL per 100 g of tissue per minute, respectively in one study.[268] The respective values in the caudal cruciate ligament were 2.8, 2.8, and 3.2 mL per 100 g of tissue per minute. The cruciate ligaments are relatively hypovascular, with blood flow only 50% of that of the synovium of the suprapatellar pouch.[269] Application of a cranial stress diminishes the blood flow to the ligament to one fifth of the baseline value, an effect that is reversible. Division of the infrapatellar fat pad causes a twofold decrease in perfusion to the ligament, whereas dissection of the enveloping synovium results in a complete cessation of blood flow.

Many factors affect the ligament-bone unit response. Increasing age weakens the tissue-bone preparation.[270] Tendons also respond biochemically to the stresses placed on them.[271] In compression regions of rabbit tendons, the GAG content is 15 times greater than in tension-transmitting regions. Dermatan sulfate predominates in regions of tension, whereas chondroitin sulfate is the major GAG in regions of compression. The relative GAG content changes with alterations in the stresses placed on tendons induced by transposing the tendon, and the content reverts back to normal when the tendon is replaced to its original position. Other factors affecting tendons and ligaments include the amount of activity the animal undergoes and medications the patient may receive.

Response of Ligaments and Tendons to Disuse and Immobilization

Immobilization is commonly used to treat injuries to ligaments and tendons. There are many models of ligament and tendon disuse, including internal and external fixation methods, casts and slings, denervation, disarticulation, and bed rest. These models have allowed the study of histologic, biochemical, and biomechanical changes that occur with disuse. There is an adverse decline in structural and material properties of ligaments and tendons with immobilization of the joints that they cross. Even if joint motion is allowed, stress deprivation rapidly reduces the mechanical properties of the tendon and ligament tissues (Box 7-8).[272] The bone-tendon/bone-ligament complex is especially affected by immobilization. The effects of stress deprivation appear to be time and dose dependent, and differences may exist in the responses of various ligaments and tendons within the same animal because of differences in cellular, structural, and biochemical properties. For example, the CCL may be more resistant to stress

Box 7-8 Summary of the Effects of Disuse and Immobilization on Ligaments and Tendons

- Stress deprivation rapidly reduces mechanical properties of tendons and ligaments.
- Bone-tendon and bone-ligament complexes are especially affected.
- Time and dose of immobilization and differences in cellular, structural, and biochemical properties affect degree of changes.
- Decreased cross-sectional area, disorganization of parallel structure of fibrils and cells, a net decrease in collagen mass, and reduced GAG, HA, chondroitin sulfate, dermatan sulfate, and water content with immobilization.
- May have a 0.2%/week loss of ligament strength and energy to failure with cage confinement.
- May have a 45% loss of load to failure, and up to 75% decrease in cranial cruciate ligament stiffness with limb immobilization for 12 weeks.
- Stifle medial collateral stiffness may decrease 40% with 6 weeks of immobilization and take 18 weeks to return to normal biomechanical properties after remobilization.
- Continued joint motion during reduced weight bearing helps preserve ligament properties.
- Effects of immobilization on tendon are less severe compared with ligaments.
- Recovery of bony insertion sites is prolonged compared with ligament/tendon midsubstance, up to 1 year.
  - Bony resorption occurs at ligament bone interface.

GAG, Glycosaminoglycan; *HA,* hyaluronic acid.

deprivation than the MCL.[272,273] Typically, there is reduction of cross-sectional area; disorganization of the parallel structure of fibrils and cells; increased collagen turnover with increased collagen synthesis and degradation; a net decrease in collagen mass; and reduced GAG, HA, chondroitin sulfate, dermatan sulfate, and water content.[272] Remobilization returns the mechanical properties to nearly normal over time, but the recovery of the bony insertion sites is prolonged compared with the ligament-tendon midsubstance. Knowledge of the detrimental effects of immobilization may influence the management of ligament and tendon injuries.

It is well recognized that a reduction of activity, such as confinement to a cage, may affect ligament and tendon properties.[273] A study of related rhesus monkeys showed a 0.2% loss in strength and energy to failure per week of captivity in the CCL.[274] More profound effects may be expected with complete immobilization of a limb. In a classic study, the effect of body cast immobilization on the biomechanical properties of bone-CCL-bone units of rhesus monkeys was studied.[275] After 8 weeks of total-body plaster immobilization, there were significant decreases in maximum failure load and energy absorbed to failure (39% and 32%, respectively). There was also a 69% decrease in ligament stiffness. These changes likely indicate an alteration in the functional capacity of the ligament to resist loading and elongation, factors that relate to the ligament's ability to provide joint stability. The immobilized ligament units frequently failed by avulsion fractures of the bone, which correlated with bone resorption in the cortex immediately beneath the ligament insertion site. The effect of immobility on knee ligaments depend, in part, on the histologic characteristics of the ligament-bone attachment. The ligament-bone junctions with zones of fibrocartilage were little affected. In contrast, the MCL, which inserts directly into the tibia and periosteum without well-defined zones of cartilage, showed marked interruption after immobility as the result of bone resorption in the subperiosteal and outer cortical regions. In some places the ligament was attached only to the overlying periosteum. After 5 months of remobilization following total body immobilization, there was only partial recovery in ligament strength, although ligament stiffness and compliance parameters returned to control values. Up to 12 months were required for nearly complete recovery of ligament strength.

Similar effects of immobilization have been noted on canine ligaments. The effect of immobilization on the CCL of dogs was studied using a model of internal skeletal fixation for 12 weeks.[29] Failure of the femur-ligament-tibia complex occurred through the tibial insertion of the ligament for both experimental and control limbs. The load at failure and stiffness of the immobilized limbs were 45% and 73%, respectively, of the nonimmobilized CCL. Even though there was a 13% reduction in soft tissue mass, the loss of collagen was greater in the tibia and femur than in the CCL, and correlated with mechanical failure at the bony insertion. Bone atrophy was the result of increased resorption of bone rather than decreased bone formation. This study demonstrated that there was significant atrophy of the CCL and bone in immobilized joints of dogs.

Other studies have confirmed the importance of bone resorption and the reduction of bone collagen resulting from disuse and its relationship to strength of the medial collateral and CCL complexes.[273,276] In a similar study of canine stifles immobilized in a cast for 12 weeks, changes in the tibial insertion of the ligament were apparent, including the presence of many osteoclasts, numerous large fibroblasts, and replacement of bone by loosely arranged fibrous tissue.[273] Interestingly, in this study the femoral attachments of the medial and lateral collateral ligaments and the origin of the CCL showed little bone resorption, indicating that the response to immobilization may be site-specific. Even so, MCL strength decreased 40% following 6 weeks of immobilization.[277] Restriction of activity to cage confinement also causes significant bone atrophy of the tibial insertion sites of the collateral ligaments if adequate activity is not allowed. Additionally, there is decreased

thickness of ligament and tendon fiber bundles, greater extensibility per unit load, and unchanged collagen content after immobilization.

Although stress deprivation with immobilization appears to reduce the mechanical properties of ligaments and tendons in a time- and dose-dependent manner, stress deprivation with continued mobility may have different effects. Maintaining joint motion and reducing the period of immobilization may help preserve ligament properties. The effects of stress deprivation with joint motion on the CCL were studied in dogs by freeing the tibial insertion of the ligament and moving it caudally and securing it with a screw.[278] Stifle joints were allowed complete mobility. The cross-sectional area of the ligament was 115% larger than controls at 6 weeks. The tensile strength was unchanged at 6 weeks, but was only 67% of control values at 12 weeks. This model of stress deprivation with continued joint motion on the CCL suggests that there may be an increase in ligament cross-sectional area and a concurrent decrease in biomechanical properties after a time, although the changes are less severe than immobilization without joint motion.[272]

Other models of stress deprivation with joint motion for relatively short periods have shown different results. The effect of active joint motion on ligament and meniscus mass in a non–weight-bearing model of disuse was studied. A unilateral tarsal disarticulation model of disuse that maintained active knee motion without weight bearing for 8 weeks was studied in dogs.[30] A large and similar loss of collagen and calcium mass occurred in the femur and tibia, indicating net bone resorption. No loss of tissue mass occurred in the collateral and cruciate ligaments or menisci. The strength of the femur-CCL-tibia complex was not qualitatively or quantitatively different between control and experimental limbs. So although immobilization for greater than 8 weeks results in atrophy of bone, ligament, tendon, and meniscus in some canine studies, the absence of weight bearing for 6 to 8 weeks with maintenance of joint motion appears to help preserve femur-ligament-tibia complex mechanical strength, despite the occurrence of bone atrophy.

The effects of immobilization on the biomechanical and morphologic properties of the femur-MCL-tibia (FMT) complex and its components have also been investigated in rabbits.[279] After 9 weeks of immobilization, FMT stiffness was significantly reduced, and the ultimate load to failure and energy absorbing capacity were reduced by 69% and 82%, respectively, and an increased number of failures occurred by tibial avulsion. On histologic examination, the femoral and tibial insertion sites showed increased osteoclastic activity, resorption of bone, and disruption of the normal attachment of the bone to the ligament. The cross-sectional area of the CCL also decreased with immobilization of the stifle joint.[280] Diminished stress also results in increased collagen turnover, with formation of immature collagen.[264]

Compared with ligament, relatively less is known regarding the effects of immobilization and subsequent recovery on tendon. In a study of healthy young men, the nondominant leg was immobilized for 2 weeks.[281] Immobilization caused a 6% decrease in muscle size and 9% decrease in muscle strength, although Achilles tendon size and collagen turnover were unchanged. Another study evaluated people with ankle fractures that were immobilized for 7 weeks.[282] Achilles tendon cross-sectional area did not change significantly during immobilization, whereas calf muscle cross-sectional area and strength decreased 15% and 54%, respectively. Local collagen turnover increased in the immobilized leg after 7 weeks of immobilization. Therefore tendon seems to be more resistant to short-term immobilization as compared with muscle.

Small animals may have some loss of tendon properties with immobilization. There is a dose-dependent effect of stress shielding on the mechanical properties of rabbit patellar tendons.[272] In addition to decreased ultimate stress of tendons with stress shielding, there is a significant increase in the cross-sectional area of tendons, despite fewer numbers of collagen fibrils per unit area. There was a significant loss of ultimate stress on tendons after 1 to 3 weeks.[272]

Tenascin-C, a protein with limited expression in normal musculoskeletal tissues, provides elasticity in mesenchymal tissues subjected to heavy tensile loading. Tenascin-C is expressed in normal rat myotendinous and myofascial junctions, and the Achilles tendon, but is absent in normal skeletal muscle. Following cast immobilization for 3 weeks, tenascin-C was decreased or was completely absent in the regions expressing tenascin-C normally.[283]

As the previously discussed studies have indicated, ligaments and tendons are not metabolically inert structures, although their oxygen consumption is approximately 10 times lower than liver tissue and 7.5 times less than skeletal muscles. Immobilization of rat limbs causes an additional 36% reduction in oxygen consumption and decreased aerobic enzyme activity of ligaments and tendons, suggesting that the metabolic activity is further lowered with decreased levels of physical activity.[284]

Response of Ligaments and Tendons to Remobilization

Although the mechanical properties of immobilized ligaments return to normal relatively quickly,[272] the load to failure of the bone-ligament-bone complex lags behind, indicating that there is asynchronous healing of the bone-ligament-bone complex. After 6 weeks of immobilization of the lower limbs of dogs, 18 weeks of remobilization was necessary for return of the normal structural properties of the FMT complex.[273] In fact, up to 1 year of remobilization may be required for normalization of the ligament-tibia complex in some instances, whereas the mechanical properties of the ligament return to normal in a relatively short

period of time, as newly synthesized collagen fibers gradually mature and strengthen with subsequent stress resumption.

In the study of immobilized stifles of rhesus monkeys, 5 or 12 months of remobilization was allowed after casting for 8 weeks.[275] Following 5 months of rehabilitation, there was still a 20% deficit in load to failure and energy stored at failure, but only a 7% deficit in stiffness. After 12 months of rehabilitation, there was only a 9% deficit in load to failure and an 8% deficit in energy stored to failure.

The effects of remobilization following 9 weeks of immobilization were studied in the rabbit FMT complex.[279] With 9 weeks of remobilization, the ultimate load and energy-absorbing capabilities of the bone-ligament complex improved to approximately 80% of normal. Following 12 weeks of immobilization, 1 year was required to recover the functional properties. Failure by tibial avulsion became less frequent, and the stress-strain characteristics of the MCL returned to normal. Histologic examination found that the sites of insertion of the ligament also showed evidence of recovery.

Little is known about the effect of stretching on the biochemical and morphologic features of tendons that have been immobilized. One study evaluated the response of rat tendons to stretching after immobilization.[285] Immobilization followed by stretching resulted in greater chondroitin sulfate at the compression region of tendons. Immobilized animals exhibited the greatest disorganization of collagen bundles, which was partially recovered after stretching or with remobilization. Although the results of this study did not result in recovery with stretching to the degree that occurred with free activity, a revision in stretching procedures, such as duration and frequency of the sessions, may be beneficial in cases of immobilized tendons.

In the study of healthy young men previously reviewed, the nondominant leg was immobilized for 2 weeks, followed by a recovery period of 2 weeks. Although there was a decrease in muscle size and mass with immobilization, there was no change in Achilles tendon size or collagen turnover. Remobilization resulted in increased muscle size, but strength was unchanged. No differences in tendon size could be detected between the two legs after 2 weeks of recovery, although collagen synthesis was increased in the previously immobilized leg.[281] A further study evaluated people with ankle fractures that were immobilized for 7 weeks, followed by a 7-week remobilization period.[286] Again, Achilles tendon cross-sectional area did not change significantly during either immobilization or remobilization, but calf muscle size and strength decreased during immobilization; although there was some muscle size and strength recovery after 7 weeks of remobilization, it was still not complete.

Tenascin-C expression increased in rats with free cage activity for 8 weeks following immobilization, although not to normal levels.[283] More strenuous loading after immobilization in the form of 8 weeks of low- and high-intensity treadmill running resulted in greater tenascin-C expression in myotendinous and myofascial junctions and the Achilles tendon, reaching the level of normal tissues, suggesting that some level of increased loading is beneficial in recovery of tendon structure after immobilization.

Postoperative Healing of Tendons and Ligaments

The function of tendons and ligaments is to transmit tensile forces. Early studies suggested that healing ligaments should be immobilized for proper healing. Immobilization of surgically repaired tendons or ligaments for 6 weeks results in a wound with approximately 50% the normal tensile strength for tendons and ligaments.[287] Recently, it has been suggested that early, controlled mobilization of healing tendon and ligament repairs may be beneficial. Earlier tension across a ligament or tendon wound orients the healing fibers and results in stronger healing. For example, MCLs of canine stifles sutured with polyester suture in a locking loop pattern and immobilized for 3 weeks, followed by active motion, resulted in valgus-varus laxity of 150% and ultimate strength of 92% compared with intact controls. In comparison, doubling the length of the immobilization period to 6 weeks resulted in valgus-varus laxity of 300% and only 14% of the ultimate strength of controls. Repaired canine tendons immobilized for 3 to 6 weeks after surgery also apparently have sufficient strength to allow protected active motion. Current knowledge of healing tendons and ligaments suggests that postoperative immobilization for 3 weeks will allow acceptable return to function while minimizing the risk of damage if appropriate suturing techniques are used. The immobilization splint should be designed to allow some active motion of the joints if possible, but should limit the amount of weight bearing (Figure 7-10).[288] After immobilization is completed and coaptation devices are removed, the amount and type of exercise allowed should be severely limited and increased gradually over time. Consideration should be made to protective coaptation if the owner is unable to adequately restrict the animal. By 6 weeks after repair, tendons have approximately 50% of tensile strength as compared with normal. It is believed that tendons need only 25-33% of their original strength to withstand the forces of normal physiologic muscle contractions, indicating that some light activity should not be detrimental to healing.[287]

Medial Collateral Ligament

Several studies have evaluated early mobilization following injury to the MCL. In one study, injury to the MCL was induced by making incisions through the superficial and deep portions of the ligament, and then repair was performed by suturing the incision.[277] Ligaments that were not immobilized during the 6-week healing period were



Figure 7-10 Coaptation following repair of a superficial digital flexor laceration. Note the external skeletal fixator is configured to prevent flexion of the tarsus and weight bearing on the digits to limit stress on the healing repair.

stronger than those that were immobilized with a cast, suggesting that ligament strength is sensitive to physical activity. The effects of early mobilization and exercise on healing of injured MCL were further studied.[289-291] Transected canine MCL were subjected to either no surgical repair with 6 weeks of mobilization, surgical repair with 3 weeks immobilization at 90 degrees followed by 3 weeks remobilization, or surgical repair with 6 weeks of immobilization. Knees were tested in tension to determine the structural properties of the FMT complex and the mechanical properties of the healing MCL. Laxity increased in all knees, but early mobilization resulted in the best joint stability. The structural properties of the FMT complex and the MCL substance were also improved in the early mobilization groups. Stiffness of the MCL in early mobilization groups was approximately 60% of controls, whereas stiffness of prolonged immobilization stifles was only 36% of control stifles. The load at failure of the FMT complex was approximately 40% and 20% of normal stifles in early mobilized versus immobilized stifles, respectively. Similarly, the tensile strength of the MCL was approximately four times greater in stifles that underwent early mobilization, but values of immobilized limbs were only 20% of normal stifles. These studies indicate that early mobilization (less than 3 weeks of immobilization) is the treatment of choice in cases with isolated MCL injuries. It is important to realize that an intact CCL provides significant stability to the stifle joint and allows the MCL to heal. If the CCL is intact, implementation of early motion is recommended.

The long-term effects of surgical repair and immobilization on the healing of the transected MCL have also been studied in a canine model.[292] In one group, the transected MCL of the left knee was not repaired, and the joint was not immobilized, whereas in the other group the MCL was repaired and the joint was immobilized for 6 weeks. Types I and III collagen, reducible collagen cross-links, and total collagen concentration for both groups returned to normal levels by 48 weeks postoperatively. Biomechanically, the nonimmobilized group achieved better results than the group that was immobilized in terms of varus-valgus knee motion and ultimate load of the FMT complex. However, the healing MCLs had tensile strength of only 62% and 45% of controls for the two groups at 48 weeks. These results suggested that conservative treatment with early mobilization might be better than surgical treatment with immobilization for an isolated grade III MCL injury.

Healing ligaments may be less able to resist excessive strain during the repetitive loading experienced during daily activity. The ability of ligaments to resist repetitive low cyclic loading (<10% of the normal ligament failure strength) was studied in normal and healing rabbit MCLs.[293] MCLs were surgically gapped in either a unilateral or bilateral fashion with immobilization of the right hindlimb in the bilateral group. MCL scars were allowed to heal for 3, 6, and 14 weeks and were cyclic creep tested at increasing forces. The cyclic modulus of the nonimmobilized scars was less than that of normal ligaments. The percent increase in modulus during cycling was greater for scars than for normal ligaments. Furthermore, immobilization significantly decreased the ability of scars to resist strain, with a majority of immobilized scars failing during repetitive loading. Failures were preceded by a reduction in cyclic modulus indicating damage to the healing ligaments that resulted in eventual total failure. These results suggest that joints with healing ligaments may have increased strain in joint structures while they are under stress, potentially leading to joint instability. Although immobilization could be used temporarily to maintain joint stability, remobilization would likely lead to total failure of the previously immobilized ligament.

Injuries to multiple stifle ligaments may affect healing of the MCL. Although the previous studies have suggested that during normal stifle motion the functional deficit of the MCL in valgus rotation is compensated for by the remaining structures, especially the CCL, MCL strength was just 14% of normal after 14 weeks of healing when both ligaments were transected, compared with transection of the MCL alone, which was 52% of control values.[294] There were also marked arthritic changes in the joint when both ligaments were transected.

Thermal shrinkage has been used in other ligaments to improve healing. The effects of immobilization on healing collagenous tissue treated with thermal shrinkage were investigated in a rabbit model.[295] Thermal shrinkage was

performed on the medial collateral ligaments of rabbit knees. The joints were immobilized in half of the knees. Animals were evaluated 0, 3, 6, and 9 weeks postoperatively. The failure load of shrunken ligaments was significantly lower than that of the intact ligaments on the day of operation. Shrunken ligaments reached their highest failure loads in the third week. The immobilized ligaments remained weaker than the mobile group, but this difference was statistically significant only in the ninth week. Immobilization seems to have negative effects on the healing ligament.

Lateral collateral ligament healing is similar to healing of the MCL.[264] Repaired ligaments heal faster with more organized tissue than nonrepaired ligaments, but both are still weaker than control ligaments after 10 weeks of healing.[296]

Cranial Cruciate Ligament

Transected ends of the CCL retract and do not join together, as other ligaments may. Repair of transected ligaments results in resorption of 40% of the ligament 24 months after repair, and weakness persists after 4 years of healing.[297] The CCL is not normally in contact with synovial fluid because of an overlying synovial membrane. However, when the ligament is damaged, synovial fluid may bathe the damaged ligament, which may inhibit healing because of the inhibitory effects of synovial fluid on fibroblast proliferation.[264] In addition, there are increased levels of collagenase with cruciate ligament and meniscal injuries. A study of experimentally created CCL and MCL transection indicated that postoperative immobilization for 6 weeks resulted in greater arthritic changes in articular cartilage than in dogs undergoing early stifle mobilization.[298]

Human patients undergoing rehabilitation consisting of immediate knee motion and early weight bearing do not have an increased incidence of abnormal knee displacement in the early phases after undergoing bone–patellar tendon–bone allograft or iliotibial band extraarticular stabilization of ACL injuries.[299] Although similar studies have not been conducted in dogs, our clinical impression is that there is not an increased incidence of stifle instability in dogs undergoing rehabilitation, and, in fact, there may be less displacement because of the additional stability afforded by greater muscle mass of patients undergoing rehabilitation.

Flexor Tendons

Immediate immobilization of flexor tendons following repair results in diminished ultimate tensile strength in the early postoperative period. Using an early passive motion protocol, ultimate tensile strength and ultimate load are increased 2-3 times more than if the limb is immobilized.[300] In addition, joint motion is significantly greater than if the joints are immobilized. Early protected passive motion augments biomechanical properties and excursion of healing flexor tendons. These results were confirmed in a study that compared different suturing techniques in dogs undergoing early mobilization following repair of lacerated deep digital flexor tendons. The mechanical and histologic healing of tendon repairs using early active motion was evaluated with three flexor tendon repair techniques over a 21-day period.[288] Adhesions occurred in 15% of the tendon splint repairs. Smooth tendon gliding was obtained in the other specimens in which repairs were successful. Histologic examination demonstrated evidence of progressive healing without surrounding adhesions in repaired tendons. Improved suture techniques have the potential to withstand the stress produced by active digital motion protocols. Immobilization may also result in adhesion formation, reducing the excursion of healing tendons and reduced ROM. In one study, 10 minutes of daily passive motion, with 1.5 to 1.7 mm of tendon excursion prevented adhesions in a sharp transection model of the deep digital flexor tendon.[301,302] Doubling the excursion resulted in no additional improvement. Therefore less aggressive passive-motion exercises may be sufficient to prevent adhesion formation. In fact, overly aggressive ROM rehabilitation may be deleterious. Tendons undergoing excessive motion with formation of a large gap between tendon ends are at risk for rupture during early healing.[303] Increased rate of passive motion also has beneficial effects. In one study of healing canine digital flexor tendons, passive motion at a rate of 12 cycles/min for 5 minutes daily resulted in similar gliding function with significantly greater tensile properties than dogs receiving 1 cycle/min for 60 minutes.[304] Repair of tendons should occur within 7 days for optimum restoration of gliding function.[305] Although repair of tendons as long as 21 days after injury results in similar strength and stiffness as tendons undergoing immediate repair, there is significant loss of gliding function with delayed repair.

There may be some benefit to delaying rehabilitation for several days after tendon repair. One study compared the short-term outcome following flexor tendon repair and postoperative rehabilitation beginning on day 1 versus day 5 in dogs.[306] Initiating postoperative mobilization at day 5 resulted in no tendon ruptures compared with tendon ruptures in four of the dogs (33%) in the group subjected to mobilization starting at day 1. There was a trend toward lower resistance of flexion in the day 5 start group, but there were no differences between the groups by day 10. Starting rehabilitation on day 5, when initial gliding resistance is lower, may have an advantage over earlier starting times, when surgical edema and other factors increase the initial force requirements to initiate tendon gliding.

Rehabilitation of partial flexor tendon lacerations may be different than for complete lacerations. In one study of partial lacerations of the deep digital flexor tendon, dogs not undergoing surgical repair had higher ultimate loads and stiffness than repair groups.[307] In addition, passive

rehabilitation of nonrepaired tendons resulted in less gap formation than unrestricted active mobilization of repaired tendons.

Both passive and active rehabilitation are superior to immobilization following partial tendon laceration, but few studies have directly compared the two. Gap formation, adhesions, gliding function, and structural properties of repaired and unrepaired tendons were evaluated following 3 weeks of unrestricted active rehabilitation versus passive mobilization for partial laceration of canine deep digital flexor tendons.[308] There were no differences in tendon excursion, total joint rotation, or adhesions between any groups. Gap size was higher with active mobilization. There was no effect of rehabilitation protocol on the strength or stiffness of healing tendons at 3 weeks. Tendon repair did not affect tendon strength, but did produce higher stiffness in healing tendons at 3 weeks. The authors concluded that active rehabilitation appears safe for partial lacerations less than 60%. Although repair appears to weaken the tendon in the early stages of healing, it may provide some biomechanical benefit by the middle stages of healing.

Retaining vascularity does not seem to prevent significant reduction in tendon properties that occur postoperatively, nor does it accelerate the return in strength and stiffness.[309] The medial half of the patellar tendon was placed as a vascularized graft to replace the ACL on one side and as a nonvascularized graft on the other in monkeys. Grafts had only 57% of control ACL stiffness and 39% of control maximum force by 1 year. Elastic modulus and maximum stress also increased over time, but at 1 year were only 34% and 26% of normal values, respectively.

Little is known about the localized changes in bone mass that occur following tendon or ligament injury. Interruption of normal load transfer at the insertion site may lead to localized loss of bone. In a canine study, the deep digital flexor tendon was transected from its insertion and either repaired or left unrepaired.[310] Postoperatively, limbs in the repair group were cast immobilized except for 10 minutes of daily passive mobilization rehabilitation, whereas the laceration-only group dogs were allowed full weight bearing. At 5-42 days postinjury, bone mineral density (BMD) was assessed. There was significant bone loss in the distal phalanx after tendon laceration, repair, and postoperative passive mobilization, with BMD decreases of 20%, 40%, and 41% at 10, 21, and 42 days. In contrast, BMD was reduced by 28% in bones subjected to tendon laceration and full weight bearing. Osteoclastic resorption occurred in both groups. Bone loss may affect healing by impeding restoration of a strong tendon-bone interface.

Modalities, such as therapeutic ultrasound, may aid tendon healing. The Achilles tendon of five dogs was severed proximal to its insertion.[311] Tenorrhaphy was performed using locking-loop sutures. A cortical screw was used to immobilize the tibiotarsal joint, and it was removed 4 weeks after tenorrhaphy. Ultrasound therapy was given to the treated group starting the third day postoperatively using 0.5 W/cm^2 for 10 minutes daily for 10 days. The dogs showed significant lameness for the first 4-5 days, which disappeared earlier in the ultrasound-treated dogs than the controls. By day 40, the echogenicity was improved in the treated group compared with controls. However, the tendon showed near normal echoic texture in both groups by day 120. The Achilles tendon in treated dogs showed comparatively fewer adhesions than in controls. Histologic features of tendon healing were improved in treated dogs throughout the study, and by day 120, the tendon tissue was comparatively acellular and looked like a normal tendon.

Rotator Cuff Tendons

The response of healing tendons in different areas of the body may differ from flexor tendons. In studies of animal models of rotator cuff repair, immobilization may improve mechanical properties more than passive motion. The effects of daily passive motion or continuous immobilization on joint mechanics in a rat rotator cuff repair model following injury to the supraspinatus muscle were evaluated.[312] Continuous immobilization or passive motion was performed for 2 weeks before all underwent a remobilization protocol for 4 weeks. Total ROM was less with passive motion than with continuous immobilization at 2 weeks and remained significantly less following 4 weeks of remobilization. Joint stiffness at 2 weeks was increased with passive motion in comparison with continuous immobilization. There were no differences between the groups in terms of collagen organization or mechanical properties. The authors concluded that in this model, immediate postoperative passive motion was detrimental to passive shoulder mechanics, perhaps because passive motion increased scar formation in the subacromial space, resulting in decreased motion and increased joint stiffness.

A similar study concurred with these findings. Gimbel and colleagues investigated the effect of short and long durations of various activity levels on healing supraspinatus tendon to bone insertion sites in rats.[313] The postoperative activity level was controlled through shoulder immobilization, cage activity, or moderate exercise for 4 or 16 weeks. Decreased activity level had a positive effect on stiffness and modulus of elasticity of the insertion site at 16 weeks (immobilization > cage activity = exercise). Collagen disorganization was decreased with a decrease in activity level at 4 weeks, but was similar between groups at 16 weeks. Decreasing activity level by immobilizing the shoulder improved tendon to bone healing in this study.

Thermal shrinkage of the joint capsule is occasionally performed in mild cases of medial glenohumeral instability. It is important to immobilize the shoulder joint after surgery, but the optimal time of immobilization is unknown.

Ideal timing for remobilization is still controversial, and the length of time that the canine shoulder joint should be immobilized after thermal shrinkage is unknown, but abandoning immobilization protocols for longer than 3 weeks should be considered based on healing of stifle medial collateral ligaments with thermal shrinkage.[295]

Adaptations of Ligaments and Tendons to Increased Mechanical Stress

Much like other musculoskeletal tissue, ligaments and tendons also adapt to applied stress and motion. Ligaments and tendons are morphologically, biomechanically, and biochemically sensitive to stress deprivation as well as stress enhancement. Although testing procedures vary among studies, most have consistently demonstrated that training increases the strength of ligaments and tendons. Compared with other tissues, such as muscle, the metabolic activity of ligaments and tendons is lower, probably because of poorer vascularity and circulation.[314] The responses of ligaments and tendons to increased training stresses are therefore slower and less pronounced than in muscles. It is also important to consider the conditions under which laboratory animals are housed when interpreting the results of experimental studies because ligaments and tendons are very sensitive to stress deprivation, and limiting normal activity with cage confinement may affect strength of these tissues.[315]

Ligaments retain 80-90% of their baseline mechanical properties with minimal stimulation. The effects of exercise on ligaments and tendons are less profound than the effects of immobilization. In a healthy patient, ligament exercise is not likely to increase the strength and stiffness by more than about 10-15%. Sprint training results in increases in ligament weight but not ligament-bone junction strength.[315] However, endurance activity with regular mechanical loads results in increases in junction strength and ligament stiffness. Low-duration (30 minutes) and high-frequency (6 days/week) endurance exercise appears to have the most benefit to ligament strength. A program of low-intensity exercise combined with short bouts of high-intensity exercise in young horses resulted in thicker and stronger superficial digital flexor tendons as compared with horses that were stall rested or stall rested with a single daily bout of high-intensity exercise.[316]

The effects of exercise on morphologic characteristics and mechanical properties of the canine stifle medial collateral ligament have been investigated.[277,317] Dogs undergoing treadmill exercise for 6 weeks had a 12% increase in failure load of the medial collateral ligament, and a 15% increase in hydroxyproline content (indicative of collagen content) of the lateral collateral ligament compared with control dogs. In addition, collagen fiber diameter was greater following training, although GAG content, water content, collagen concentration/dry weight or collagen concentration per weight/length unit were unchanged.

Another study evaluated the effect of exercise on the structural properties of swine FMT complex. Animals were trained on a treadmill daily for 12 months. Exercised pigs had a significant increase in the ultimate load to failure of their ligaments when normalized for body weight, but there were no significant changes in deformation, or energy absorbed of ligaments. Results found in young rabbits that were trained on running wheels indicated that training increased the breaking strength of many ligaments and tendons, including the tibia-CCL-femur complex.[317] Studies in rats and mice have shown similar results and have suggested a possible additive role of testosterone in enhancing ligament and tendon strength with training.[58]

Studies in other species have not always demonstrated a corresponding increase in breaking strength, fiber diameter, and collagen content, however. For example, there was no increase in hydroxyproline content despite an increase in breaking strength in rats undergoing training.[58] In addition, there was an increase in collagen turnover with training. More of the newly formed collagen may be in the soluble form because of fewer cross-links. Metabolic changes have been noted in tendons and ligaments of rodents undergoing training, including increased tendon nitrogen content; malate dehydrogenase; reduced nicotinamide adenine dinucleotide oxidase; lactate dehydrogenase; isocitrate dehydrogenase; and succinate dehydrogenase early in the training period.[58] The water content does not change with training, but the number of cell nuclei per unit volume of tendon are greater in trained rats. Training results in an increase in oxygen consumption of less than 10% in ligaments and tendons, although there is a 58% elevation in muscle tissue. It is unclear why chronic exercise does not produce a greater effect.[284] Changes in the content of an elastic extracellular matrix protein, tenascin-C, have also been noted with immobilization and remobilization of ligaments and tendons.[318] Although rat tenascin-C was present at the normal rat bone-patellar tendon junction, it was almost completely absent after 3 weeks of cast immobilization. Remobilization in the form of cage activity for 8 weeks resulted in some increase, but not to normal levels. Tenascin-C activity around the chondrocytes and fibroblasts of the bone-tendon junction and the collagen fibers of the tendon belly was normal following remobilization with 8 weeks of treadmill training, however.

Stretching is frequently recommended in dogs in training or during rehabilitation to help improve joint ROM. In a study of rat tail tendons, stretching to 108% of the original length resulted in return to 104% of the original length when the stretch was released.[319] A second stretch resulted in more stretch per unit load (less stiff). Histologic examination found evidence of damage to some strands of collagen. Despite the changes in stiffness, the maximum breaking load was not significantly altered after the second stretch.

Effects of Medications on Ligaments and Tendons

Medications are sometimes administered locally or systemically in the treatment of musculoskeletal conditions. In particular, corticosteroids may be administered intraarticularly and have effects on ligaments and tendons. The effect of intraarticular corticosteroid injections on the mechanical properties of anterior cruciate bone-ligament-bone units was determined in rhesus monkeys.[320] Alterations in ligament strength and elongation properties depended on corticosteroid dosage and time after injection. Fifteen weeks after three weekly injections of large doses of methylprednisolone acetate (6 mg/kg), there were significant decreases in the maximum failure load (20%), energy absorption prior to failure (11%), and stiffness (11%) of the ligament unit. In contrast, only minimal alterations occurred after 6 weeks suggesting effects on the ligaments was not immediately seen but was long lasting. A third group of animals received intraarticular injections (0.6 mg/kg) spaced 2 weeks apart. Although decreases occurred in maximum failure load (9%) and energy absorption (8%) by 15 weeks, the magnitude of changes was less. There was microscopic evidence of collagen fiber failure at multiple levels throughout the ligament. Although high and frequent doses of intraarticular corticosteroids produce the greatest alterations in ligament strength and function, even low doses given infrequently affected ligament properties. A single intraarticular injection or injections repeated at intervals of several months is recommended to minimize ligament damage. Furthermore, direct injection of corticosteroid into the ligament should be avoided because fibrocyte death and alterations in ligament strength and stiffness may occur for up to 1 year.[320]

Injection of corticosteroid inside a tendon has deleterious effects, with collagen necrosis and decreases in tensile strength occurring.[321] Peritendinous injections are generally safer, and with proper indications, there may be fewer complications. Systemic administration of corticosteroids also has fewer adverse effects on tendons than intratendinous injection. However, administration of very high doses (10 mg/kg) may result in reduced breaking strength of sutured canine tendons.[321] In rabbits receiving corticosteroids, bony insertion sites became osteoporotic and failure tended to occur by fracture.[322] Tissues with shorter turnover times, such as bone, tend to weaken faster than tissues with longer turnover times, such tendons and ligaments, when corticosteroids are administered. In fact, tendons may initially become slightly stiffer early in the course of steroid administration because fibroblast proliferation and collagen production are reduced, resulting in decreased insoluble collagen. Long-term steroid use eventually results in weakening of ligaments and tendons because of the continued inhibition of collagen synthesis.

Treatment with topical dimethyl sulfoxide (DMSO) has been recommended for the treatment of local inflammatory conditions, including injuries to tendons and muscles. However, topical treatment with DMSO of the Achilles tendons of mice indicated that the tensile strength of the tendon decreased in relation to the length of time the mice were treated.[323] The maximum decrease of 20% was seen 7 days after treatment began. These data suggest that vigorous muscle activity should be avoided during treatment with DMSO.

Fluoroquinolone antibiotics may contribute to tendinopathies. The incidence of tendinitis and tendon rupture in human patients treated with ciprofloxacin suggests that these antibiotics alter tendon fibroblast metabolism. There was a 67% decrease in cell proliferation compared with control cells, and a 36% to 48% decrease in collagen synthesis of canine Achilles tendon, paratenon, and shoulder capsule fibroblasts incubated with serum levels of ciprofloxacin in cell culture.[324] Ciprofloxacin also caused a 14% to 60% decrease in PG synthesis and significant increases in matrix-degrading proteolytic activity. These changes suggest that ciprofloxacin stimulates matrix-degrading protease activity from fibroblasts and inhibits fibroblast metabolism, which may contribute to the clinically described tendinopathies associated with ciprofloxacin therapy. In fact, there is a black box warning with the use of any fluoroquinolone antibiotics for possible tendinopathy, especially in patients older than 60 years.

Certain pharmacologic agents may positively affect ligaments and tendons. For example, treatment with bone morphogenetic protein-2 results in more extensive bone formation around tendons placed through bone tunnels with closer apposition of new bone to the tendon in treated limbs, resulting in higher tendon pull-out strength.[325] Bone morphogenetic protein can accelerate the healing process when a tendon graft is transplanted into a bone tunnel.

Bone

Normal Bone

Bone consists of organic and inorganic components. The organic component consists of collagen fibers embedded in a matrix of GAGs, among other components of ground substance. The inorganic component consists primarily of calcium and phosphorus in the form of hydroxyapatite crystals. These crystals are embedded within the organized collagen fibers. Bone provides the musculoskeletal tissues with compressive strength during weight bearing, and the organic component provides tensile strength.

Long bones have a compact portion of lamellar bone found in the outer cortex, and loosely woven trabecular bone found in the inner medullary bone. The inner medullary bone is typically more metabolically active because of the relatively large surface area, but it is also relatively

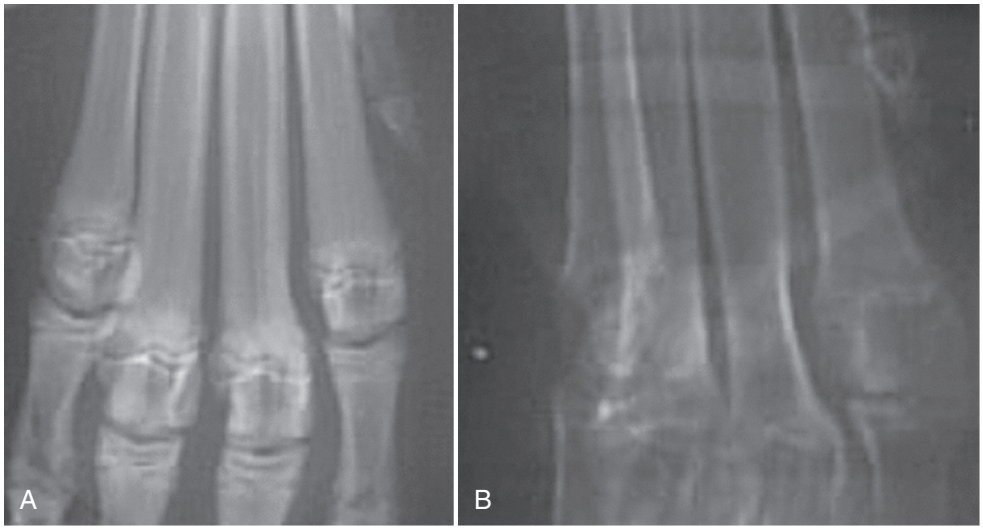


Figure 7-11 Radiographs of the metacarpal bones of a 10-month-old dog with fractures of the distal radius and ulna. **A,** At the time of fracture. **B,** Four weeks after cast placement. Note thinning of the cortices and generalized bone atrophy.

weaker as compared with the more dense outer cortical bone.

Effects of Immobilization on Bone

Many models exist to study the effects of immobilization on the skeleton, including local or systemic immobilization. Local immobilization involves casting, splinting, denervation, paralysis, disarticulation, and limb suspension, whereas systemic immobilization is accomplished with body casting, bed rest, or spaceflight. Reduced stress on bones may also result from stress shielding following application of implants for repair of fractures. Care must be taken in employing the various models. They have similar patterns of bone loss, but they respond slightly differently in the location and the amount of bone loss, depending on the degree of unloading.

Models of forelimb and HI that prevent weight bearing on the limb induce a reduction in cortical and cancellous bone mass, reduce cortical bone density and stiffness, and increase turnover in cancellous bone.[326,327] The changes that occur following immobilization vary depending on the length of immobilization and remobilization, age of the animal, and the bone involved. The effects appear to be more profound in younger dogs (Figure 7-11). In fact, immobilization during growth may result in permanent changes in bone mass and the relation of bone mineral content (BMC) to body weight.[328] Older animals may have less preexisting bone mass as a result of senile osteoporosis, and these dogs may lose less bone following immobilization than those with higher initial bone mass.[83,329] Trabecular bone is affected to a greater degree than cortical bone, and the effects of immobilization are more extensive in the more distal weight-bearing bones (Figures 7-12 to 7-14).[329,330] Diaphyseal bone loss during immobilization affects the cranial and caudal cortices to a greater extent than the medial and lateral cortices.[331] These changes are consistent with those seen in other species, including rats.[332]

Immobilization models may produce loss of 60% of the trabecular bone mass.[329] This level may suggest a physiologic threshold at which basal hormonal and cellular activity determined by genetics is reached in the absence of mechanical stimuli.[329] During the acute phase of immobilization, the greatest bone loss occurs in medullary bone from an increase in bone resorption and a decrease in formation. During the chronic phase, bone mass plateaus and cellular activities may revert back to normal levels.

The response to cast immobilization of bone in young adult dogs may be divided into three stages of histologic response.[333] In stage I, there is rapid initial loss of bone, reaching its maximum rate of loss at 6 weeks, with relatively equal contributions of the periosteal, Haversian, and endosteal regions to the loss. The rate of bone loss slows during stage II, which consists of 24 to 32 weeks of immobilization. The majority of the bone loss during this stage comes from the periosteal envelope. Stage III, characterized by greater than 32 weeks of immobilization, results in bone mass that has been reduced to 30% to 50% of original values. This pattern is qualitatively similar in all bones of the forelimb, but the distal bones are affected to a greater degree. Another study of immature dogs demonstrated a 55% decrease in bone mass of the distal tibial metaphysis following 4 weeks of unilateral hindlimb cast immobilization.[334]

The extent of resorption surface and the increase in the total histologically "active" periosteal envelope parallel the phases of bone loss. The type of metabolic bone activity also varies depending on the length of immobilization. In

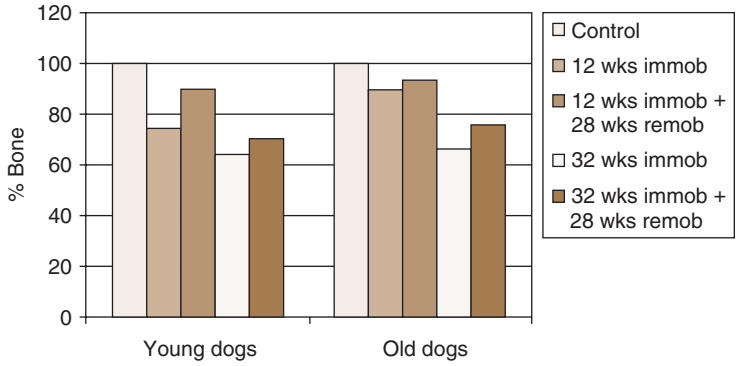


Figure 7-12 Trabecular bone volume of the radius following various periods of immobilization and remobilization. Note the relatively greater loss of bone volume and trabecular bone compared with the diaphyseal bone in Figure 7-13.

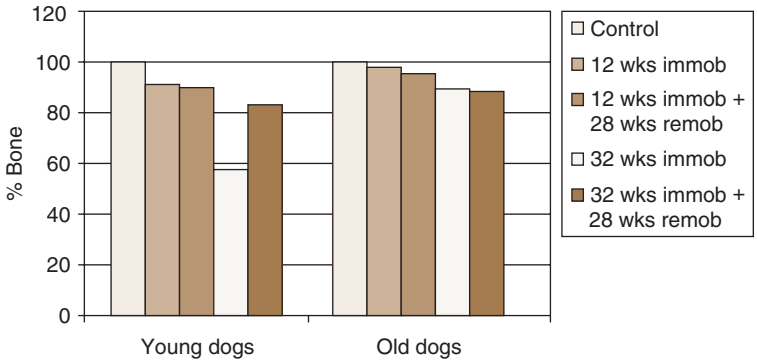


Figure 7-13 Bone volume of the diathesis of the radius after varying periods of immobilization and remobilization. Bone loss is greater in young dogs immobilized for long periods.

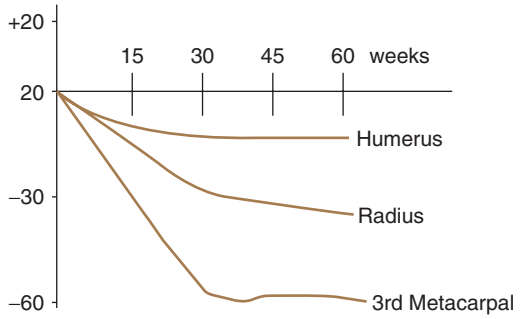


Figure 7-14 Effects of immobilization on density of various bones.

cast-immobilized young adult beagle dogs, the total active metacarpal bone surface (resorption and formation) was reduced 60% below control values during the first 2-4 weeks, increased 50% above controls at 6 weeks, and subsequently returned to control values at 8-12 weeks.[333] The response of bone to immobilization differs in mature dogs and is related to the length of immobilization.[83] In one study, bone volume was estimated in the third metacarpus, radius, ulna, and humerus at the mid-diaphysis and the distal metaphysis of dogs with forelimb immobilization for up to 32 weeks. In both young and mature dogs, bone volume decreased with the duration of immobilization and the types of changes were similar in the metaphysis and diaphysis. The old dogs, which began the study with 10% less bone than the younger dogs, showed smaller proportional losses than younger dogs, most evident in the diaphysis. In both age groups, the distal, weight-bearing bones tended to show greater losses. Following 32 weeks of immobilization, young dogs had 54% less bone volume in the third metacarpal diaphysis, whereas older dogs had 38% less. Metacarpal trabecular bone decreased 50% in young dogs and 47% in older dogs. Adult dogs had a 45% decrease in bone mass of the distal radial metaphysis following 16 weeks of cast immobilization in another study.[335]

The amount of bone loss associated with the length of immobilization apparently has limits. Although immobilization studies have demonstrated bone loss of up to 60% of the trabecular bone mass, there appears to be a maintenance bone turnover level that occurs with continued immobilization.[329] The remaining maintenance level suggests a physiologic threshold at which basal hormonal and cellular activity is reached in the absence of mechanical stimuli. During the acute phase of immobilization, bone loss occurs in bone adjacent to marrow, and from an increase in bone resorption and a decrease in bone formation.[330] There may be more erosion of the inner cortical surface in older dogs. Young growing dogs also have an interruption of periosteal bone deposition, resulting in smaller bone diameter. Immobilization also results in

increased regional blood flow and bone marrow pressure. The flow of the entire long bone is approximately four times greater than that of cortical bone, indicating that cancellous bone receives much of the blood flow. There is no significant difference in flow rate between the endosteal and periosteal cortices, however. There are variations of blood flow among long bones, which might be due to differences in their structure and function, and which may help to explain the differences in bone loss following immobilization.[336] Secondary factors, such as low dietary calcium or fluctuating hormone levels may also be involved. At steady state, or chronic phase, bone mass plateaus to a maintenance level with cellular activities returning back to normal levels.

Biomechanical properties of cortical and cancellous bone are also significantly affected by immobilization. Forelimbs of dogs immobilized for 16 weeks had decreases in cortical load, yield, and stiffness as well as cancellous bone failure stress, yield stress, and modulus, compared with control limbs.[82] In general, immobilized limb cancellous bone mechanical properties were 28-74% of control values, and cortical bone mechanical properties were 71-98% of control values. Similar to the differences in bone loss that occur in different bones, biomechanical properties of different bones in the same immobilized limb may also vary. Torsional stiffness of radiuses and metacarpal bones immobilized for 12 weeks decreased approximately 12% and 30%, respectively, as compared with controls.[329,337] Increasing the length of immobilization results in further deterioration of biomechanical properties. Metacarpal bone immobilized for 26 weeks retained only 50% of normal stiffness.

There is increased urinary and fecal excretion of calcium and phosphorus in people with cast immobilization of a limb, and similar findings may be expected in dogs.[58] Excretion of hydroxyproline, an indicator of collagen content, also increases with disuse. Relatively equal proportions of bone matrix and bone mineral are lost during bone atrophy, suggesting that the organic and mineral composition of bone is similar following a period of immobilization. Histologic examination reveals an increase in osteoclast activity and vascularization with disuse, suggesting an increase in bone resorption. Prostaglandin E (PGE) may also be involved in the development of immobilization osteoporosis.[334] Osteoporosis induced by fiberglass-cast immobilization of the hindlimb of immature dogs for 4 weeks resulted in significant loss of bone mass and a twofold increase in bone PGE, suggesting that PGE plays a role in immobilization osteoporosis.

Bone resorption also occurs at insertion sites of ligaments as a result of disuse, and there is decreased mechanical strength and bone collagen associated with disuse.[275] An internal skeletal fixation model applied for 12 weeks resulted in significant atrophy of bone and loss of collagen in the tibia and femur at the insertion and origin of the CCL.[29] When a fast rate of deformation was applied, failure of the femur-ligament-tibia complex occurred through the tibial insertion of the ligament. Bone atrophy was the result of increased resorption of bone rather than decreased bone formation. Preservation of active joint motion during immobilization may have a protective effect on the bone-ligament-bone complex.[30] A unilateral tarsal disarticulation model of disuse that maintained active knee motion without weight bearing for 8 weeks maintained the strength of the femur-CCL-tibia complex despite loss of collagen and calcium mass in the femur and tibia, suggesting that active joint motion helps to preserve the mechanical strength of the femur-ligament-tibia complex.

Bone loss may be reduced in paraplegic human patients who are ambulatory as compared with those who are nonambulatory, who develop osteoporosis of the pelvis and limbs.[58] A relatively low degree of physical training may inhibit the development of osteoporosis. Slow oscillation of bedridden human patients with intact neuromuscular function from a horizontal position to a 20 degrees foot-down position every 2 minutes may help preserve bone mass. The contraction of antigravity muscles during the downward tilt may be partially responsible for maintenance of bone mass by adding stress to bones during muscle contraction. There also appears to be an association of bone mass with muscle mass, with similar degrees of bone and muscle loss with disuse. Non–weight-bearing exercise of bedridden patients is relatively less effective in reducing calcium loss, but standing for 3 hours reduces urinary excretion, suggesting that weight bearing rather than physical activity is primarily responsible for reducing bone atrophy during bed rest.

In summary, young dogs tend to lose bone more quickly following immobilization as compared with older animals, even though older dogs may have less bone mass to begin with. The degree of bone loss increases with the length of immobilization, with the rate of loss slowing as the time of immobilization increases. Bone loss is greater in the more distal weight-bearing bones as compared with proximal minimally weight-bearing bones (Box 7-9).

Bone Changes Resulting from Disease Conditions

Bone changes may be detected after total hip replacement and complications of this procedure. In one study, dogs with unilateral primary cementless total hip arthroplasty had no right-left difference in tibial BMC or cortical bone cross-sectional geometry after 6 months, but after 2 years there was a 5-6% difference in BMC, perhaps because of subclinical disuse of the operated limb.[338] In dogs with failed cemented prostheses, tibial BMC may be more than 20% lower in the operated limb. Successful revision of the total hip replacement results in improved BMC, suggesting that improved limb function after revision surgery may result in gain of previously lost bone.

Bone loss may also occur following knee surgery as a result of relative disuse of the affected limb. Following

Box 7-9 Summary of the Effects of Disuse and Immobilization on Bone

- Models of limb immobilization decrease cortical and cancellous bone mass and cortical bone density and stiffness and increase turnover of cancellous bone.
- Limb immobilization results in decreases in cortical load, yield, and stiffness as well as cancellous bone failure stress, yield stress, and modulus.
- The changes that occur during immobilization vary depending on the length of immobilization and remobilization, age of the animal, and the bone involved.
  - Longer periods of immobilization result in greater bone loss.
    - Generally no further bone loss after 32 weeks of immobilization.
  - Effects are more pronounced in younger dogs.
    - Immobilization during growth may result in permanent reduction of bone mass.
  - Older animals may have less preexisting bone mass.
  - Trabecular bone is affected to a greater degree than cortical bone.
  - Effects of immobilization are more extensive in the more distal weight-bearing bones.
- Recovery of mechanical and morphologic properties of bone following immobilization depends on the length and type of immobilization, the type and intensity of remobilization, and the age of the animal.
  - Longer periods of immobilization result in longer recovery times.
    - Immobilization for 6 weeks requires 8 to 12 weeks for near full recovery of bone mass.
  - Immobilization for 24 to 28 weeks requires longer for near full recovery of bone mass.
  - With greater than 32 weeks of immobilization, bone mass remains at 30% to 50% of original values, despite remobilization.
  - Younger animals recover lost bone mass more quickly than older animals.
  - Training after immobilization results in more complete and rapid recovery of lost bone mass.
    - An initial period of mild treadmill training three times per week is recommended, because bone biomechanical properties may initially worsen during the initial remobilization period.
    - This can be followed by a period of higher intensity training.
    - Jump training may result in recovery of more normal trabecular bone mass.

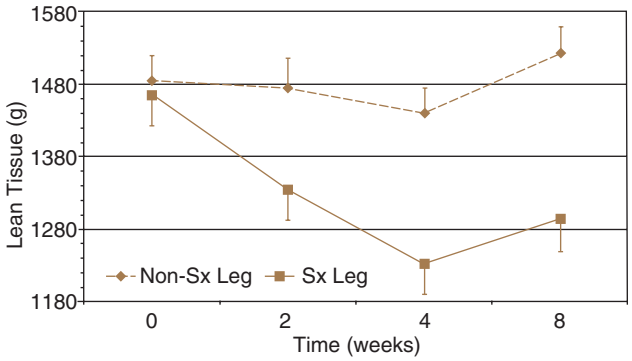


Figure 7-15 Changes in mean lean tissue content over time for the surgical and nonsurgical legs following transection of the cranial cruciate ligament and immediate stifle stabilization. (From Francis DA, Millis DL, Head LL: Bone and lean tissue changes following cranial cruciate ligament transaction and stifle stabilization, *J Am Anim Hosp Assoc* 42(2):127-35, 2006. ©From the Journal of the American Animal Hospital Association (jaaha.org) 2006. All rights reserved.)

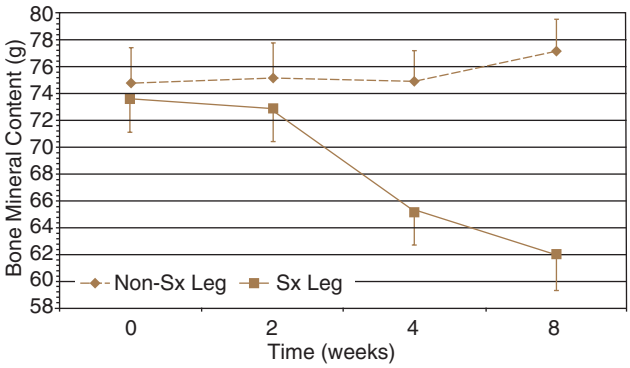


Figure 7-16 Changes in mean bone mineral content over time for the surgical and nonsurgical legs following transection of the cranial cruciate ligament and immediate stifle stabilization. (From Francis DA, Millis DL, Head LL: Bone and lean tissue changes following cranial cruciate ligament transaction and stifle stabilization, *J Am Anim Hosp Assoc* 42(2):127-35, 2006. ©From the Journal of the American Animal Hospital Association (jaaha.org) 2006. All rights reserved.)

CCL transection and extracapsular stabilization, DEXA was used to analyze BMC and lean tissue mass of the entire surgical and nonsurgical legs.[191] Regional BMC and BMD of the proximal, mid-, and distal tibia of the surgical and nonsurgical legs was also determined. There was significant loss of BMC and lean tissue in the entire surgical leg compared with the nonsurgical leg. Interestingly, the pattern of tissue loss was different. There was an immediate loss of lean tissue mass for the first 4 weeks, followed by slight recovery of lean tissue mass between 4 and 8 weeks (Figure 7-15). In contrast, there was little change in BMC for the first 2 weeks (Figure 7-16). This was followed by a rapid loss of bone mineral from 2 to 8 weeks, with no apparent recovery of bone mineral during that period. Regarding regional changes in bone, significant loss of BMC and BMD was detected in the tibia of the surgical leg and was most pronounced in the metaphyseal regions (Figure 7-17).

Another study of dogs with surgically created CCL transection demonstrated that there was a dramatic 22-34% decrease in BMD of the affected distal femur 12 weeks after surgery.[339] Studies of humans indicate similar decreases in BMD of approximately 20% in the distal femur, patella, and proximal tibia up to 1 year after surgery

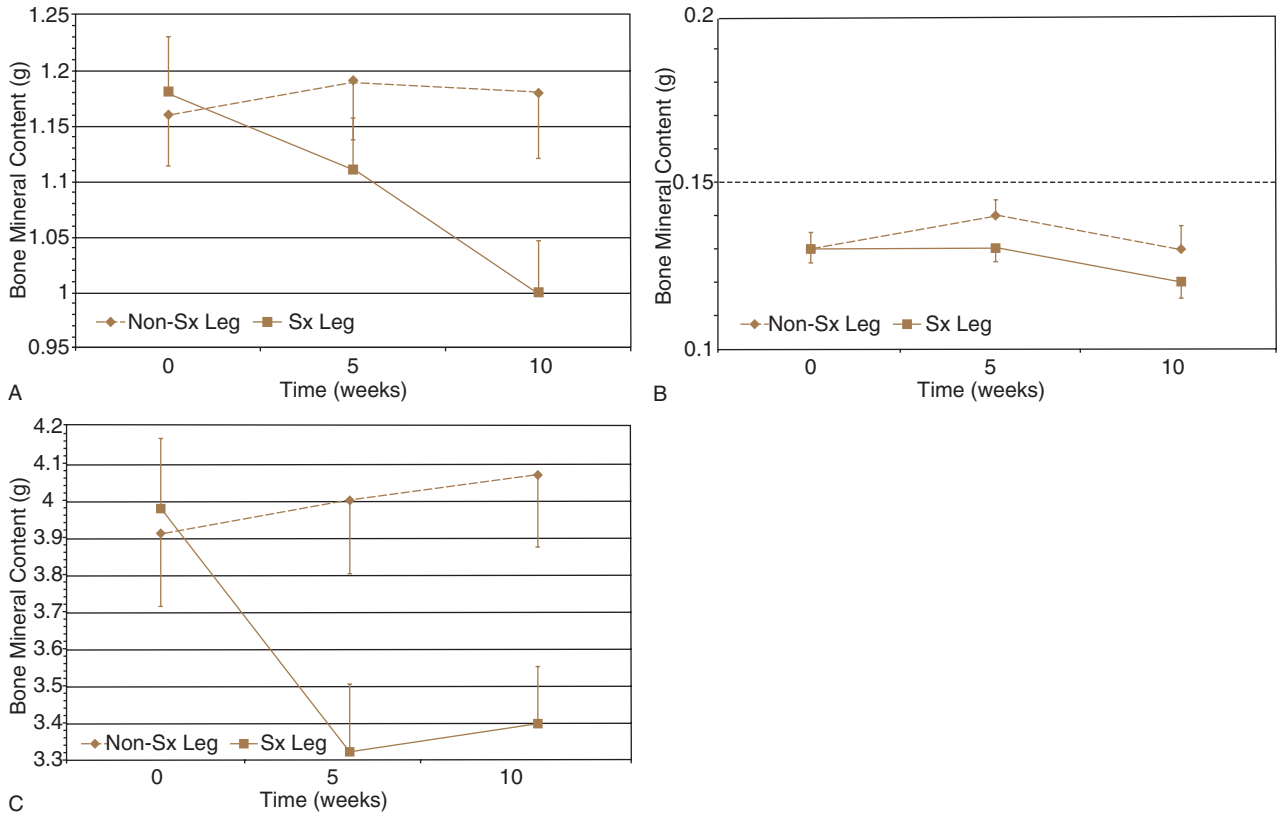


Figure 7-17 Changes in bone mineral content over time at the proximal tibial metaphysis (*A*), midtibial diaphysis (*B*), and distal tibial metaphysis (*C*) of the surgical and nonsurgical legs.

for ACL injury.[340,341] In addition, 10-11 years after surgery bone density may be decreased by 3% to 9%.[342] Bone density had significant correlation with functional scores of the knee in people, but not with knee stability.

In addition to bone changes following ligament injury, it is possible that there may be decreased bone density following fracture. In humans, 2% to 7% decreases in bone density were found in the distal femur, patella, proximal tibia, and calcaneus 10 years after femur fracture.[343] Relative bone density had correlation with pain and functional scores of the affected limb, but not with fracture type, fracture location, or non–weight-bearing time after injury. Similar decreases in bone density were seen 9 years after tibial fractures in another study.[344]

Denervation of a limb of a growing dog results in decreased lengths of the humerus and radius.[345] Furthermore, loss of innervation to a region may result in decreased bone density.[345] However, active muscle contraction, either through reflex walking or electrical stimulation, helps attenuate loss of bone density.[346] A functional neuromuscular stimulation–induced knee extension exercise system was used in human patients with spinal cord injuries up to three times per week for 36 sessions using a progressive resistance load.[347] Maintenance of bone density in some subjects suggests that training may retard the rate of bone loss that typically occurs with spinal cord injuries.

Stress Protection

Stress shielding or stress protection occurs when metal implants, such as bone plates and screws, are used to repair fractures or in joint replacement surgery. Although rigid metal plates stabilize the fracture site, help maintain contact between bone fragments, and allow early weight bearing and patient mobility, the higher stiffness of the implant results in bone loss as a result of decreased physiologic loading of the bone. Calculations were made of the alterations in cyclic bone stresses caused by the application of bone plates on canine femurs.[340] The magnitude of the reduction in the loads borne by the bone and the degree of shift in the bone stress neutral axis during the stance phase of gait was influenced by plate geometry, plate stiffness, and plate location. Bone remodeling is very sensitive to small changes in cyclic bone stresses. Changes in cyclic bone stresses of less than 1% of the ultimate strength can cause measurable differences in bone remodeling after a period of a few months.

The pattern of cortical atrophy induced by plate fixation for 7 months on canine femurs was evaluated in another study.[348] A pronounced reduction of cortical bone (approximately 15% less cortical area) was observed in the diaphysis of the plated bone at the end of the study period. The loss of cortical bone was mainly caused by endosteal resorption with enlargement of the medullary cavity.

Neither periosteal resorption nor formation of woven bone under the site of the plate was observed. Osteoporosis caused by rigid plate fixation may occur by thinning of the cortex rather than by reduction of the mechanical properties of the osseous tissue.[349] In addition to enlargement of the medullary canal, there may also be decreased cortical thickness under the plate as a result of endosteal resorption. The stiffer the implant and the longer the implant is in place, the greater the bone loss and the lower the recovery potential of the bone. For example, 6 weeks after screw application, some resorption of bone is evident and by 14 weeks, more significant resorption occurs.

Implants may also interfere with blood circulation, and the weakened bone can refracture after plate removal.[350-352] Disturbance of blood supply may be the main cause of osteopenia in the early stage, whereas disuse of the affected limb may be the most important factor in later stages of fracture healing. Osteopenia may be treated, in part, by active exercises and normal weight bearing before plate removal.[352]

Stress shielding may also occur following total hip arthroplasty.[353] Stiffness of the femoral stem is a design variable that is partially responsible for femoral remodeling. The basic pattern of bone remodeling is characterized by proximal cortical atrophy, and distal cortical and medullary bone hypertrophy. Low-stiffness stems may alter this pattern, leading to reduced proximal bone loss, increased proximal medullary bone hypertrophy, and no distal cortical hypertrophy, suggesting that stem stiffness has a profound effect on stress shielding.

Effects of Remobilization on Bone

The recovery of mechanical and morphologic properties of bone following a period of immobilization depends on the length and type of immobilization, the type and intensity of remobilization, and the age of the animal.[354]

The potential for recovery of bone lost during disuse, both in the diaphyseal cortical and metaphyseal cancellous bone, was evaluated in young adult and old beagle dogs.[83] Following immobilization of a forelimb for up to 32 weeks, there was considerable recovery of the original bone loss during remobilization. In both age groups, the residual deficits increased with the duration of immobilization and were similar in the metaphysis and in the diaphysis. In addition, the distal, weight-bearing bones tended to show greater losses and also greater recovery both in diaphyseal and metaphyseal bone. The older dogs had greater residual deficits, most evident in the diaphysis. Following 32 weeks of immobilization and 28 weeks of remobilization, a 50% loss in the third metacarpal diaphysis of younger dogs immediately following the immobilization period decreased to 15% (a 70% recovery), whereas older dogs had a 38% loss that decreased to 23% (a 40% recovery). In contrast, immobilization for 6 or 12 weeks resulted in complete recovery of bone after remobilization of 10 or 28 weeks, respectively. In fact, bone volume of third metacarpal trabecular bone exceeded the original control values in some dogs.

The stages of bone loss that young adult dogs undergo in response to cast immobilization have been discussed.[333] The response to remobilization may be similarly divided into stages. In stage I, in which there is a rapid initial loss of bone following 6 weeks of immobilization, there is near full recovery of bone mass within 8 to 12 weeks of cast removal. Radiographic evidence of periosteal stress reactions of the metacarpal bones, indicated by external bone formation in the distal metacarpal metaphysis, was present in 60% of younger dogs, but none of the older dogs following 12 weeks of immobilization and up to 28 weeks of remobilization.[331] In stage II, bone loss slows, but recovery is also slower following 24 to 32 weeks of immobilization. During the remobilization period, young and old dogs immobilized for 32 weeks may have periosteal stress reactions of the metacarpal bones, indicated by external bone formation in the distal metacarpal metaphysis.[331] Although there was no evidence of a break in bone continuity, microdamage (mechanical fatigue) may have occurred at these sites, and periosteal stress-induced reactions may occur at sites of increased stress in the absence of actual fractures. Continued stress to bones during the remobilization period may indeed lead to stress reactions. Immobilization for longer than 32 weeks characterizes stage III, in which the loss of bone mass remains at 30% to 50% of original values, despite remobilization.

Complete recovery of trabecular bone following immobilization may be limited by changes in bony architecture.[354] Following 18 weeks of immobilization, trabecular area of rat tibias increased by 42% and 51% following 10 and 20 weeks of remobilization. Remobilization resulted in increased bone area by decreasing porosity during the first 10 weeks of remobilization, but little further increase occurred between 10 and 20 weeks of remobilization. The recovery of bone loss was mainly the result of an increase in trabecular width. In fact, there was no recovery of the number of trabeculae lost during immobilization. Remobilization added bone on existing surfaces, but was not anabolic enough to reconnect trabeculae. This may explain why there may be a persistent loss of bone following long periods of immobilization, despite a long remobilization period.

Although it is clear that a simple period of free exercise during the remobilization period may not result in complete recovery of bone following immobilization, high-intensity training following immobilization may result in more complete recovery. One study of rats undergoing limb immobilization and subsequent remobilization indicated that high-intensity treadmill running resulted in restored BMC and BMD of the affected limb, whereas animals undergoing free activity had BMC and BMD

values less than those of controls.[355,356] The expression of osteocalcin, a marker of osteoblastic activity, corresponded to bone formation during the remobilization period.[357] When the training period ceased, however, the beneficial effects of training were lost. It appears that intensified remobilization is necessary to restore bone mineral after disuse, but the benefits may be lost if activity is terminated.

A period of mild treadmill activity following free remobilization may also be beneficial for dogs with immobilized limbs. Forelimb immobilization of 1- to 2-year-old dogs for 16 weeks, followed by a remobilization period of 16 weeks of kennel confinement and 16 weeks of treadmill exercise administered three times per week, resulted in the return of cortical and cancellous BMD and mechanical properties to essentially normal levels.[82]

Other activities may also be beneficial after a period of immobilization. The effects of jump exercise during remobilization were examined in rats undergoing immobilization by tail suspension.[358] Comparisons were made with nonsuspended controls, suspended controls, and suspended controls with spontaneous recovery. The jump exercise protocol consisted of 10 jumps/day, 5 days/week, and the jump height was 40 cm. Tail suspension induced a 13.6% decrease in total femoral BMD and marked deterioration of trabecular architecture. After 5 weeks of free remobilization, femoral BMD, calf muscle weight, and body weight returned to age-matched control levels, but trabeculae remained thinner and less connected. On the other hand, rats undergoing jump training after immobilization showed significant increases in trabecular thickness, number, and connectivity compared with suspended rats with spontaneous recovery.

Despite the positive effects of training on bone recovery after a period of immobilization, some caution is necessary when instituting a training program. One experiment evaluated the effects of immobilization and remobilization on mechanical properties of femoral shafts of rats.[359] Rats had the right hindlimb immobilized for 3 weeks by taping and were compared with controls. Immobilization was followed by 4 weeks of free remobilization. Immobilization followed by remobilization resulted in lower maximum bending moment, yield moment, stiffness, and work to failure compared with controls. Remobilization resulted in worsening of properties of femora. The results suggested that the strength of bone decreases during the first period of free remobilization and may be associated with increasing brittleness of cortical bone. Similar findings have been obtained in another study of rats.[360]

Adaptation of Bone to Increased Stress

The bone response to mechanical loading is proportional to an applied load of given magnitude and frequency of application. There are also proportional increases in second messengers, growth factors, bone matrix, and bone strength with loading.[361] Animal models using dogs, turkeys, mice, rats, chickens, and pigs have demonstrated a threshold-response and dose-response relationship to mechanical loading of bone, either by mechanical means or exercise. In general, relatively low-intensity training may have no or only mild effects on bone density or bone growth, whereas higher-intensity training results in greater bone density, but may inhibit growth in bone length and girth in growing animals.[58]

Despite much knowledge regarding the influence of physical activity on bone, precise information regarding the type, intensity, frequency, and duration of exercise on bone remains elusive. The remodeling of bone is slow, taking several months to complete. The age at which training begins is important. Initiation of activity early in life appears to be the most beneficial.[362] Within limits, loading of growing bone leads to a better result than loading of adult bone. The production of prostaglandins and growth factors may modulate the process of bone modeling. Although the optimal pattern of bone loading is unknown, evidence suggests that bone should be loaded with high peak forces and strain rates (high impacts), create strain throughout the bony column, consist of relatively few repetitions, and be long term and progressive. In humans, weightlifters have the greatest bone mass, followed by throwers, runners, soccer players, and swimmers. This suggests that weight and strength training appears to provide the greatest effects on bone, and swimming the least, perhaps because the buoyancy of water alters the load placed on bone as compared with weight lifting on land.

Resistance exercise training may also affect bone of animals. Rats conditioned to press levers, facilitating full extension and full flexion of the hindlimbs while wearing a weighted vest, had an increase in cancellous bone area of the proximal tibia after 6 weeks.[363] In addition, osteocalcin, trabecular number, thickness, and osteoid covering cancellous bone were greater in trained animals, suggesting that resistance training increases cancellous bone area by stimulating bone formation. A study of rats undergoing exercise with or without sudden impact loading of the femur indicated that cortical wall thickness increased with any activity, and breaking strength of the affected bones was greater in bones undergoing impact loading as compared with sedentary controls.[364] In spite of the enhanced biomechanical properties of impact-loaded bones, there was little difference in BMC in the exercised groups, suggesting that significant improvements may occur in biomechanical properties with little change in bone mass with impact loading.

In dogs, mild increases in loading of bones have little effect on bone mass or bone density. Impact loading or chronic moderate increases in loading result in increased bone mass and bone density. A study of mature beagle dogs with an ostectomy of a radius showed that there was little change in bone mass and density of the contralateral

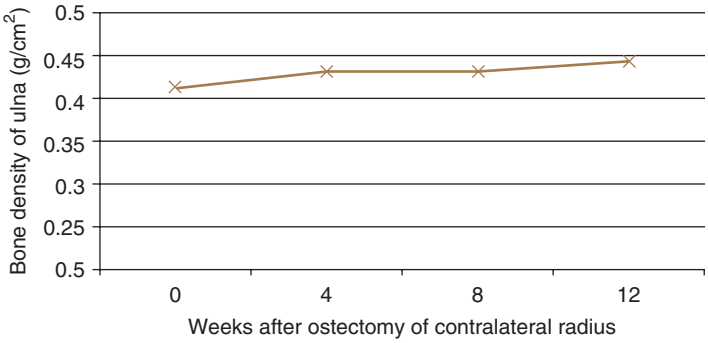


Figure 7-18 Change in bone density of the contralateral ulna following ostectomy of a radius. Compared with the situation in Figure 7-19, there is little change in bone density because both the intact radius and ulna continue to share loading.

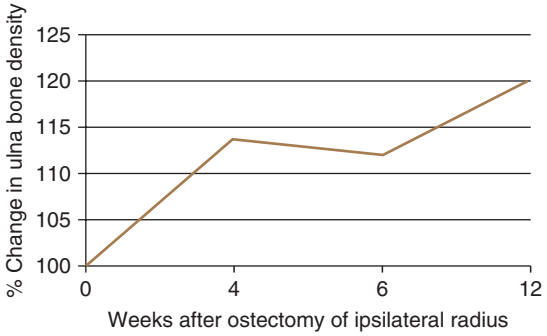


Figure 7-19 Relative change of the bone density of the ulna following ostectomy of the ipsilateral radius. Note the increased density as a result of the shift of weight bearing from a paired two-bone system to the single remaining bone.

radius and ulna (Figure 7-18).[365] This model allows weight bearing on the limb undergoing ostectomy of the radius because the intact ulna allows for weight bearing. However, because all of the weight is borne by the intact ulna following ostectomy, the load on that bone is significantly greater as compared with the load-sharing seen with the intact radius and ulna of the contralateral limb. The increased load results in significant increases in bone mass and density in the ipsilateral ulna within a relatively short period (Figure 7-19).

Increasing weight bearing on bones by exercising dogs with lead-weighted jackets results in increased bone mass.[366] Increasing the load carried to 130% of body weight and exercising on a treadmill for 75 minutes at 3.3 km per hour during a 70-week period resulted in a mild increase in BMC of the tibia when adjusted for actual body weight. This form of activity may increase bone mass and help reduce bone loss during aging.

The effects of long distance running training on bone growth have been studied in young, growing dogs.[224] Young beagle dogs trained on a treadmill for 1 year, gradually increasing running to 40 km/day at a speed of 5.5-6.8 km/hour with a 15-degree inclination during the last 15 weeks, while control dogs were kept in their cages. Ulna and radius bone mass as a ratio to body mass increased approximately 10% in trained dogs as compared with controls, although there was no change in mass of cranial tibial and semitendinosus muscles. Long-distance running apparently has a mild positive effect on bone mass in growing dogs.

In contrast, another study of immature dogs with a similar training regimen indicated that, although intensive endurance training may increase bone mass of weight-bearing bones, the appendicular skeleton may have reduced bone density.[367] Trained dogs had larger and heavier radius, ulna, and hip bones, but reduced BMD of thoracic vertebrae as compared with untrained controls. In addition, trained dogs developed more osteophytes in the spines but not in the extremities. Intense aerobic training of young dogs may alter bone mass, possibly because of altered biomechanical forces placed on the bones.

High-level activity, such as racing, may affect bone to a greater extent as compared with bone changes occurring with endurance training. One study of racing Greyhounds found that the cross-sectional and cortical areas of left metacarpals IV and V were greater than the right metacarpals.[368] In addition, osteonal density was greater on the dorsal surface of the bones than the medial and lateral quadrants. These changes indicate site-specific adaptations to the bones as a result of asymmetric loading from running in a counterclockwise fashion and greater loading of the inside limb.

A lifetime of weight-bearing physical exercise may decrease the amount of bone lost through the normal aging process in dogs.[369] Dogs in a trained group exercised for 90 minutes, five days/week, for 527 weeks, running an average of 3.3 km/hr on a treadmill while carrying approximately 11.5 kg. The control group had normal cage activity in dog runs. Long-term exercise significantly reduced cortical porosity and marginally increased cortical area in aged dogs, because of a decrease in bone resorption rather than an increase in bone formation. The results of this study have implications for the role of exercise in delaying bone loss during aging. Training appears to alter mineral uptake and retention by bone. For example, in one study of geriatric dogs exercising 40 min/d at 2.4 km/hr on a 15-degree grade, dogs had less calcium excretion on exercise days.[370] Similar studies of humans support these

findings and also indicate that bone deposition rate is increased with training.[56]

Although mechanical stimulation is important in promoting bone formation in normal dogs, fracture healing may also be enhanced with weight bearing. Dogs with surgically created defects in the tibial cortex that were allowed to bear weight on the limb had significantly more woven bone in the defects as compared with dogs that were not permitted to bear weight.[371] This suggests that weight bearing is a permissive factor for bone formation in bony defects. In addition, training may enhance healing if a fracture should occur after initiating training. A study of mice undergoing training prior to fracture had higher rates of collagen synthesis and higher concentrations of calcium in the healing fracture callus as compared with sedentary rats.[372]

Although exercise is generally thought to have favorable effects on bone health, it has also been suggested that exercise may reduce fracture risk.[373] Cross-sectional studies and interventional studies using assessments such as BMD and falls support this belief. For rehabilitation programs to improve bone health, they should include not just weight-supported activities (e.g., cycling) but also weight-bearing activities (e.g., walking, resistance exercise).[373]

Effects of Medications on Bone

A number of studies have evaluated the effects of various compounds on bone mass in dogs. Some of these substances are osteoprotective, whereas others, such as corticosteroids, may hasten bone loss in immobilized limbs.

Some medications and hormones help protect against bone loss during immobilization. Dogs receiving 60 to 240 ng/kg of vitamin D_3 for 4 weeks had trabecular bone volume of the calcaneus in direct relation to the dosage of vitamin D_3 in a plaster cast disuse osteoporosis model for 4 weeks, with the highest dose resulting in bone volume equal to the control limb.[374] Unfortunately, this dose is considered to be toxic. Vitamin D_3 administered to beagle dogs after ovariectomy was effective in maintaining normal urinary hydroxyproline excretion, suggesting that the treatment was effective in helping prevent the bone loss associated with ovariectomy.[375] In fact, BMD of the lumbar vertebrae and femur of ovariectomized dogs receiving 2 or 10 mcg/kg of vitamin D_3 were the same as non-ovariectomized control animals 30 months after surgery, whereas density was 25% lower in untreated ovariectomized dogs.

Bone loss during disuse has been prevented with bisphosphonate compounds in humans following paralysis or bed rest. Using a forelimb immobilization model in dogs, pamidronate, a bisphosphonate compound, was administered for 7 days followed by 3 weeks without treatment. This cycle was repeated three times for a total of 12 weeks. This treatment was effective in preventing cancellous bone loss and maintaining cortical bone density and stiffness.[326] In addition, indices of bone formation and resorption were significantly reduced.

In another study, risedronate, also a potent inhibitor of osteoclastic activity, was evaluated to determine if it could effectively prevent cancellous bone loss in middle-aged female beagles subjected to single forelimb immobilization for 12 months.[376] Age-matched, nonimmobilized dogs served as controls. Half the animals from each group received risedronate. Immobilization caused a dramatic reduction in cancellous bone mass (−71%) of distal second metacarpals, characterized by decreases in trabecular width (−51%) and number (−41%), and fourfold increases in indices of bone resorption. Bone formation indices were also significantly higher in immobilized dogs. Risedronate treatment reduced, but did not abolish, cancellous bone loss caused by immobilization. Immobilized animals treated with risedronate lost nearly 50% of cancellous bone mass, whereas trabecular width and number were reduced by 31% and 25%, respectively. Also, overall bone formation parameters remained roughly at intact control levels; however, mineral apposition rate relative to intact control was reduced in treated animals, indicating that high-dose risedronate treatment might suppress osteoblastic function, especially with long-term disuse. Risedronate treatment was partially effective in preventing cancellous bone loss during long-term disuse. Although bisphosphonates can impair the ability of mature osteoclasts to resorb bone, they do not completely overcome osteoclast recruitment caused by long-term disuse.

There may be differences between the effectiveness of bisphosphonates on osteoporosis resulting from disuse versus other forms of osteoporosis. One study evaluated whether risedronate can preserve cortical bone mass and mechanical properties during disuse for 12 months on metacarpal diaphyses in dogs.[377] Risedronate slowed cortical thinning and partially preserved mechanical properties, but it was unable to suppress bone loss to the degree seen in other osteoporoses. Immobilization caused marked reduction in cortical area (−42%) and cortical thinning (−40%). Both bone resorption and formation were significantly elevated over control levels. Immobilization also decreased maximum load and stiffness by approximately 80% compared with controls. Risedronate reduced both periosteal bone loss and marrow cavity expansion, and also reduced cortical bone loss, partially conserving tissue mechanical properties. This modest effect contrasts with more dramatic actions of the bisphosphonate in other osteoporoses.

Bisphosphonate compounds also affect bone resorption in other species. A study evaluated tiludronate, a bisphosphonate, on biochemical biomarkers of bone metabolism and on bone density and structure in a model of disuse osteoporosis induced by cast application in horses.[378] Horses were immobilized for 8 weeks, followed by a 4-week remobilization period and an 8-week standardized

training protocol. A rapid decrease was seen in type I collagen cross-links, an indicator of bone resorption, in horses receiving tiludronate. No significant differences between groups were seen in bone alkaline phosphatase, a marker of bone formation. The loss of BMD in the immobilized metacarpal III bone was significantly less in the tiludronate group as compared with immobilized placebo controls. Therefore this bisphosphonate significantly reduced bone resorption during immobilization, and helped prevent osteopenia in the immobilized limb.

Tamoxifen citrate administered to growing dogs with a hindlimb placed in a fiberglass cast for 28 days was moderately effective in sparing bone mass as compared with immobilized controls.[327] The calculated bone mass sparing effect of tamoxifen was 24.4%. This drug is a nonsteroidal antiestrogen compound, with some ability to inhibit prostaglandin synthesis, modulate signal transduction processes that control cell growth, and induce TGF-β.

Aspirin may also be effective in diminishing bone loss during immobilization. Osteoporosis was induced in growing dogs using a cast immobilization model for 4 weeks.[334] Bone PGE concentrations doubled and bone mass was reduced in untreated immobilized dogs. Dogs receiving 25 mg/kg of buffered aspirin every 8 hours had a 13% bone mass sparing effect and a 65% reduction in bone PGE. Similar bone-sparing effects have been observed with other NSAIDs, such as ibuprofen, in the treatment of osteolytic conditions such as osteomyelitis and bone metastases.

Bone morphogenetic proteins have also been used to help manage bone atrophy as a result of fracture. In one case report, recombinant human BMP-2 solution on a collagen sponge was placed along the diaphysis of an atrophic radius, which had a history of recurring fractures.[379] Two months after rhBMP-2 treatment, new mineralized bone was present, which significantly increased the diameter of the radius and allowed the removal of the ESF.

Rehabilitation Considerations

The results of these studies are useful to help structure rehabilitation programs for musculoskeletal injuries. The injury must be protected from further damage in the early stages of healing. Acceleration of the remodeling process of healing tissues should be promoted by applying adequate stress and strain to the tissues in appropriate stages. The deleterious effects on biomechanical properties of tissues suggests that immobilization should be avoided or minimized whenever possible. Joints and limbs should be mobilized early, while simultaneously protecting the weakened tissue during healing, especially considering the length of time needed to achieve near-normal strength of tissues. Although certain tissues, such as the ligament substance, may recover rapidly following remobilization, other tissues, such as the bone-ligament complex, may take as long as 1 year to return to normal. Early joint mobilization and limb usage must be tempered with the degree of injury and the repair performed, if any. The healing tissues must be protected from excessive force, and a progressive rehabilitation program should be used that applies low cyclic loads to promote healing and enhance remodeling. Progressive increases in the frequency and magnitude of loads applied to the healing tissues enhance healing and tissue strength. Although little is known regarding the optimal stress to apply to tissues to obtain an optimal response, the goal of rehabilitation following injury is to enhance the definitive quantity and quality of repair tissue.

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8 Basics of Exercise Physiology

Robert Gillette and R. Barry Dale

A good basic knowledge of exercise physiology is necessary when designing and implementing a rehabilitation or conditioning program. The definition of *training,* for the purposes of this chapter, refers to working with the dog in an obedience training program that addresses behavioral aspects, whereas *conditioning* refers to an exercise or workout program that addresses the animal's physiologic capacity to perform work. *Exercise physiology* is a discipline that examines how exercise affects the body and is applicable in the field of physical rehabilitation.

The level of conditioning of a dog constantly changes any time the body performs at a specific metabolic level proportionate to its current adaptation to physical stress (Figure 8-1). The systems of the body are conditioned to maintain homeostasis at this level. If the demands placed on the body increase over time, the body adapts and conditions itself to maintain homeostasis at this new level. An understanding of the physiologic changes involved in this process helps in the development of programs that allow regulation of the conditioning or reconditioning of the body. These programs may be used to condition the entire body or to focus on individual body segments. In physical rehabilitation, programs may be developed to treat injuries, to stimulate healing of the injuries, and to enhance reconditioning of the injured segment. Because of the biomechanical forces acting on the body during the reparative phases of healing, certain segments may become stronger than other segments. Exercise programs should be implemented to remedy any conditioning imbalances of the body. The basic goals should be to allow the tissues to heal, then to recondition the tissues so that they can accept predetermined workloads, and finally to recondition the entire body to balance any conditioning deficits.

Cardiopulmonary Physiology

The circulatory system carries various oxygen, substrates, and chemical messengers; removes carbon dioxide and metabolic byproducts; assists with thermoregulation; and is an important mediator for acid-base balance.[1]

Blood consists of fluid and cellular components. Blood plasma is the liquid portion of blood and consists of water, proteins, electrolytes, dissolved gases, and nutrients.[1] The cellular constituents consist of erythrocytes, leukocytes, and platelets. Oxygen and carbon dioxide (discussed in the next section) dissolve to a limited extent in blood plasma, but are largely carried bound to hemoglobin found in erythrocytes.

The heart serves to pump blood to the pulmonary and peripheral circulation. Resting heart rate is approximately 80 beats per minute for a large dog.[1] In response to exercise, heart rate and myocardial contractility increase, which concomitantly increases cardiac output. A large dog typically has increases of 30% for stroke volume, 300% for heart rate, and approximately 400% for cardiac output during vigorous exercise when compared with rest.[1]

Muscle Physiology

Muscle is a tissue greatly affected by exercise or disuse associated with injury. Understanding muscle function and the molecular events of muscle contraction provides a basis for the concepts of physical rehabilitation and exercise physiology. There are three types of muscle in the body: skeletal, smooth, and cardiac. Skeletal muscles connect one bone to another. Each muscle consists of thousands of myocytes. A muscle fiber is long, fusiform, and surrounded by a plasma membrane called the sarcolemma. Inside these fibers are myofibrils, which consist of filaments composed of contractile proteins. These contractile proteins are arranged in units called *sarcomeres.*

Actin and myosin are two types of protein chains in the sarcomere. They interact as a result of enzymatic and chemical reactions to produce muscle contraction. Calcium and phosphate are chemical components involved with the production of muscle contractions. Phosphate is in the form of adenosine triphosphate (ATP). ATP binds to a receptor site at the end of the myosin leverage arm. The actin filament includes troponin, which is bound to strands of tropomyosin. A calcium ion attaches to the troponin molecule, which changes the shape of the tropomyosin. This action opens a myosin-specific binding site on the actin protein chain. Energy is emitted when ATP releases a phosphate ion, producing adenosine diphosphate (ADP). The resultant energy allows the ADP to create a bond between the open binding site on the actin filament and the myosin leverage arm. This bond changes the myosin

structure, allowing leverage to produce a contraction between the two fibers. The ADP is released and the lever arm is freed. Energy is then required to add a phosphate group to the ADP, re-creating ATP, which is then used for further contractions (Figure 8-2). The accumulated contractions of the muscle fibers create contraction of an entire muscle.

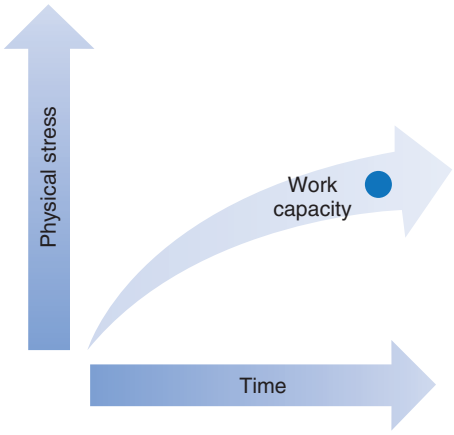


Figure 8-1 Work capacity increases when physical stress increases and adequate time allows for adaptation.

Muscle Structure and Function

The innervation to a muscle controls the muscle contraction by directing an action potential along an efferent motor neuron. A single motor nerve diverges to innervate many muscle fibers. The combination of a nerve and the muscle fiber it innervates is called a *motor unit.* Contraction of an entire muscle is a result of the cumulative contraction of many motor units.

The muscle fibers are grouped together and organized with other fibers by a sheath of connective tissue that is named according to its level of organization. Endomysium covers each of the muscle fibers themselves, and perimysium separates discrete bundles of fibers. Epimysium is the connective tissue layer that surrounds the grouped bundles. These three layers of connective tissue are longitudinally continuous throughout the muscle belly and blend together at each end with the proximal and distal tendons. Muscle fascia is the sheath that covers the epimysium and serves to protect each muscle from movement over hard structures or movement from adjacent muscles. The arrangement of these fibers plays a role in the function of each muscle. It is the combination of motor unit group, the characteristics of the muscle fibers, and fiber arrangement that dictates the resultant type of muscular function (strength, power, or endurance).

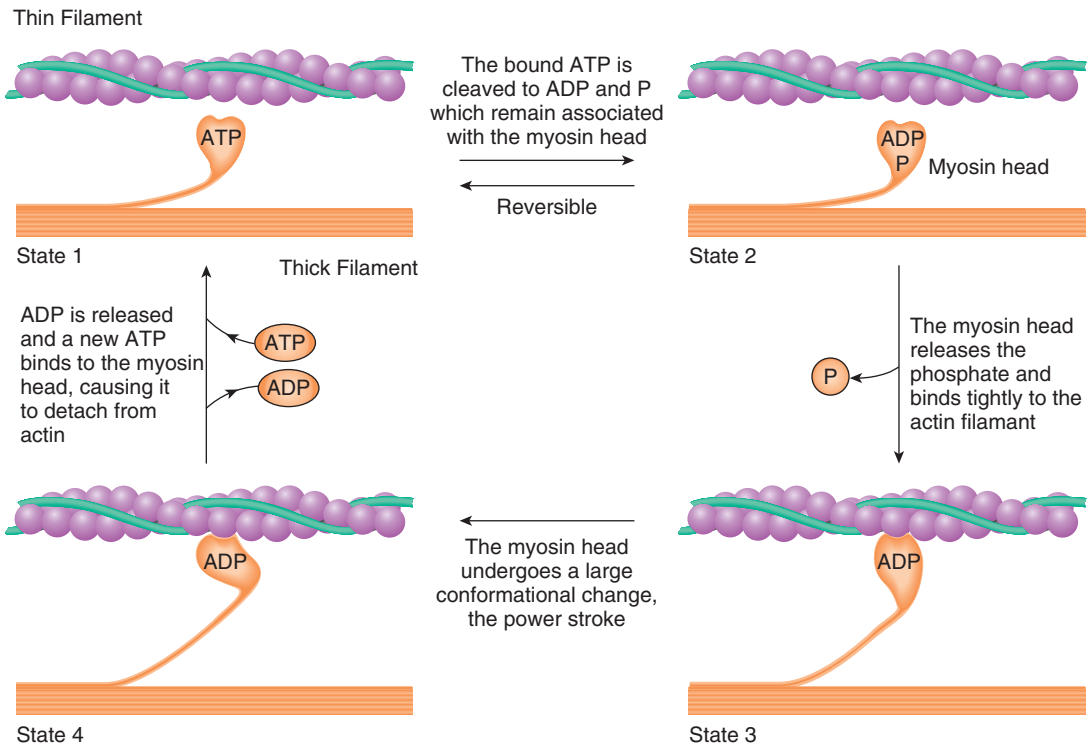


Figure 8-2 Actin and myosin interaction during contraction. *ADP,* Adenosine diphosphate; *ATP,* adenosine triphosphate; *P,* phosphate.

In general, muscle contraction and work are transferred through the tendon and its attachment to a bone. The musculotendinous junction is a layered transition between muscle fibers and the collagen of the tendons. Tendons of origin and insertion may run throughout the length of the structure. Musculotendinous structures are closely tied to functional requirements. Structural shapes of muscle include fusiform and pennate forms. Pennate muscles may be divided into unipennate, bipennate, and multipennate forms. Pennate structures allow a muscle to lift great loads but through a small range of motion, such as in vertebrae. Fusiform structures have the ability to lift a small load at a great velocity through a large range of motion. These types of muscles include the biceps brachii and brachialis muscles in the antebrachium. These two forms of muscle shapes can work together if both strength and speed of movement are needed in a particular joint, such as the shoulder or hip joint.

Muscle Energy Systems

Muscles require energy to maintain basal metabolism and additional energy during physical activity. The body uses three systems to provide this energy: (1) immediate energy sources, (2) glycolytic metabolism, and (3) oxidative metabolism. The type of activity the muscle is performing determines which of the systems will be used. The cellular environment must also be conducive to these processes. Factors affecting the cellular environment include pH, hydration status, temperature, and presence of the proper enzymes. Changes in any of these factors may alter the reactions needed for energy production. While energy production is occurring, metabolic byproducts are simultaneously produced. Byproduct removal is essential, because local accumulation of these substances alters the cellular environment.

The immediate energy source involves intracellular ATP, creatine phosphate (CP), and the ADP-myokinase reaction to provide energy for activity. Intracellular ATP is the first energy source used for contraction. A limited amount of ATP is stored at the myosin crossbridges, near mitochondria, and beneath sarcolemma. ATP breakdown is a hydrolytic reaction and is highly regulated. The body does not allow large changes in ATP content. CP is a high-energy compound used to ensure that the ATP concentration does not become depleted. It is located near the actin-myosin filaments and in the mitochondrial membrane. The CP molecule donates a phosphate group to ADP to replenish the ATP used at the contraction site. The third immediate energy source comes from the myokinase reaction. Myokinase is the enzyme that allows two ADP molecules to combine to form ATP and adenosine monophosphate (AMP). This reaction provides little energy to the system, but AMP serves as one of the allosteric modulators to stimulate carbohydrate (glucose) breakdown in glycolytic metabolism. Energy from this system lasts anywhere from 5 to 10 seconds with high-intensity exercise and occasionally up to 20 seconds in some elite athletes.

The glycolytic pathway provides energy from 5 to 20 seconds up to 2 minutes as a result of the anaerobic breakdown of glucose. This is a more complex form of energy production, using multiple enzymes and reactions. In the first phase of glycolysis, a glucose molecule enters the cell where the enzyme hexokinase adds a phosphate group to the glucose molecule, which creates glucose 6-phosphate (G6P). G6P then enters a series of reactions to produce fructose 1,6-biphosphate. One of the reactions involves the enzyme phosphofructokinase, which adds another phosphate to the molecule. As a result, two ATP molecules are used in phase 1. In the first reaction of the second phase of the glycolytic pathway, fructose 1,6-biphosphate is converted into two three-carbon molecules. Glyceraldehyde 3-phosphate is then phosphorylated and oxidized, which releases two hydrogen molecules and two electrons. The two electrons and one hydrogen molecule combine with nicotinamide adenine dinucleotide (NAD^-) to form NADH, which can be used in oxidative metabolism. The four remaining reactions of phase 2 result in the production of two ATPs. Combined with two ATPs from the other three-carbon chain, this results in a total of four ATPs produced in phase 2. The net result of the glycolytic pathway is the production of pyruvate and two ATPs that may be used as energy (Figure 8-3).

If the pyruvate is not able to enter the oxidative energy system, it combines with NADH via the enzyme lactate dehydrogenase to produce lactic acid. When released into an environment with physiologic pH, lactic acid releases a proton and becomes lactate. Without a buffer, lactate production results in a decrease in cellular pH. Lactate production in itself is not necessarily detrimental to muscle

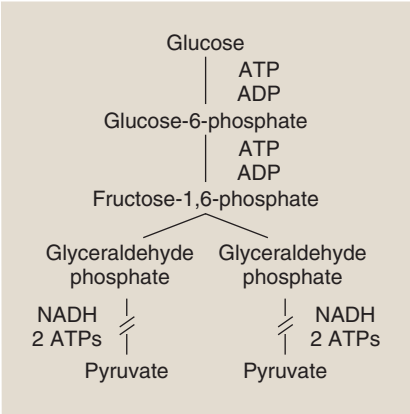


Figure 8-3 Glycolytic pathway with production of pyruvate and adenosine triphosphate (ATP). *ADP,* Adenosine diphosphate; *NADH,* nicotinamide adenine dinucleotide, reduced.

metabolism. After bouts of intense exercise, lactate is oxidized back to pyruvate, which can be converted to glucose in the liver, or converted to pyruvate in muscle and other tissues for ATP production. Lactate concentrations coincide with the release of a proton and potential decreases in pH. It is this decrease in cell and blood pH that has detrimental effects on energy metabolism and enzyme activity. Therefore, although associated with the release of protons, the lactate itself is not a problem, but the associated acidosis is.

The third energy source comes from oxidative metabolism, which predominates approximately 2 minutes after the beginning of exercise. It is the most complicated energy system and uses glycolysis, the citric acid cycle, and the electron transport chain. Each of these are complex multiple reaction cascades that result in the production of ATP and energy. Carbohydrates, lipids, and proteins are used as energy sources, and this is the system used to the greatest extent during long-term activity. Pyruvate is converted to acetyl–coenzyme A (CoA) by the enzyme pyruvate dehydrogenase, which also produces carbon dioxide and NADH. Free fatty acids are converted to acetyl-CoA by a process called β-oxidation. The resultant acetyl-CoA then enters the citric acid cycle. The products of this cycle are carbon dioxide, ATP, NADH, and flavin adenine dinucleotide, reduced (FADH).

Oxygen is used in the electron transport chain. The electrons acquired in NADH and FADH are added to hydrogen atoms and oxygen atoms to form water, along with energy to create ATP from ADP. The last electron receiver in the chain is oxygen. This process is termed *oxidative phosphorylation.* Three ATPs are produced for each NADH molecule, and two for each FADH molecule. If oxygen is not present to receive the electrons that flow down the chain, the chain stops, and the result is an accumulation of the components of the electron transport chain and the systems that produce them, halting energy production.

In a rehabilitation program, the conditioning program places energy demands on the muscles. The energy systems used depend on the forces required by the program and the duration of the workout. If the energy systems are insufficient to meet the demands required, the program will not benefit the patient and may actually harm the rehabilitative process. An understanding of muscle physiology will help in designing a conditioning program that is beneficial to the patient.

Musculoskeletal Conditioning

When the body performs at a level greater than its normal daily routine, there is a greater demand for energy. The systems must work together to provide energy to the areas of need, and at the same time maintain homeostasis. Therefore the fuel resources must be sufficient to meet this demand and be available to the body. In other words, energy must be stored in the body to perform physical activity, and the body's energy systems must be conditioned to efficiently produce, transport, and use this energy in the location where it is required. Homeostatic metabolism is associated with normal daily needs and allows the body to function properly throughout the day. If a short-term stress is placed on the system, the body is able to adapt, within reason, to meet this altered demand. If the stress is repeated on a daily basis, the daily routine is altered and the energy systems are conditioned to meet these new demands. Workout repetition results in adaptation to meet these demands, including pooling energy sources at the location of greatest need (e.g., intracellular ATP, CP, and glucose). The concentrations of enzymes required by the specific energy systems used the most may also increase. Workout repetition conditions the body to the stresses of the new demands and minimizes the chances of systemic or cellular injury.

The muscle response to different types of conditioning programs is determined by the muscle fiber type. The fiber types are divided into type I (slow-twitch) and type II (fast-twitch) muscle fibers. Type I muscle fibers are fatigue resistant, use aerobic metabolism, and have a low contractile force and less ATPase activity. Type II fibers are subdivided by their metabolic characteristics: type IIa (oxidative) and type IIb (glycolytic) fibers. Type IIa muscle fibers use both the oxidative energy system (aerobic metabolism) and the glycolytic energy system (anaerobic metabolism). They may be trained to use one system preferentially according to the demands placed on them. Type IIb fibers primarily use the glycolytic system (anaerobic metabolism), although there is some disagreement over whether dogs have true type IIb muscle fibers as compared with other species.[2-4] These fibers are generally termed type II Dog. Other intermediate fiber types have also been reported. Some change in muscle fiber composition may occur with exercise and conditioning.[5]

How a dog responds to a conditioning program may be affected by its fiber type composition. The type I/type II ratio of the muscle fiber types in a particular muscle is genetically predetermined, although some change in muscle fiber composition may occur with specific conditioning.[6] Greyhounds bred for sprint activity tend to have a greater percentage of type II fibers than other breeds, whereas foxhounds, which are bred for endurance, have a greater percentage of type I fibers.

In addition to genetic determination, the distribution of fiber types throughout the body and within individual muscles depends on the muscle's function. A higher percentage of type I fibers populate the muscles responsible for maintaining posture, whereas a higher percentage of type II fibers are present in muscles responsible for speed and power.[7] Altering the muscle function can change the fiber type. Muscle fiber type changes have been produced by tenotomy, tendon transfers,[8] and altered training.[9] Knowing the breed of dog and the anatomic area of focus helps determine the type of conditioning program that will be most beneficial for the patient.

There are two general categories of conditioning programs: strength-power programs and endurance programs. In human exercise physiology, endurance events usually last longer than 2 to 4 minutes and are performed at intensities of 90% or less of maximal aerobic power (Vo_{2max}). Strength-power events are of short duration (<2 minutes) and are performed at intensities that are maximal or supramaximal.[10] The metabolic changes that occur during a conditioning program depend on the principles of intensity, duration, frequency, and method of training. The program should incorporate these principles to result in proper system and organ adaptation.

Central and peripheral physiologic changes occur because of conditioning. Central changes include alterations to the cardiovascular, pulmonary, and endocrine systems. Peripheral changes occur in the musculoskeletal system. The conditioning program chosen should be specific to the type of activity the dog will be performing.

Short-duration, maximal-intensity workouts are recommended if the purpose of the program is to increase strength. This type of program affects peripheral conditioning more than central conditioning. If an increase in speed is the goal, the conditioning program should include sprint workouts at maximal intensity. Workouts that include exercises for the spinal muscles should be included, because it has been theorized that the spinal muscles are the first to fatigue during sprinting.[11] Uphill sprint running helps to strengthen and condition the spinal muscles. Running with resistance helps build body strength. Running with a harness and pulling weights is an example of resistance running. In a strength program, the most important change to the muscles is an increase in cross-sectional area because of increased myofibrillar protein content. In addition, there is an increase in muscle resting glycogen and muscle buffering capacity.

If the purpose of the program is to increase endurance, long-duration workouts of submaximal intensity are recommended. Examples are distance running or swimming. An endurance conditioning program may also include some strength activities. Endurance conditioning improves aerobic capacity to provide energy and conditions the musculoskeletal system to use energy more efficiently. In humans, changes occurring in the cardiovascular system include increased stroke volume and cardiac output.[12] Pulmonary changes include an improved ventilation-to-perfusion ratio, tidal volume, and alveolar and arterial partial pressures of oxygen.[13] Peripheral changes that occur in skeletal muscle are improved capillary density, glycolytic and oxidative enzyme activity, and lactate transport and clearance.[14]

Environmental Considerations with Conditioning

Dogs are homeotherms that maintain a normal body temperature between 37.9-39.9° C.[1] Environmental temperatures that exceed body temperature and metabolic heat gain from physical activity require that the animal employ heat loss mechanisms to maintain homeostasis. Heat transfer mechanisms for gain or loss include conduction, convection, and radiation. Evaporation is solely a heat loss mechanism, and dogs rely almost exclusively on panting for heat loss, especially when ambient temperatures are high.[1] This is because canine sweat glands are poorly developed and largely nonfunctional. Fluid loss from panting can be substantial; thus proper hydration is paramount. Cold environments require the dog to produce more heat via catabolism while conserving heat loss with peripheral vasoconstriction and countercurrent heat exchange.[1]

Tissue Injury

Injury of musculoskeletal tissues may occur when a sudden stress occurs that is too great for the body to overcome, or when chronic stresses are greater than the ability of the body to repair itself. This may occur in any of the musculoskeletal tissues, but the type and location of injury depend on the forces and the direction of the forces that cause the injury. For example, a sudden high-energy force exerted on a bone may cause a fracture, whereas chronic prolonged stresses may result in periostitis or stress fractures of the bone. These are examples of bone injuries, but similar stresses may also affect muscle, tendon, ligaments, or synovial tissue.

After injury occurs, the body adapts and the dog alters its movements according to limitations as a result of the injury. If pain is involved, gait is altered to minimize painful movement while allowing locomotion.[15,16] Injury also changes structure, which alters how forces are received by the body.[17,18] As the altered movement continues, the musculoskeletal system adapts to produce movement with minimal abnormal stress on the system.[19] However, the altered movement may create additional abnormal stresses that may lead to secondary and sometimes tertiary injuries at locations other than the initial insult.

The altered movement and forces resulting from a musculoskeletal insult should be addressed early before further injury occurs. The tissues involved should be determined by a proper diagnostic workup; then a treatment program that addresses the pain and altered structure is initiated. A physical rehabilitation regimen should be implemented soon after medical treatment is initiated for the initial injury to prevent biomechanical alterations that may result in secondary problems. Secondary injuries complicate the rehabilitative process and may prolong the recovery time.

Further injury may also occur during healing and rehabilitation. If the rehabilitation program places too much stress on the healing tissue, tissue healing may be impaired and the tissue may be reinjured. For this reason, return to normal function should be delayed until the original tissue insult has healed to a point that it can withstand normal activity. A properly managed rehabilitation program should

minimize the time of restricted activity without creating further injury.

Disuse and Immobilization

Following injury and rest, the body adapts to deconditioning, with changes occurring as a result of disuse or immobilization. *Disuse* refers to the state in which the segment is not used as a result of injury or dysfunction. Immobilization occurs when a limb is maintained in a fixed position by splints, casts, or external fixation. Both of these states result in disuse atrophy, which has effects on muscle, bone, ligaments, tendon, and cartilage.[20,21] Coincidently, many of the adverse effects associated with aging are a result of progressive reduction of physical activity.

Disuse atrophy of muscle results in decreased circumference and muscle fiber size. Type I muscle fibers are more affected than type II fibers, with a decrease in overall muscle strength. Muscle atrophy and weakness from immobilization occur quickly, and appear to affect antigravity muscles to a greater extent initially. Because of the decrease in strength and endurance, there is increased stress on the affected joints, abnormal movement, and an increased risk of injury.

Bone responds with an increase in bone resorption and perhaps decreased bone deposition and is guided by the external forces exerted on the bone. Immobilization also decreases loads on ligaments. Ligament mass decreases after just a few weeks of immobilization so that after 6 to 9 weeks of immobilization, the ligament complexes are only approximately 50% as strong as normal controls.[22,23] Tendons behave in a similar manner, with alterations in the balance of collagen synthesis and degradation. Articular cartilage also responds by atrophy and thinning of the surface layer of cartilage. If the joint is immobilized in a weight-bearing position, changes similar to those that occur with osteoarthritis may develop.

Immobilization may nevertheless be indicated as part of the initial treatment of many musculoskeletal injuries to allow an adequate healing environment, in spite of the changes that may occur with disuse and immobilization. The detrimental effects of immobilization should be considered in designing the treatment protocol because the recovery pattern is related to the duration of immobilization.

Reconditioning

A reconditioning program should address aspects of tissue healing, musculoskeletal physiology, and joint anatomy. It must include not only the injured area, but also the entire body. Nutrition, psychological aspects, and the effects of tissue injury on the body must also be considered. The goals should be to maximize healing of injured tissue and minimize degradation of healthy tissue. The primary long-term goal of rehabilitation is to maintain the physical condition of the injured segment during healing and not cause further damage. The secondary goal is to return the tissues of the affected segment to normal strength and character after injury and therapy. The third goal is to recondition the entire body to increase strength, coordination, and endurance to balance any secondary biomechanical disturbances that developed during injury and recovery.

The goal of maintaining condition of the ligaments, tendons, and cartilage may be achieved to some extent by passive range of motion. These tissues maintain their strength or conditioning level by mechanical action or movement, and by the forces placed on them. Bone depends more on loading forces and less on mechanical action and motion. Muscle requires active contractions to preserve tissue strength. In most instances, bone and muscle tissue are most affected during healing and rehabilitation.

After sufficient healing has occurred, the next step is to return strength to the involved tissues. The conditioning program should emphasize strengthening exercises. Before any reconditioning program is begun, the level of strength should be determined, and therapeutic goals should be set by the clinician and owner. The reconditioning program should then be designed to achieve these goals.

There are three types of strengthening exercises: isometric, isokinetic, and isotonic. Isometric exercises involve muscle contraction without movement. This type of exercise is difficult to achieve in dogs. Isokinetic exercises provide constant velocity throughout joint range of motion. Because of the equipment needed to perform this form of exercise, it has not been used in canine rehabilitation. Isotonic exercises involve forces of varying magnitude applied throughout the range of motion and is the most common form of exercise in dogs.

Isotonic exercise involves the muscle contracting while it shortens (concentric) or the muscle contracting while it lengthens (eccentric) while it is loaded throughout a range of motion. Loading the muscle stimulates strength gains. The effort should be high-intensity submaximal loading. Strength gain in reconditioning is similar to that of a regular strength program, except that the initial conditioning status of the tissue is less than normal. Exercises should be focused on strengthening the limb that is most affected by the injury, taking care to avoid overloading the healing tissues to avoid further damage.

As rehabilitation progresses, the focus shifts from localized exercise to overall body conditioning. This includes strength workouts balanced with endurance workouts. The strength portion of the program may still be focused on the areas of weakness, and the endurance portion can act on the entire body.

Nutrition

An important component of any conditioning program is nutrition. Nutritional needs change throughout the

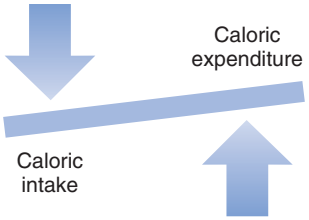


Figure 8-4 Caloric balance depends on intake and expenditure. Body mass is maintained when intake equals expenditure.

treatment and time of rehabilitation. Initially, there is a decrease in energy use by the patient. This should be addressed to keep the patient from becoming overweight during rehabilitation. After reconditioning begins, nutritional components may be added to allow for the nutritional demands of increased physical activity. The basic feeding program should provide for the basic nutritional needs of the patient. The content of protein, carbohydrate, and fat in the feeding program is determined by the type of conditioning. For example, dogs that are performing strength workouts use the immediate and glycolytic energy systems. Therefore they may require a lower percentage of fat in their diets. Caloric balance is the ultimate consideration for diet in dogs (Figure 8-4). If caloric expenditure exceeds caloric intake, then energy will come from various deposits within the body, including stored fat, glycogen, and muscle, and body mass will decrease. Conversely, excessive caloric intake and inadequate energy expenditure is associated with energy storage as glycogen or fat, and increased body mass.

Body mass varies greatly across the variety of dog breeds. Body composition may vary with breed, but largely depends on age. Metabolic rate adjusted for body mass may also vary between some breeds.[24] As in human physiology, it appears that canine resting metabolic rate (RMR) declines with age; dogs aged 7 to 8 years have one-half of the RMR of dogs aged 1 to 2 years.[24] These factors underscore the importance of a lifetime of physical activity, which stimulates the various physiologic systems and maintains a healthy weight when in balance with caloric intake.

Supplemental feeding should be timed to benefit conditioning. An important feeding strategy is to provide a post-workout snack. This is the optimal time to replenish nutrients that become depleted during the workout. Immediately after the workout, the body is devoted to replenishing the deficits of the energy systems. Feeding a snack during this time promotes replenishment of the deficient nutrients. This snack should consist of simple carbohydrates, a meat protein source, calcium, and phosphorus.

Summary

A proper conditioning program provides a good healing environment for injured tissues. It should minimize the detrimental effects of injury to the healthy tissues and the entire body. As healing progresses, the program should recondition and strengthen the tissues that are affected by injury. It should also provide nutrients to allow proper healing and be designed to consider the psychological aspects of rehabilitation. An understanding of exercise physiology is necessary to design and implement rehabilitative programs that aid tissue healing and recovery of the patient.

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9 Exercise Physiology of the Canine Athlete

Ralph Millard

Exercise physiology is the study of the effects of exercise and training on normal functions of the body. When implementing a rehabilitative exercise or a conditioning program it is important to have a thorough understanding of normal and abnormal physiologic functions of the body. Sufficient knowledge of physiologic functions of the body increases understanding of the body's response to exercise and allows one to maximize the effectiveness of a rehabilitative program. Exercise has many profound effects on the musculoskeletal system. There is a vast amount of coordination and integration of individual body systems, and, although exercise programs and our rehabilitative efforts mainly focus on improving the function of the musculoskeletal system, it is important to understand the response of other major body systems to the influence of exercise. Physical rehabilitation is typically used to treat or condition areas of the body that are injured or are suffering from abnormal function. Because there may be limitations during healing of an injury, one should understand the body's limitations during injury as well as compensatory methods used by the body. A good general understanding of physiology is very beneficial to the clinician performing rehabilitation, in designing a rehabilitation program and understanding how exercise and training affect the body.

Skeletal Muscle

Muscle Physiology

Skeletal muscle plays a large role in exercise and determining athletic performance, and it is important to understand its structure and function. The muscular system is a collection of contractile units that provide the forces for various functions to take place such as locomotion, posture, respiration, and circulation. The three types of muscle in the body are skeletal, smooth, and cardiac muscle. This section focuses on skeletal muscle physiology.

The functional unit of skeletal muscle is the muscle fiber, or myofiber (Figure 9-1). Most skeletal muscles contain muscle fibers that span the entire length of the muscle, cross over at least one joint, and are typically innervated by only one alpha motor neuron. The sarcolemma is the cell membrane that surrounds each muscle fiber. Each muscle fiber contains several hundred to several thousand myofibrils. Myofibrils are composed of contractile protein filaments called *myosin* (thick filaments) and *actin* (thin filaments). Myofibrils are divided into segments called *sarcomeres,* each of which lies between two Z discs (filamentous protein different from actin and myosin). From each Z disc actin filaments extend in both directions to interdigitate with myosin filaments.

Contraction of a muscle fiber is first initiated by an action potential, which starts at the central nervous system, travels down an alpha motoneuron to its own axon and the neuromuscular junction. The action potential causes a large influx of calcium ions through calcium channels. The calcium influx results in the release of acetylcholine into the extracellular space between the motoneuron and the neuromuscular junction of the muscle fiber. The acetylcholine then activates nicotinic acetylcholine receptors, opening sodium-potassium channels, triggering an action potential. The action potential travels along a muscle fiber's T-tubule network, depolarizing the muscle membrane. The action potential causes the sarcoplasmic reticulum to release large quantities of calcium into the cytoplasm. A protein called *tropomyosin* is associated with each actin filament. In a resting state tropomyosin covers the active sites on the actin filaments so that attraction between an actin and myosin filament cannot occur and cause contraction. Calcium released from the sarcoplasmic reticulum binds to a protein called *troponin,* which causes the tropomyosin to shift positions and uncover the binding site between actin and the cross bridge portion of myosin filaments. Prior to binding of actin and a myosin cross bridge, adenosine triphosphate (ATP) bound to myosin is cleaved; however, the adenosine diphosphate (ADP) and phosphate ion remain bound to the cross bridge. When the myosin head portion of the cross bridge bonds with the active site on the actin filament, a conformational change is created, causing a power stroke or pulling of the actin filament. The previously cleaved ATP supplies the energy for a power stroke. After the power stroke has been completed, the ADP and phosphate ion are released from the myosin head, allowing for binding of a new ATP molecule. The binding of a new ATP molecule causes detachment of the myosin head from the actin filament. The ATP is then

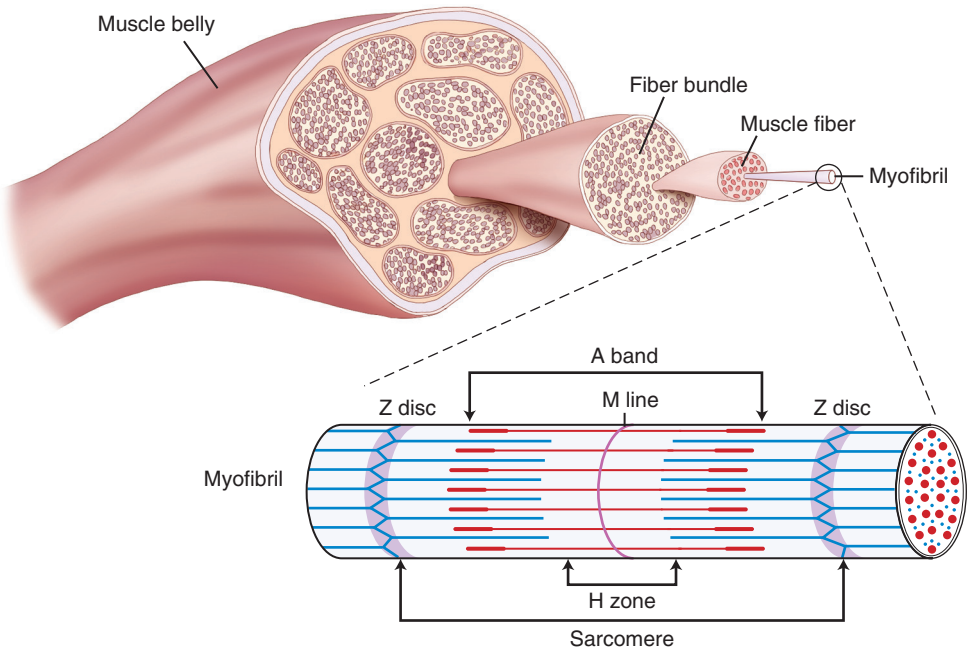


Figure 9-1 Skeletal muscle diagram.

cleaved to begin a new power stroke. This process is repeated again and again to contract a muscle.

Skeletal muscle cells are classified into types based on the specific individual contractile and metabolic properties of each cell. A general classification scheme divides muscle fibers based on their speed of contraction, which is a result of their contractile and metabolic properties. Generally speaking, muscle fibers are considered to be either slow twitch (type I) or fast twitch (type II). Type I fibers contain higher levels of oxidative enzymes, myoglobin, and mitochondria, and are well suited to perform aerobic activities and resist fatigue. Type II fibers have smaller amounts of oxidative enzymes and limited aerobic capabilities. However, type II fibers have large amounts of glycolytic enzymes for anaerobic metabolism and are capable of generating higher levels of tension and shortening velocity, but are much less resistant to fatigue than type I fibers.

Dogs are rather unique in that, along with the aerobic abilities of slow twitch fibers, their fast fibers also have significant oxidative capabilities. In fact, there is significant evidence that suggests an absence of purely glycolytic muscle fibers in the skeletal muscle of the trunk and limbs of dogs.[1] It has been suggested that the only location of type IIB muscle fibers in dogs is in laryngeal muscles.[1-3] It is believed that previous studies that relied on immunostaining alone have resulted in misclassification of muscle fibers. Newer studies have implemented electrophoresis, immunoblots, immunohistochemistry, and image analysis to characterize canine muscle fibers.[1,3]

Currently, five major fiber types are described in the trunk and limb muscles of dogs, which include three pure fibers (I, IIA, and IIX) and two hybrid fibers (I+IIA and IIAX).[1,3] The oxidative/glycolytic ratio of canine muscle fibers decreases from type I (highest) to IIX (lowest). Type IIA is intermediate, with both hybrids between their respective pure isoforms.[1] The decrease in oxidative potential from type I to type IIX is three to four times less than other mammals, which is another finding supporting the superior oxidative capacity of canine skeletal muscle. Even the least oxidative muscle fibers in dogs have a substantial capability for aerobic metabolism compared with fast fibers of other species. The maximum shortening velocity of canine muscle fibers increases from type I (slowest) to type IIX (fastest).[3]

Muscle Fatigue

Contraction of skeletal muscle at some rates required for exercise cannot continue indefinitely. When a muscle is unable to maintain power output during contraction, it has reached the point of fatigue. There are several explanations for the cause of fatigue. One controversial explanation is that fatigue can originate from the central nervous system (CNS), possibly because of psychological factors that can either enhance or limit performance. Dogs may serve as a good example for this theory because the personality of certain breeds of dogs might allow them to continue to exercise beyond the point that other breeds or species would continue. The drive to run or retrieve might allow some dogs to continue to participate in intense physical activity despite obvious exhaustion, simply as the result of verbal encouragement, whereas other breeds or species would not continue regardless of the stimulus. The

evidence on CNS involvement in fatigue is unclear and unproven.

However, there are several changes at the cellular level of skeletal muscle that result in fatigue during exercise. Fatigue is mainly an imbalance between ATP requirements by muscle and the ability to produce ATP. During exercise, the need for ATP by skeletal muscle dramatically increases. As the components needed to regenerate ATP levels become depleted, there is less ATP available to provide energy for sustained muscle contraction, resulting in an inability to exercise or perform at the same level or rate as before. Phosphate is one of the components needed for formation of ATP. When ATP formation cannot keep up with ATP consumption, excessive inorganic phosphate ions accumulate in the muscle cell. High concentrations of phosphate ions can directly interfere with cross bridging of actin and myosin, as well as inhibit calcium release from the sarcoplasmic reticulum.[4,5] Lactic acid (lactate) is an end product of glucose metabolism by way of the glycolytic pathway. Lactate is formed during conditions of inadequate oxygen supply to muscle or by muscle fibers with low numbers of mitochondria. Accumulation of lactic acid as a result of anaerobic metabolism within muscle cells can also inhibit enzymes involved in production of ATP.

Glycogen is an important energy source used during exercise. Depletion of muscle glycogen is a critical factor associated with fatigue in elite athletes during prolonged periods of exercise.[6] Sled dogs are a good example of an elite endurance athlete. High-endurance canne athletes can have a remarkable ability to maintain muscle glycogen concentrations during periods of prolonged exercise. Despite limited carbohydrate intake, sled dogs running 160 km (99.4 miles) per day for 5 consecutive days did not develop cumulative muscle glycogen depletion.[7,8] On the first day of exercise there was a decrease in muscle glycogen content; however, each of the subsequent periods of exercise resulted in stable muscle glycogen content, which was greater than the content after the first day. It has been determined that muscle glycogen preservation in high-endurance athletes, such as sled dogs, occurs as a result of attenuation of depletion rather than rapid replenishment.[8] During the first portion of multiday endurance racing, sled dogs use a significant portion of muscle glycogen. On subsequent days they use only a small percentage of their remaining muscle glycogen stores. Rather than glycogen, sled dogs use other energy sources such as muscle triglycerides. Dogs are likely capable of this form of metabolism because of their innate ability to use free fatty acids, their ability to perform high levels of training in endurance activities, and the consumption of a diet with a large percentage of fat as one of its components. Canine skeletal muscle is composed mostly of type I, IIA, and IIAX muscle fibers, all of which are capable of high levels of oxidative metabolism.[1] Highly aerobic mammals such as dogs have an increased capability to use fat-based substrates for energy metabolism.[9] The high-fat, high-protein diet of sled dogs can help preserve muscle glycogen stores during exercise. Evaluation of fasted rats subjected to several bouts of repeated exercise showed similar results of muscle glycogen preservation, suggesting that metabolic adjustments allow certain species, such as dogs, to preserve muscle glycogen stores during bouts of repeated exercise. There may be a critical concentration of muscle glycogen that the body protects against excessive consumption during repeated exercise. Compared with humans, conditioned canine athletes may have an advantage regarding prolonged exercise based on their highly aerobic metabolism and ability to use alternative substrates for energy and maintenance of muscle glycogen stores.

Conditioning and Training of Skeletal Muscle

Conditioning of skeletal muscle involves performing physical exercise that prepares muscle for the specific task that it will be asked to perform. Training pertains to incorporating the specific exercise into the type of sporting event or activity the dog will be involved in. A portion of training also involves behavior modification required to perform the specific activity. During exercise training athletes are frequently pushed to perform an activity until physically unable to continue. This is commonly termed the *overload principle,* which refers to the need for a system to be exercised to a level beyond that which it is accustomed for training to have an effect. Placing the system in an overloaded state causes it to adapt to new conditions, thereby improving a system's ability to perform a task at a higher level of performance. This principle applies to the cardiovascular system, the musculoskeletal system, and other body systems. Factors that influence overload include intensity, duration, and frequency of a particular exercise. Most types of exercise training can be divided into either endurance or strength training activities. It is important that training uses the systems and structures involved in the activity. This is a training principle called *specificity.* For example, a dog that competes in a sport that requires pulling loads would be better served to run while pulling against resistance as opposed to trotting for a long duration on a treadmill. In humans, engaging in training activities that target specific muscle groups results in adaptations in the muscle types targeted. For example, endurance-type exercises, such as running prolonged distances at a submaximal heart rate, increases the capacity to produce energy in an aerobic manner by increasing capillaries and mitochondria, especially in type I muscle fibers.[10]

High levels of endurance are very important in dogs that participate in prolonged periods of exercise such as sporting, herding, and long-distance racing dogs. Endurance relates to the ability of a muscle or group of muscles to undergo many repetitions of contraction under low loads.

Endurance exercise usually targets specific muscle groups involved in the specific action and lasts for a prolonged time, usually greater than 15 minutes. Endurance exercises for dogs include trotting, running, swimming, land and underwater treadmill activity, and long-distance sled pulling. It was previously believed that endurance exercise does not result in an increase in type I muscle fibers. More recent research indicates that prolonged endurance exercise can promote a shift of type II muscle fibers to type I in humans.[11] Other changes associated with long-term endurance training include an increase in capillary density and vascularity of muscle tissue, increasing the delivery of oxygen and nutrients to the muscle. The issue of muscle fiber type conversion after endurance exercise in humans remains somewhat controversial, but can be explained by the fact that muscle fibers are classified by staining them and as their aerobic capacities are altered, fibers that were borderline in classification can "shift" into another category.

Studies have evaluated skeletal muscle adaptation in dogs during periods of endurance training. One study of foxhounds undergoing endurance exercise failed to show the biochemical and histochemical adaptations in skeletal muscle that have been found in some studies involving humans.[12] Foxhounds did not show alterations in the activity of succinate dehydrogenase (SDH), muscle fiber type, or capillary density of skeletal muscle after a 12-week endurance training program.[12] These findings contrast with those of humans, who do undergo detectable skeletal muscle adaptations after endurance training. SDH is a measure of the oxidative capacity of a muscle. There was no difference in SDH activity, muscle fiber staining, or capillary density in trained and untrained foxhounds, suggesting that an untrained dog is already well suited to support the highly oxidative metabolism of endurance exercise, or that the training program employed in that study was not challenging enough to result in muscle fiber changes. The lack of a change in fiber type staining after endurance exercise may support the theory that dogs, such as foxhounds, are highly reliant on oxidative enzyme metabolism during exercise independent of the pattern of motor unit recruitment.[12]

Another study, however, concluded that endurance exercise can induce a shift of type II to type I muscle fibers.[13] Beagles exercising on a treadmill 5 days per week for 55 weeks demonstrated an increase in type I fibers in the thoracic spinal muscles as well as the triceps muscle.[13] The unique nature of canine skeletal muscle as well as the shortcomings of various staining and imaging techniques has made classification of canine skeletal muscle a difficult task, much less classifying changes as a result of endurance training. It is fairly well accepted that canine skeletal muscle has a relatively high capacity for aerobic metabolism.[1] It may be likely that dogs experience changes in muscle fiber type in response to exercise; however, some of the shared characteristics of type I and type II fibers in dogs have led to misclassification, and therefore uncertainty in potential changes in muscles and their fiber types.[1] Newer techniques used to identify canine muscle fiber types might provide insight into identification of possible fiber type conversion as a result of endurance exercise. In addition to a lack of concrete evidence to support the theory of muscle fiber type conversion, dogs undergoing endurance exercise have not shown an increase in muscle capillary density, capillary to fiber ratio, or capillary area to fiber ratio in some studies.[12] The lack of a change in capillary density in skeletal muscle after endurance training suggests that microcirculation of untrained foxhounds is already adequate to meet the demands of endurance training or the overload needed to promote changes was not achieved in these studies. Even untrained dogs have a high aerobic capacity and appear to be genetically adapted for efficient oxygen extraction and use.

However, it is important not to confuse an untrained dog with a sedentary, inactive, or obese dog. There is a lack of studies involving changes in skeletal muscle in sedentary or overweight dogs after undergoing exercise training. It is likely that untrained but active dogs possess advantages over sedentary or obese dogs at the cellular level of skeletal muscle. Significant biochemical and histochemical changes have yet to be documented in sedentary dogs after undergoing endurance exercise training. However, endurance training is likely very beneficial for the overall health of dogs and improved athletic function. Perhaps future studies may discover previously unknown adaptations of canine skeletal muscle, which are uniquely different from those of humans and other species.

Other adaptations and alterations in body systems occur after endurance training that likely allow for improved performance during exercise. Although there may not be specific biochemical or histochemical change in muscle, there is some support that endurance exercise can improve the functional blood tissue gas exchange properties of skeletal muscle in dogs. A study evaluating endurance-trained dogs and dogs with one limb immobilized found that at a constant rate of oxygen delivery, endurance-trained dogs had an increased Vo_2 max, mainly as a result of increased oxygen diffusive conductance (Do_2) in skeletal muscle.[14] An interesting discovery, however, was that despite a 31% reduction in muscle weight, there was no difference in Vo_2 or Do_2 between immobilized dogs and a control group during exercise.[14]

Strength of muscle refers to the maximal force a muscle or group of muscles can generate during one repetition. Muscle strength is associated with an increase in muscle size (hypertrophy). Type II muscle fibers are capable of developing more force per cross-sectional area and contract at a higher velocity than type I fibers (Figure 9-2). Strength training increases the size of type II fibers more than type I.[10] High-resistance training increases the number

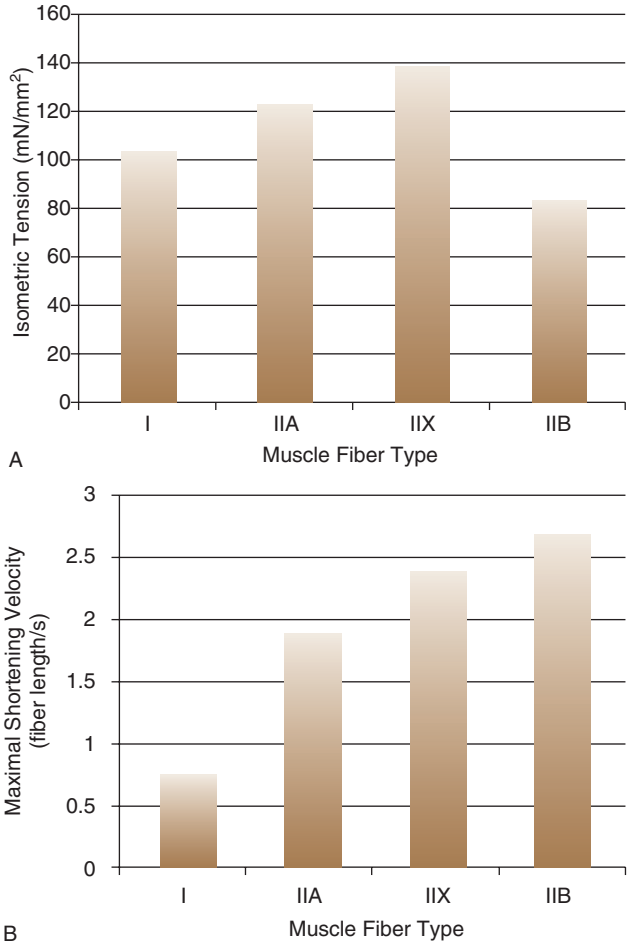


Figure 9-2 **A,** Comparison of maximal force generated between canine fiber types. **B,** Comparison of maximal shortening velocities between canine fiber types. (Adapted from Toniolo L, Maccatrozzo L, Patruno M et al: Fiber types in canine muscles: myosin isoform expression and functional characterization. *Am J Physiol Cell Physiol* 292:C1915-C1926, 2007.)

of contractile proteins in type II fibers, which increases the cross-sectional area and the force they are able to generate. The enlargement of muscles that occurs with strength training is thought to mostly occur as a result of hypertrophy of muscle fibers and not hyperplasia (adding muscle fibers). Strength in dogs is mainly related to speed and the ability to carry or pull loads. Strength is also important for dogs that must accelerate and decelerate very rapidly, such as dogs that participate in agility competitions. Like strength, sprinting speed is also most affected by type II muscle fibers. The distribution of certain fiber types also depends on genetic factors. Sprinting breeds, such as greyhounds, have a greater percentage of type II muscle fibers, whereas breeds such as foxhounds have larger percentages of type I fibers and are more adept at endurance exercise. Short-duration, maximal-intensity workout programs are best suited for promoting an increase in strength or speed.

In addition to conditioning muscles of the limbs it is also important to focus on spinal muscles. During sprinting exercise it has been observed that spinal muscles are among the first to experience fatigue.[15] Incline treadmill running and uphill sprinting are good activities to strengthen muscles, including the spinal muscles. Other strengthening exercises include running or racing, pulling or carrying resistance loads, swimming against jets, and trotting or running up inclines or stairs. Performing strength exercise can increase the size of muscle groups and the force they are capable of generating. The opposite is true if skeletal muscle is subjected to periods of disuse or immobilization. Under these conditions, the muscles undergo atrophy and decreased muscle strength of the limb. A study that evaluated muscle atrophy caused by immobilization found that immobilization of the hindlimb for a period of 10 weeks reduced the diameter of type II fibers by 50% and type I fibers by 35% in the vastus lateralis muscle and maximum production of tetanic torque by the affected limb decreased by 50%.[16] When removed from the immobilized state, growth hormones increased the size of muscle fibers and increased strength of the previously immobilized limb during the recovery period significantly more than a control group.[16]

Respiration

The term *respiration* can relate to pulmonary or cellular respiration. This section discusses pulmonary respiration and the effect of exercise on ventilation. The term *pulmonary respiration* refers to ventilation of air into and out of the lungs and the exchange of gases between inspired or expired air and lung tissue. *Ventilation* refers to the mechanical movement of air into and out of the lungs. Diffusion is the process by which gas molecules move either from air into blood or blood into air. In addition to providing a means of supplying oxygen to the blood and tissues of the body and allowing for release of carbon dioxide, the pulmonary system plays an important role in regulating acid-base balance during rest and during exercise, as well as thermoregulation. Inflow of air to the lungs occurs as a result of intrapulmonary pressure dropping below atmospheric pressure, and expiration occurs when intrapulmonary pressure rises above atmospheric pressure. The most important inspiratory muscle is the diaphragm; however, any muscle capable of increasing the volume of the chest cavity can contribute to inspiration. Because of the elastic nature of pulmonary tissue and the thoracic cavity, expiration at rest is typically a passive event. During exercise, expiration can become partially active because of contraction of muscles of the abdominal wall.

Precise control of ventilation and pulmonary gas exchange during exercise is required to maintain P_{O_2}, P_{CO_2}, and acid-base balance. During exercise a change in ventilation is required to keep up with metabolic needs, which

requires an increase in ventilation.[17,18] Higher demand for oxygen and a need to rid the body of large amounts of carbon dioxide necessitate higher rates of ventilation. The respiratory control center in the medulla oblongata initiates the drive to inspire and expire. To increase ventilation, input to the respiratory center can come from both humoral and neural sources. At the onset of exercise, one theory states that impulses from the motor cortex to control skeletal muscle movement can "spill" over into the respiratory center as they pass through the medulla, causing an increase in ventilation.[19] This is further supported by the fact that during strenuous exercise, arterial PO_2, PCO_2, and pH often remain almost the same as during resting. At the initial onset of exercise, ventilation increases prior to any significant changes in blood chemicals. This supports the theory that initial ventilatory changes are associated with signals sent to the respiratory center of the brainstem at the same time that signals are sent to skeletal muscle. Chemoreceptors in respiratory muscles may respond to potassium and H^+ and send afferent information to the respiratory center. Central chemoreceptors detect increases in PCO_2 and decreases in pH. Peripheral chemoreceptors are sensitive to increases in PCO_2 and decreases in PO_2 or pH. All of these receptors act to fine-tune ventilation during exercise. Input from higher brain centers, regulation of PCO_2, and lowering of pH caused by increased blood lactate levels are all methods thought to increase ventilation during exercise.

The process of breathing at rest is clearly initiated in the CNS after input from other areas of the CNS, as well as peripheral signals. However, the exact causes of breathing in dogs during exercise prompted investigation to determine whether ventilation is an active or passive process. Some have proposed that the passive forces from limb movement in trotting dogs contribute a substantial amount to hyperpnea during exercise.[20] The forces believed to affect respiration include the cranial and caudal displacement of the abdominal organs during acceleration or deceleration; concussive forces of limb impact, which change intrathoracic pressure; flexion and extension of the lumbosacral region resulting in compressive forces within the abdomen thus leading to intrathoracic pressure changes; and inertial forces on the chest wall caused by limb contact with the ground.[20]

A subsequent study demonstrated different results that contradict the proposition that breathing in dogs during exercise is largely a passive event associated with stride. It was concluded that breathing in exercising dogs is a neural-driven event. Diaphragmatic electromyographic activity, esophageal pressure changes, and phasic diaphragmatic shortening and breathing frequency remain strictly synchronous during exercise and are not significantly associated with stride.[21] Active recruitment of the diaphragm and abdominal muscles is the major contributor to intrathoracic pressure changes and generation of airflow, and oscillations of abdominal contents associated with locomotion are not critical for airflow in exercising dogs.[21]

The diaphragm is required to perform very high levels of work to provide adequate ventilation during exercise. The diaphragm muscle is highly oxidative, preventing it from fatiguing easily. Historically, the muscles of the respiratory system were not thought to be a limiting factor during exercise. *Respiratory muscle fatigue* is defined as a condition in which there is a loss in the capacity for developing force or velocity of muscle contraction of respiratory muscles, resulting from muscle activity under load, which is reversible by rest.[22] Studies have confirmed that respiratory muscle fatigue can occur during high-intensity exercise in humans.[23] It is also believed that during prolonged high-intensity exercise, respiratory muscle fatigue can limit exercise performance in humans. One notable finding in humans was that fatiguing respiratory muscles cause reflex vasoconstriction to skeletal muscle of the limbs, resulting in exercise limitation of muscles, thus limiting some of the oxygen demand.[23] There have been no studies evaluating respiratory muscle fatigue in healthy exercising dogs; however, the condition occurs in cases of cardiogenic and septic shock.[24,25] It is likely that the highly aerobic nature of the canine diaphragm muscle normally protects it from fatigue.

Healthy dogs are likely to have a low likelihood of developing respiratory muscle fatigue. However, dogs with underlying conditions, especially those involving the respiratory system, are more likely to be susceptible to fatigue. Dysfunction of the respiratory system, such as pneumonia, increases the work load for respiratory muscles. Dogs engaged in strenuous exercise can reach high rates of ventilation. Although respiratory muscles such as the diaphragm may not fatigue easily during exercise, energy is still required to maintain such a high rate of ventilation. One consideration is that the cost of energy to maintain high rates of ventilation can represent a significant amount of total energy expenditure of a dog during exercise and thus represent a potential limiting factor of performance. A study that calculated the dynamic respiratory work of exercising dogs suggested that the higher rates of ventilation and elevated body temperature associated with exercise may induce changes that decrease the cost of breathing.[26] Dogs exercising on a treadmill showed a decrease in dynamic respiratory work as a result of decreased airway resistance that resulted in up to an eightfold savings of energy expenditure of respiratory muscles. When body temperature rises to more than 39° C and respiratory rate increases to 120 breaths per minute, there is an apparent decrease in dynamic respiratory work as a result of lower airway resistance.[26] It is unknown whether there is a progressive decrease in airway resistance as body temperature rises or if a sudden change in breathing frequency induces the change. It is believed that the most likely site of resistance reduction is at the level of the larynx. During

states of hyperthermia, cats have reduced laryngeal resistance to almost half of normal values.[27] The lower airway resistance in exercising dogs decreases the amount of energy required to maintain the rates of ventilation required for strenuous exercise. This mechanism may help resist respiratory muscle fatigue as well as conserve energy for use by skeletal muscles in the trunk and limbs.

Depending on the environment a dog exercises in, it is possible for ambient temperature to have direct effects on the respiratory system of athletes. Repeated exercise in cold environments can result in airway disease. Dogs exercising in cold conditions can develop pulmonary changes similar to a condition known as "ski asthma" in humans. Dogs exercising for prolonged periods in cold environments show significantly more reactivity to aerosol histamine and overall greater airway resistance when compared with dogs not racing in cold environments.[28] This type of dysfunction can persist for at least 4 months after rest. It is believed that continuous cold air hyperventilation causes excessive heat and water loss, leading to airway mucosal injury. The specific effects of cold weather changes of the respiratory system on exercise performance have not been fully evaluated. However, any condition that results in inflammatory changes of the airways would likely have some degree of negative effect on performance.

Cardiovascular System

The primary purpose of the cardiovascular system is to deliver oxygen and nutrients to the tissues of the body as well as remove wastes and aid in temperature regulation. To accomplish these tasks, the cardiovascular system works in conjunction with the respiratory system. During exercise, cardiac output must increase in proportion to the increase in metabolic rate. In human athletes there is a linear relationship between percent maximal oxygen uptake (Vo_2 max) and cardiac output. Cardiac output is the quantity of blood pumped into the aorta each minute by the heart and is determined by the product of the heart rate and volume of blood ejected per heartbeat (stroke volume). This in turn determines the amount of blood that flows through the circulation. An increased demand for oxygen and nutrients by skeletal muscles during exercise requires an increase in cardiac output as well as redistribution of blood from inactive organs to active skeletal muscle.

The onset of exercise induces changes that increase cardiac output. The initial changes in the cardiovascular system at the onset of exercise are thought to originate from centrally generated motor signals followed by modification and fine-tuning from baroreceptors, chemoreceptors, and mechanoreceptors throughout the body.[29] Both inhibition of the parasympathetic and activation of the sympathetic nervous system act to increase heart rate and strength of contraction during exercise. Stroke volume is also increased during exercise. Venous return to the heart is increased by venoconstriction, which decreases the storage capacity of the venous system and causes blood to move back toward the heart. The mechanical action of contracting skeletal muscle compresses veins to push blood toward the heart. The rhythmic action of breathing also promotes blood flow from the abdomen to the thorax. Increased flow of blood to the heart increases the volume of blood in the ventricles at the end of diastole (preload) available for the heart to pump back into circulation for skeletal muscle demands. The increased preload also acts to increase the strength of muscular contraction by way of the Frank-Starling mechanism.

Other methods of meeting the increased oxygen demands of skeletal muscle during exercise are to increase blood flow to muscle by decreasing blood flow to other organs and vasodilatation of skeletal muscle arterioles. Skeletal muscles in resting humans receive approximately 15-20% of total cardiac output; during maximal exercise this increases to 80-85% of total cardiac output.[29] There is some controversy as to the exact mechanism by which skeletal muscle blood flow increases, and blood flow to other organs decreases during exercise. During exercise, vascular resistance in skeletal muscles decreases while vascular resistance in visceral organs increases.[30] Initial vascular resistance within skeletal muscle is believed to decrease as the result of withdrawal or attenuation of sympathetic input to skeletal muscle arterioles; however, maintenance of vasodilatation is likely due to intrinsic control caused by local changes in metabolites such as pH, oxygen tension, and nitric oxide.[29,31] Another possible mechanism that acts to increase blood flow to muscle in dogs is the concept of *sympatholysis,* which was demonstrated by attenuation of vasoconstrictor response during dynamic exercise compared with a resting state.[32]

Studies have been performed regarding the effect exercise has on the cardiovascular system of dogs. Similar to humans, changes in the cardiovascular system of dogs are required to meet the metabolic needs of skeletal muscle during exercise. Sled dogs, some of the most elite canine athletes, have provided invaluable information on the cardiovascular system during maximal exercise. The heart rate of sled dogs can dramatically increase at the onset of exercise. Heart rate can increase from a resting rate of 50 beats per minute to 300 beats per minute and remain between 250-300 beats per minute during a 60-minute run.[33] Foxhounds exercising on a treadmill show similar trends of change in heart rate. Heart rate continues to increase as dogs are subjected in increasing levels of submaximal exercise. When foxhounds reach a state of maximal treadmill exercise (approximately 12 km/hr on a 28% grade), heart rates approach 300 beats per minute (Figure 9-3, *A*). When comparing heart rate of exercise-trained and untrained foxhounds at different levels of submaximal exercise, trained dogs have on average a heart rate 14 beats per minute lower than untrained dogs at each level of

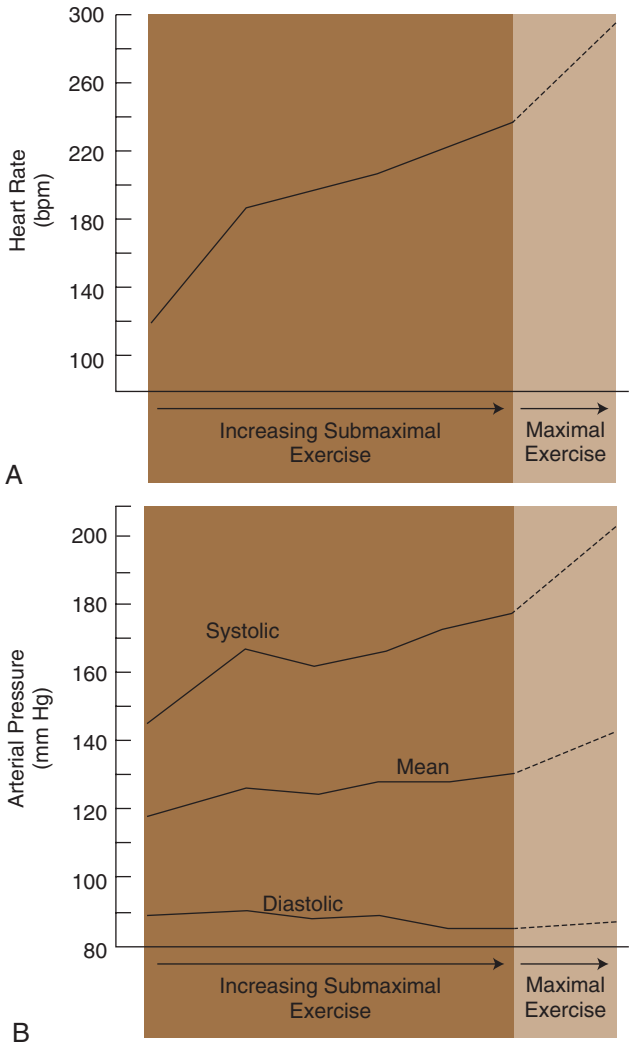


Figure 9-3 **A,** Changes in heart rate of foxhounds as a function of relative work rate. **B,** Changes in arterial blood pressure of foxhounds as a function of relative work rate. (Adapted from Musch TI, Haidet GC, Ordway GA et al: Dynamic exercise training in foxhounds. I. Oxygen consumption and hemodynamic responses. *J Appl Physiol* 59:183-189, 1985.)

measured submaximal exercise.[34] Exercise also affects blood pressure. Systolic pressures can increase to more than 200 mm Hg during exercise. However, diastolic pressures during exercise are frequently lower than resting values, resulting in a mean pressure similar to that of resting values[33] (Figure 9-3, *B*). These changes are the result of arteriolar vasodilatation at the level of skeletal muscle, which decreases diastolic blood pressure.

The heart itself undergoes changes as a result of exercise, to better meet demands of the body.[35] The term *athletic heart* has been used to describe cardiac changes that occur as a result of prolonged high-intensity exercise. In human athletes, strenuous repetitive exercise enhances vagal tone and causes left- and right-ventricular enlargement.[36] These cardiac changes can result in resting bradycardia, increased end-diastolic volume, and increased stroke volume. Changes such as physiologic murmurs, bradyarrhythmias, and cardiac chamber enlargement are thought to be associated with cardiac adaptations to sustained endurance exercise and not pathologic conditions.[36] Auscultatory and electrocardiographic (ECG) studies have found that highly trained dogs have cardiac changes very similar to human elite athletes.[37,38] Murmurs evident on auscultation are frequently heard in highly trained dogs compared with untrained dogs. Murmurs in athletic dogs are typically grade I-II systolic murmurs heard best over the left heart base.[37] The murmurs are likely a result of increased blood velocity passing through the aortic valve in response to physiologic heart hypertrophy and increased stroke volume.[37] It has been observed that highly trained dogs without a low-grade murmur tend to perform more poorly than dogs that have a low-grade murmur.[37] This is further evidence that low-grade murmurs in endurance-trained dogs are a physiologic response to exercise and not evidence of a pathologic cardiac condition. Cardiac studies on canine athletes have shown that endurance training results in a significant reduction in resting heart rate as well physiologic hypertrophy of the heart.[38,39] ECG monitoring of canine athletes engaged in endurance training has shown an increase in QRS duration, which is reflective of physiologic cardiac hypertrophy.[38] Hypertrophy of the canine heart as a result of endurance training is believed to be a reversible event.[38]

Blood flow changes in exercising dogs are similar to changes seen in exercising humans. Blood flow to the terminal aorta and coronary vessels of dogs significantly increases during exercise as a result of increased cardiac output.[33] Increased blood flow to the coronary vessels is important because just as skeletal muscle increases its oxygen demands during exercise, so does cardiac muscle. Some studies of splanchnic blood flow in dogs during exercise are in disagreement with one another. One study of blood flow during exercise determined that mesenteric and renal blood flow were unchanged in racing sled dogs compared to resting state.[33] These findings suggested that increased cardiac output and autoregulation of vascular resistance in skeletal muscle, and not redistribution of blood volume from other organs, mainly control increased blood flow to skeletal muscle in exercising sled dogs. This is in contrast with studies in humans that have shown diminished visceral blood flow during exercise as a compensatory change that occurs with redistribution of flow to muscles. A subsequent study involving foxhounds found different results than the sled dog study and discovered that blood flow to abdominal organs decreased during maximal exercise, similar to humans.[40] The conflicting results may be explained by different blood flow measurement techniques. Blood flow to the splanchnic region of the sled

dogs was evaluated by placing flow transducers around the cranial mesenteric artery and left renal artery. Blood flow of the foxhounds was determined by the use of radioactive labels, which allowed for inclusion of blood flow to abdominal organs from other arteries in addition to the cranial mesenteric and renal. Another variable between the two studies may be effects of ambient temperature on distribution of blood flow. It may be possible that a much cooler ambient temperature influenced splanchnic blood flow to maintain core body temperature in the sled dogs.

Vasoconstriction of inactive tissues and splenic contraction increase oxygen delivery during exercise by increasing blood flow to active skeletal muscle. As previously stated, blood flow to inactive tissues, such as abdominal organs, may be decreased during exercise because of vasoconstriction, and this is mainly a result of alpha-adrenergic influence. Unlike humans, dogs are capable of splenic contraction during exercise as a mechanism of increasing the number of circulating red blood cells and the oxygen-carrying capacity of blood. It is thought that alpha-adrenergic influence also contributes to contraction of the spleen in dogs. One study demonstrated that alpha-adrenergic blockade of dogs undergoing maximal exercise reduced oxygen consumption and increased anaerobic metabolism.[41] The reduced arterial oxygen content was caused by inhibition of splenic contraction of exercising dogs that underwent blockade. The value of splenic contraction in exercising dogs was also demonstrated in splenectomized dogs, which had reduced arterial oxygen content during exercise.[41] Dogs that have undergone splenectomy lack a major adaptation that affects their ability to undergo aerobic metabolism and influences exercise performance.

Cardiovascular System and Vo_2 Max during Exercise

Vo_2 max is the greatest rate of oxygen uptake by the body during extreme dynamic exercise and is the best index of the intensity of an aerobic exercise. Maximum oxygen uptake is the product of cardiac output and systemic oxygen extraction. In humans, increases in Vo_2 max with endurance training are a result of an equivalent increase in both maximal cardiac output (50%) and systemic oxygen extraction (50%).[42] Elevation of cardiac output is influenced by increases in preload caused by increased heart size, increased venous return, and increased myocardial contractility, as well as a decrease in the resistance the heart has to pump against (afterload). Cardiac contractility can increase with sympathetic stimulus of the ventricle, as well as by way of the Frank-Starling mechanism. Afterload can be reduced by decreasing peripheral resistance of blood vessels, especially the arterioles supplying skeletal muscle (Figure 9-4). Increased systemic oxygen extraction can result from increased capillary density in skeletal muscle or increased oxidative capacity of muscle cells. Increased blood flow allows for a greater opportunity of oxygen uptake by muscle cells.

Studies of oxygen uptake in dogs have shown some similarities to studies of humans. Foxhounds undergoing submaximal and maximal endurance exercise have a predictable increase in Vo_2 max (Figure 9-5, *A*). Similar to humans, Vo_2 increases during exercise are attributed to increased cardiac output. The increase in cardiac output in

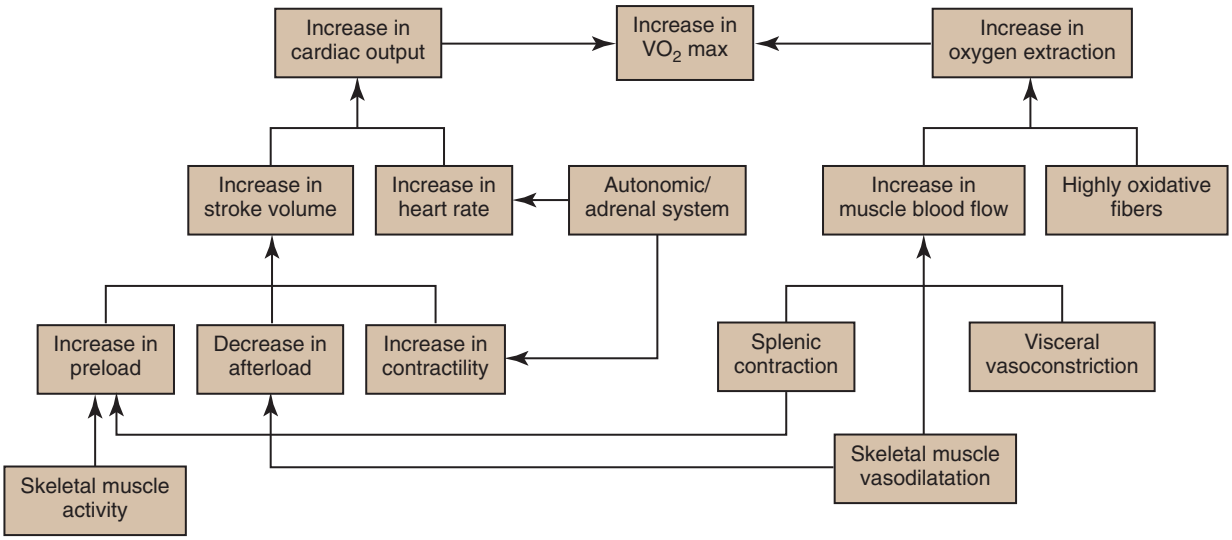


Figure 9-4 Summary of factors affecting Vo_2 max during endurance exercise.

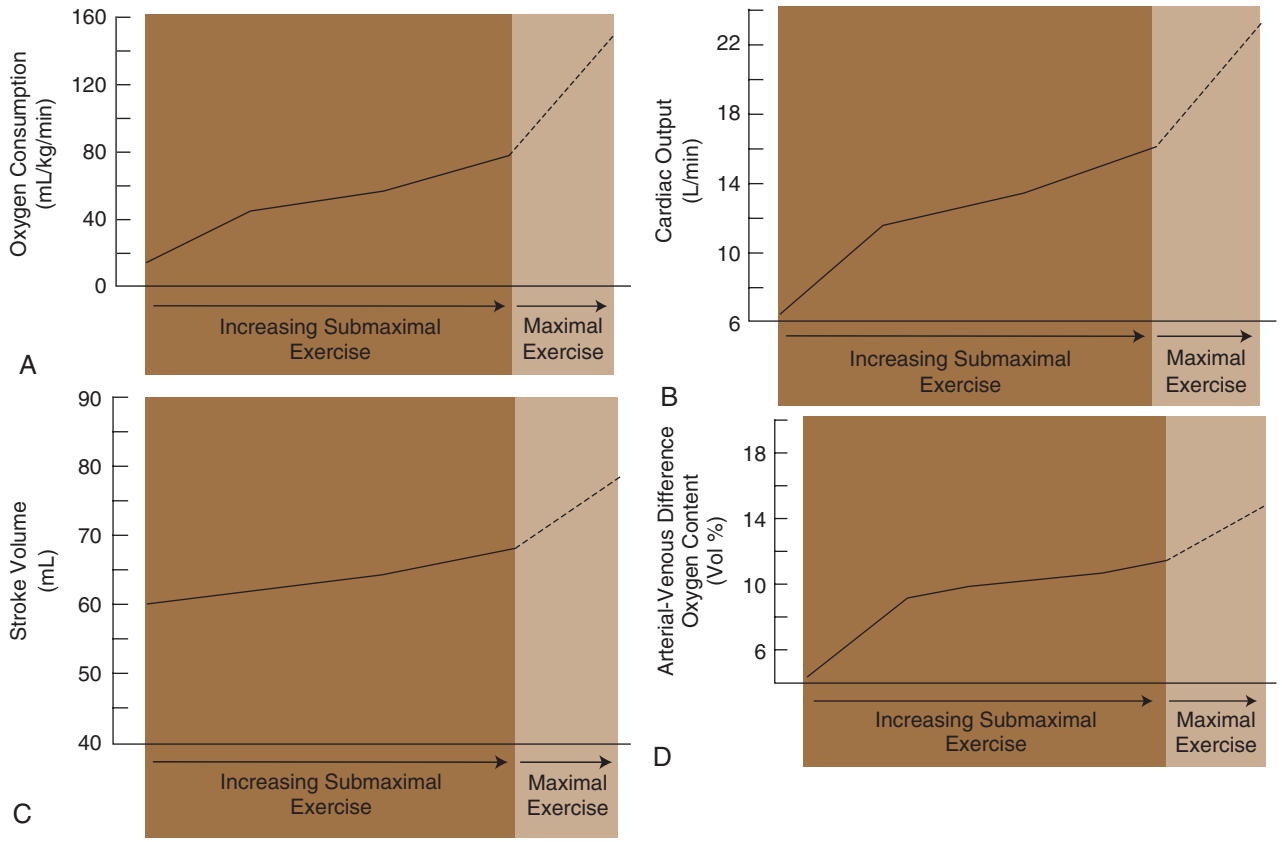


Figure 9-5 **A,** Changes in oxygen consumption of foxhounds as a function of relative work rate. **B,** Changes in cardiac output of foxhounds as a function of relative work rate. **C,** Changes in stroke volume of foxhounds as a function of relative work rate. **D,** Changes in arteriovenous O_2 difference of foxhounds as a function of relative work rate. (Adapted from Musch TI, Haidet GC, Ordway GA et al: Dynamic exercise training in foxhounds. I. Oxygen consumption and hemodynamic responses. *J Appl Physiol* 59:183-189, 1985.)

dogs during exercise is mostly attributed to increases in stroke volume up to approximately 50-70% of Vo_2 max, after which stroke volume levels off during submaximal activity and further increases in cardiac output are mostly due to increased heart rate[34] (Figure 9-5, *B* and *C*). In contrast to humans, changes in systemic oxygen extraction do not significantly contribute to increases in Vo_2 max in dogs after undergoing an endurance training program.[34] This is further supported by minimal evidence that dogs undergo biochemical or histochemical adaptations in skeletal muscle after undergoing endurance training.[12] Vo_2 max elevation in endurance-trained dogs compared with untrained dogs is derived primarily from central changes (increased cardiac output) and not from changes in oxygen extraction. Increased cardiac output accounts for 96% of the increase in Vo_2 max in endurance-trained foxhounds, whereas increased oxygen extraction accounts for only 4% of the increase.[34] Compared with a resting state, dogs engaged in exercise activity have a substantial increase in systemic oxygen extraction (Figure 9-5, *D*). However, when comparing the oxygen extraction of an untrained and trained dog during exercise, there does not seem to be significant evidence to demonstrate pronounced increases after training.

When athletes stop training, their Vo_2 max decreases. This is a result of decreased maximal cardiac output and systemic oxygen extraction. In humans, an initial decrease in cardiac output is due to a decrease in stroke volume from loss of plasma volume mainly as a result of a decrease in plasma protein levels and plasma renin and vasopressin activity, which are upregulated during periods of exercise.[43,44] As a state of detraining continues, Vo_2 max continues to decrease, along with reduced systemic oxygen extraction. In humans, periods of detraining decrease the overall oxidative capacity of skeletal muscles.[45] Because untrained dogs have an oxidative capacity similar to dogs that have undergone endurance training, it is reasonable to speculate that a decrease in Vo_2 max of a previously trained dog that has entered into a period of detraining would mostly be a result of a decreased cardiac output and not the reduced ability of skeletal muscle to extract oxygen.

Exercise Capacity and Age

The age of a dog can affect the ability to exercise. Older dogs have reduced capability for maximal exercise capacity.[46,47] A group of older dogs (10-14 years) showed significantly reduced exercise capacity and maximum oxygen consumption compared with a group of younger dogs (2-3 years) during treadmill exercise.[46] The reduction in Vo_2 in older dogs was mostly associated with reduced cardiac output.[46] Decreased Vo_2 in older exercising beagles was attributed to a 25% decrease in cardiac output and an 11% reduction in systemic oxygen extraction.[46] The relative reduction in cardiac output in older dogs appears to be caused in part by attenuation of heart rate during exercise as well as stroke volume. These findings correlate with a study that demonstrated a reduced maximal heart rate response to isoproterenol in older dogs.[48] A reduction in maximal heart rate of older adult humans during exercise has been demonstrated in numerous studies.[49,50] The reduction in stroke volume in older beagles may be a result of age-related changes to the myocardium, peripheral vasculature, pericardium, or decreased splenic contraction during exercise.[46]

In addition to cardiovascular changes in older dogs, changes in skeletal muscle can have an effect on exercise performance. Aging is associated with a decrease in muscle mass and capillary density. Older beagles have been noted to experience a 16% loss of type II muscle fibers when compared with younger dogs. The total fiber area of type I muscle fibers seems to be maintained, and may even increase in some areas, such as in the semitendinosus muscle.[47] There is also a reduction in muscle capillary density and capillary/fiber ratio in older dogs. Despite these age-related changes at the level of skeletal muscle, blood flow is relatively well maintained during exercise. During maximal exercise in older dogs there is no significant decrease in blood flow to locomotion muscles.[47] A significant decrease in vascular resistance to skeletal muscle and reduction of blood flow to splanchnic circulation seem to allow for maintenance of muscle metabolism during exercise. Despite maintaining blood flow to skeletal muscle during exercise, older dogs have a reduced maximal exercise capacity compared with younger dogs. In summary, the maximal Vo_2 for an older dog is significantly less than that of a younger dog at maximal exercise, but reduction in blood flow to muscle does not appear to be a factor for decreased Vo_2 max in older dogs. Rather, the main causes are reduced cardiac output and oxygen extraction.[46,47] Maintenance of blood flow to skeletal muscle during exercise only partially compensates for age-related changes such as decreased metabolic potential, oxygen content of arterial blood, and cardiac function, which account for the inability of older dogs to reach the same level of Vo_2 max as younger dogs.[47] It should be expected that the exercise performance of dogs will likely decrease with advancing age. Exercise performance may also be more likely to decline in older dogs with the onset of other medical conditions that may develop with advancing age.

Exercise and Nutrition

Nutrition requirements for athletes are different from those who lead a more sedentary lifestyle, and the nutrition requirements of athletic dogs are different from that of human athletes. Compared with humans, canine skeletal muscle contains a high degree of oxidative fibers. The majority of exercise studies on dogs have been performed on either greyhounds or sled dogs. However, information from effects of diet on the performance of athletes, such as sled dogs, has been applied to diet modification of less specialized athletes. One common ingredient in the diet of canine athletes is raw meat. Raw meat by itself is not a balanced diet. It is deficient in essential vitamins and minerals and if fed alone can lead to conditions such as nutritional secondary hyperparathyroidism and increased risk of fracture.[51] Calcium deficiency is the most common imbalance in a raw-meat diet. Addition of 1 g of bone meal to every 50 g of raw meat can correct the deficiency; however, excess supplementation can increase the risk of orthopedic disease, especially in larger immature dogs.[52] Risk of food-borne infection is another concern when feeding a raw-meat diet. Common pathogens such as *Salmonella, Escherichia coli, Campylobacter, Listeria,* and *Shiga*-like toxin produced by *E. coli* O:157:H7 are potential pathogens found in raw meat that can pose a very serious hazard to both dogs and humans.[53] Therefore, raw-meat diets are not recommended for canine athletes.

Daily energy requirement for maintenance depends on body weight, age, exercise, breed, and ambient temperature. Cooler temperatures require less energy for maintenance. However, higher levels of energy are required once the ambient temperature drops below the point at which metabolic rate must increase to maintain body temperature. For thin-coated breeds this temperature is approximately 20° C and for thick-coated breeds critical temperature is less than 0° C.[54] Energy requirements for movement are more proportional to the distance traveled than the speed of travel.[55] It requires more energy for a dog to trot 13 miles than it does to sprint 100 yards at top speed. Certain diets may increase stamina during exercise. Exercise stamina for humans can be increased by high-carbohydrate diets, which increase muscle glycogen levels.[55] Dogs are different than humans in that they metabolize free fatty acids at twice the rate of humans.[56] Skeletal muscles in dogs are more adapted to use fat than humans. A high-fat, low-carbohydrate diet increases the stamina of an exercising dog compared with a diet with only a moderate amount of fat.[57] High-carbohydrate diets increase muscle glycogen levels; however, during short high-intensity exercise, glycogen is used up more rapidly in dogs. Fat oxidation during

both rest and exercise is twice as active in dogs as compared with other less aerobic species.[9]

High levels of protein are another important component of the canine athlete. Anemia can develop in highly athletic dogs fed a low-protein diet. A diet of at least 32% of energy as protein prevents a drop in hematocrit in highly athletic dogs.[58] For sprint exercises, mild restriction of food intake before races to reduce body weight improved sprint performance in greyhounds.[59] Different than endurance racing dogs, feeding an increased protein and decreased carbohydrate diet to sprinting greyhounds resulted in slower performance times. However, when higher levels of fat, along with high protein and lower carbohydrate levels, were fed to greyhounds, race times improved.[60,61] To increase the stamina of working or exercising dogs, canned diets with high levels of fat and protein can be added to a commercial diet before periods of heavy exercise. An alternative is to add beef to a commercial diet that has high levels of energy as both protein and fat.[55] It may be necessary to supplement a dog's diet with components such as fat and protein to meet the energy needs during exercise; however, it is very important that the diet be balanced and complete for all needs of the body.

The components of an athletic dog's diet are important, but the timing of feedings is important as well. Strenuous exercise in untrained dogs delays gastric emptying for more than 1 hour.[62] Feeding of meals should not occur during periods of exercise, and it is best to wait until after a short period of rest.

There is little information on the vitamin, mineral, and other nutrient requirements for athletic dogs. Commercial dog foods contain sufficient amounts of vitamins and minerals for nonathletic dogs. If excessive supplements are added to a commercial dog food, toxicity issues are a possibility.

Temperature

Regulation of body temperature is a critical process to maintain homeostasis and normal function. Metabolic pathways and cellular structures are affected by temperature. Prolonged or excessive increases in body temperature can denature proteins needed for vital metabolic pathways and can even result in death. A decrease in body temperature can slow metabolic pathways to the point that vital body functions are affected. Body temperature is determined by the difference between heat gain and heat loss. Maintenance of body temperature is a highly regulated process. The thermoregulatory center is located in the anterior hypothalamus. It receives information from peripheral thermoreceptors to initiate changes in the body to either increase heat production or to increase loss of body heat. Normal metabolic processes in the body produce heat. At rest, the metabolic rate is low and the production of heat is relatively small. During intense exercise, heat production can be quite large. The body is relatively inefficient at using energy, and a significant portion of energy use for work results in heat production.

During periods of intense exercise it becomes necessary for the body to take measures to increase heat loss to prevent body temperature from rising too high. Heat loss can occur by four different mechanisms: radiation, conduction, convection, and evaporation. *Radiation* is the loss of heat in the form of infrared rays. This process involves the transfer of heat down a thermal gradient from the surface of one object to another with no physical contact. *Conduction* is the transfer of heat from a warmer object to a cooler object that it is in contact with. *Convection* is the loss of heat into either air or water that is in contact with the body. *Evaporation* is the loss of heat as it is transferred to water on a surface of the body and converted into gas. By far the most important method of heat loss for exercising dogs is evaporation. Dogs have poorly developed sweat glands, which are not significant in thermoregulation. Instead, dogs mostly use evaporation as a method of heat loss by way of panting. During exercise there is increased blood flow to the respiratory system, tongue, and oral mucosa, and an increase in saliva and respiratory secretions. Blood has a high capacity to store heat and is very efficient at transporting heat. Exercising dogs take frequent shallow breaths, allowing air to rapidly move over the oral mucosa and respiratory dead-space for evaporation and heat loss.

Effect of Ambient Temperature on Exercise

Despite countermeasures taken by the body, dogs can experience significant increases in body temperature during periods of exercise. Healthy Labrador retrievers performing field trial retrieving exercises for 10-minute periods outdoors can have a marked increase in body temperature.[63-65] In an ambient temperature of up to 28°C, normal healthy Labrador retrievers undergoing strenuous exercise can have temperature elevations up to 42°C.[63,66] An increase of up to approximately 2°C more than resting temperature is common during normal exercise among Labrador retrievers under these types of circumstances. In addition to hyperthermia, dogs typically experience a respiratory alkalosis, hypocapnia, and mild metabolic acidosis when exercising in temperatures ranging from 11-29°C.[63,64] Another study confirmed that Labradors retrievers undergo a significant increase in temperature during strenuous exercise regardless of a cool or warm ambient temperature.[65] However, despite no severe differences in body temperature in strenuously exercising dogs under the previously mentioned range of ambient temperatures, deleterious changes may occur when exercising in warmer environments.

Dogs exercising in an ambient temperature less than 21°C were less likely to develop respiratory alkalosis and hypocapnia. Dogs exercising in an ambient temperature greater than 21°C are very likely to develop respiratory

alkalosis and hypocapnia.[65] In an experimental setting, severe acute hypocapnia results in decreased cerebral and myocardial perfusion. It is uncertain what effect these hemodynamic changes have on healthy dogs during exercise. Several dogs in these studies became uncoordinated and weak during the training exercises; however, they recovered and did not experience any further problems. Sprinting greyhounds show similar dramatic increases in temperature, but in contrast to Labrador retrievers, they tend to have a more pronounced drop in blood pH after racing.[67] The lower blood pH in greyhounds may also be partially explained by the fact that greyhounds experience significantly higher blood lactate elevations during exercise.

It seems that in ambient temperatures between 21-29° C, despite significant increases in body temperature and hemodynamic changes, healthy dogs do not experience significant adverse clinical effects. Although there were no serious adverse clinical effects to the dogs performing retrieving trials in these studies, care should always be taken when exercising dogs, especially in warmer conditions. Sporting breeds such as Labrador retrievers often have a personality (drive to retrieve), which causes them to voluntarily exercise to the point of exhaustion. It is possible that uncontrolled excessive exercise in an environment with a high ambient temperature can lead to adverse effects such as heatstroke. Dogs with underlying conditions (especially respiratory related) would likely be much more susceptible to adverse effects of exercising in warm environments.

Exercising dogs should have access to plenty of water to help avoid possible dehydration as well as an area for the dog to retreat to for a cool down if necessary. If a dog shows signs of significant fatigue, it is advisable to allow for a period of rest before resuming exercise. A study that compared dogs that were cooled with ice packs during exercise with dogs that were not cooled showed that the cooled group had increased exercise performance, and lower heart rates, respiration rates, and blood lactate levels than the noncooled group.[68] Owners and trainers should monitor dogs closely for signs of heatstroke during exercise and seek immediate veterinary attention if necessary.

When exercising for a short period in a cool ambient temperature, changes in cardiac output and blood flow mainly address increasing blood flow to working skeletal muscles. When exercise is sustained long enough to raise body temperature, or when exercising in a higher ambient temperature, blood flow must change to allow for heat loss in addition to supplying the demand of skeletal muscle. Peripheral pooling of blood potentially leading to reduced cardiac output is a cost of cooling for humans exercising in the heat.[69] Humans performing heavy walking exercise in the heat can experience a significantly reduced cardiac output.[70] Perhaps yet another physiologic adaptation of dogs is that exercise in warm environments results in a higher degree of cardiac output.[66] The cooling mechanism of dogs does not involve peripheral evaporation from the skin. Because of this, dogs do not experience peripheral pooling of blood during exercise in heat, and thus do not experience the drop in cardiac output that humans can experience. Dogs seem to experience much larger rates of heat storage compared with humans. It is common for body temperature to reach 42° C in exercising dogs, which does not typically result in adverse effects in healthy animals. This type of exercise hyperthermia is also thought to be an effective method of water conservation during exercise in warm weather. This well-tolerated elevation in body temperature in dogs reduces the amount of water lost to evaporative cooling, which would occur at a higher rate if body temperature were maintained at a lower level during exercise.[71,72] Dogs that experience prolonged periods of extreme inactivity may be more susceptible to development of severe hyperthermia compared with dogs that are active on a routine basis.[73]

Dehydrated dogs that exercise have significantly higher body temperature and lower rates of water lost by evaporation. Cardiac output and carotid blood flow are significantly reduced as a result of decreased plasma volume.[71] When dehydrated, the process of reducing evaporation is a method to save total body water. However, the body temperature of dehydrated animals could become dangerously high. Dogs in a state of dehydration should be withheld from exercise, and ample water should be supplied for exercising dogs to help prevent a state of dehydration.

Adverse Effects of Exercise

Exercise and training are designed to condition the body, enhance the performance of physical activity, and improve the overall state of fitness and health. However, the act of exercise can potentially have adverse effects on the body. The body can certainly become injured as a result of external influences, such as the force of impact on the body, or variables in the environment. However, the act of physical activity alone can unmask an underlying disease or condition or it can directly affect an otherwise healthy body by causing alterations that have the potential to have adverse effects.

Unexpected injury and even death, although unlikely, can happen to dogs that are elite athletes as well as those that lead a more sedentary lifestyle. There is limited information on causes of severe injury or death of dogs during exercise. One study's aim was to evaluate necropsy findings on Alaskan sled dogs that died unexpectedly during competition. The most common postmortem findings included aspiration pneumonia, gastric mucosal lesions, and blood loss as a result of gastrointestinal ulceration.[74] Other findings included rhabdomyolysis, hepatocellular necrosis, and cardiac myodegeneration. The cause

of pneumonia in most cases was aspiration of gastric contents. All dogs with pneumonia had concurrent gastric lesions and it is thought that the lesions found in the gastrointestinal tract play a part in causing vomiting or gastroesophageal reflux, leading to aspiration. Severe injury or sudden death is a rare event and does not occur frequently during periods of exercise.

Serious injury or death during exercise is more likely to be related to an underlying condition that was exacerbated by the demands and stress associated with exercise. It is possible that signs of an underlying condition could go unnoticed during periods of inactivity. Certain conditions can manifest signs for the first time during periods of exercise. During periods of strenuous exercise, Labrador retrievers can be affected by a syndrome called exercise-induced collapse (EIC). This condition is a fairly common cause of exercise intolerance in otherwise healthy young adult Labrador retrievers. Dogs affected by this condition can tolerate moderate amounts of exercise, but may become ataxic and collapse after only 5-15 minutes of strenuous exercise. The cause of EIC has been determined to be a mutation in the dynamin 1 gene.[75] This gene plays a role in synaptic vesicle endocytosis of neurotransmission in the brain and spinal cord. The first signs of this condition are gait abnormalities, such as a wide-based pelvic limb stance along with rocking and loss of balance. If a dog is known to have this condition, exercise should be discontinued immediately if gait abnormalities are recognized. All dogs in one study had loss of patellar reflex after exercise.[76] Other conditions that may first be noticed during exercise and can lead to very serious problems or even death include malignant hyperthermia, hypoglycemia, laryngeal paralysis, myasthenia gravis, cardiac abnormalities, metabolic disturbances, and others.

The gastrointestinal tract may experience abnormal changes during exercise in otherwise healthy athletes. Gastrointestinal disease, such as diarrhea, is a common abnormality in human endurance athletes, including marathon runners. Sustained periods of exercise increase the frequency of gastric erosions and ulcerations.[77] Studies have also shown that in addition to gastric lesions, prolonged strenuous exercise can cause intestinal mucosal damage, increasing intestinal permeability.[78] It seems that gastric mucosal damage can occur at more intense levels of exercise (submaximal and maximal); however, intestinal permeability can also be altered during less intense routine training exercises.[79] It is not known specifically what effect, if any, these lesions have on the performance capability of elite athletes such as sled dogs. Administration of famotidine once daily decreases the severity and perhaps the prevalence of exercise-induced gastric ulcers in dogs.[80] Although the effect of gastric ulcers on performance is unknown, the ease of administration and lack of adverse effects may make the use of famotidine as prophylaxis against gastric ulcers beneficial in performing dogs.

Hematologic and Biochemical Effects from Exercise

Dogs undergo various hematologic and biochemical changes as a response to exercise. Most changes are not severe, and it is often not clear if the changes are truly abnormal or if they are normal physiologic responses to exercise.

Prolonged endurance exercise results in a catabolic state that can cause changes in hydration status, electrolyte alterations, plasma enzyme values, and other hematologic changes. Sprinting and agility dogs can experience changes in blood lactate levels, pH, hematocrit, protein, and potassium levels. However, the changes are not severe and do not appear to have a significant effect on exercise performance.[81,82] Well-conditioned sled dogs develop multiple abnormalities in serum chemistry values, but they are of limited severity.[83] These changes are most likely attributed to adaptation to prolonged endurance training and effects of dietary components such as high amounts of fat and protein. One of the most significant changes in racing sled dogs is the development of hypoproteinemia, mostly caused by hypoglobulinemia.[83] This change may be caused by immunosuppression, elevated protein catabolism, and protein loss in the gastrointestinal system as a result of exercise. Short-duration strenuous activities result in activation of platelets and neutrophils in dogs.[84] Activation of neutrophils is a process that occurs to prime the cells to fight against infections. Activated neutrophils are beneficial in septic conditions. In a nonseptic state, such as during exercise, activated neutrophils may have adverse effects. Activated neutrophils upregulate surface receptors and are primed for release of oxygen radicals and proteolytic enzymes, making them less likely to migrate to extravascular sites and perhaps more likely to lodge in small vessels in body.[85] Neutrophil activation has been documented in humans as well, but there are no data to support adverse effects of neutrophil activation on exercise performance. A concern with activated platelets is development of a hypercoagulable state; however, this state has not been demonstrated in exercising dogs. It is possible that increased fibrinolytic activity balances out platelet activation during exercise. Exercise training can induce a decrease in packed cell volume. This may be explained by plasma volume expansion and blood loss secondary to gastrointestinal tract bleeding.[86]

Chronic exercise training leads to expansion of plasma volume.[87] This may be an adaptive mechanism to provide a larger vascular volume to meet the increased cardiovascular and thermoregulatory needs during exercise. Greyhounds can increase their plasma volume by almost 30% after undergoing a 2-week training period.[88] Studies suggest that the primary reason for plasma volume expansion in endurance dogs is due to an increase in water intake with minimal involvement from renal control.[88]

Agility dogs can increase their hematocrit by 21%, plasma volume by 4%, and total blood volume by 12%

during exercise as a result of splenic contraction.[89] Within minutes of discontinuing exercise activity, the hematocrit decreases as a result of recapture by the spleen. Agility exercise in dogs does not seem to induce changes in red blood cell indices or morphologic findings.[82]

Exercise can also affect hormones such as thyroid hormones. Short-term treadmill exercise and sprinting increase levels of thyroid hormones.[90,91] Prolonged endurance exercise results in substantial decreases in plasma hormone levels (T4 and T3).[92] The effect of changes in thyroid hormone on exercise performance in dogs is unknown. It is important that the finding of abnormal thyroid hormones in an exercising dog does not lead to a potential misdiagnosis such as hypothyroidism.

Lactate concentration in blood increases during periods of exercise. The degree of increase is related to the intensity of activity as well as the index of glycolytic activity in skeletal muscle. Different breeds of dogs partaking in various forms of physical activity experience differences in the degree of lactic acid elevation. Lactate concentration in dogs performing in an agility competition rise to approximately 4.5 mmol/L, which is almost twice the resting value. Fifteen minutes after discontinuing the exercise, lactate levels are not significantly different than resting values.[82] Foxhounds engaged in maximal exercise and Labrador retrievers participating in field trials develop lower concentrations of lactate during exercise at approximately 2.7 mmol/L and 2.5 mmol/L, respectively.[34,64] The difference in lactate levels may be due to different levels of aerobic metabolism between foxhounds and Labrador retrievers, and the dogs participating in the agility competitions, or that the agility competitions are much shorter in duration and are a more anaerobic activity. A more dramatic contrast is seen when comparing other breeds of athletic dogs with greyhounds, which frequently experience a rise in lactate levels close to 30 mmol/L.[81] Short periods of maximal exercise experienced during racing certainly contribute a significant portion of this increase; however, the higher percentage of glycolytic muscle fibers in greyhounds may also explain this breed difference. The blood lactate elevations do not appear to have a negative effect on healthy dogs participating in these types of exercise activities.[34,64,81,82] Exercise can also result in alterations of various serum chemistry values in dogs. Successive days of endurance exercise can cause increases in serum sodium, creatine kinase, aspartate aminotransferase, alkaline phosphatase, and alanine aminotransferase, and a decrease in serum potassium.[83] In healthy dogs these changes are mild and are not of significant clinical importance.[83]

Exercise and Mental Well-Being

Exercise is an important aspect of life for both humans and animals. The association between exercise and physical health is widely accepted in the human medical world.[93] There is growing evidence that exercise has benefits regarding the mental well-being of humans as well.[94] In humans there is evidence that exercise can improve physical self-perceptions, enhance mood states, help treat clinical depression and anxiety, and even improve cognitive function.[94] In addition to the physical health benefits such as weight loss in obese dogs, and increases in strength and endurance, exercise may also have some psychological benefits in dogs.

Collecting information on the mental well-being of dogs is not easily done, leaving perceptions of owners as the only means of determining their well-being. The term *quality of life* is frequently used when attempting to describe the well-being of a dog. The owner, or the individual who spends the most time with a dog, is frequently the one who makes assessments regarding quality of life. Many people would agree that it is easy to see the delight a dog experiences when participating in various forms of exercise, whether it be retrieving, agility, sled racing, and so on. A quality of life screening program of owners evaluated aspects of a dog's life that were thought to be major influences of quality of life. The most common suggestion from owners was that more exercise for their dogs would improve their quality of life.[95] Exercise is not only a beneficial method of improving physical health and implementing therapeutic effects for rehabilitative purposes, it is an experience that often provides great enjoyment for dogs.

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SECTION III

Patient Assessment and Management

10 Orthopedic and Neurologic Evaluation

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Detection and assessment of orthopedic and neurologic disorders may be challenging. Ideally, each person involved in the treatment and rehabilitation of the patient should be able to perform a physical examination for evaluation of orthopedic and neurologic conditions, and should have knowledge of the underlying condition and its management. The evaluator should develop a systematic approach to standardize the physical examination and to prevent omissions of important findings.

History

The patient's age, gender, and breed should be recorded in the medical record. It is helpful to obtain information regarding the patient's general health. The owner should be questioned to determine if there are any signs of systemic disease, such as fever, anorexia, depression, vomiting, or diarrhea. The presence of any preexisting conditions that may produce or contribute to the lameness should be recorded.

Any known previous trauma should be recorded, along with the affected areas that were injured. The travel history of the affected animal or of other pets in the household should be assessed. Certain fungal diseases and some diseases transmitted by ticks, such as ehrlichiosis and Rocky Mountain spotted fever, are endemic in certain geographic areas, and travel to those regions may result in infection and lameness.

The owner or handler should be asked to state the chief complaint, or the reason why the dog is being evaluated. When questioning an owner or handler regarding the affected limbs, it is important to be certain that the correct limb is identified. Sometimes, the owner may be confused and misidentify the affected legs. To eliminate any confusion, the owner should point to the limb that is the source of concern.

The progression of the condition should be addressed, including when it started, changes in severity over time, changes with weather, changes with exercise or rest, and any changes that occur over the course of a typical day. Information regarding any previous diagnoses should also be obtained. The results of any previous treatments should be assessed. In particular, any medications administered should be recorded, along with the dose, dosing interval, length of treatment time, and whether or not the treatment was effective. It is relatively common for inappropriate doses of medications, such as nonsteroidal antiinflammatory drugs, to be administered, and it may be inappropriate to conclude that treatment has been ineffective in the treatment of conditions such as osteoarthritis.

The owner's impression of the degree of pain or lameness should be obtained, and whether there is multiple limb involvement. With regard to the lameness, the following short series of questions may be useful:

- How long has the lameness been present?
- Has the lameness increased or decreased in severity?
- Is the lameness worse in the morning or evening?
- Was the limb subjected to some traumatic event?
- Does the lameness improve or worsen after a few minutes of activity, such as walking?
- Does the lameness change with weather or exercise?
- Have other limbs been involved?
- Have there been any previous related diagnoses or treatment?

During the gathering of historic information it is vital that the examiner also consider that certain neurologic or oncologic problems may mimic orthopedic diseases, and the converse is true as well. Dogs with bilateral cranial cruciate ligament ruptures may have gait and stance problems suggestive of neurologic disease. Oncologic conditions, such as osteosarcoma or multiple myeloma, may present with a lameness that mimics an orthopedic problem.

Some neoplastic conditions may have paraneoplastic manifestations, such as hypertrophic osteopathy, in which a mass in another location may result in periosteal reaction of the bones of the distal limbs and lameness.

General Physical Examination

It is critical that a thorough general physical examination be performed by the attending veterinarian. Information concerning other body systems is vital to determine whether certain forms of therapy may be contraindicated, or if a systemic disease such as Lyme disease or systemic lupus erythematosus may be the underlying cause of a lameness. It is especially important if surgical intervention is possible or if long-term medication is anticipated to be certain that major body systems are healthy and able to tolerate the treatment. In particular, the cardiovascular, respiratory, gastrointestinal, hepatic, renal, and endocrine systems should be assessed to determine if underlying disease conditions are present that may affect the selection and appropriate use of various medications or anesthetic agents. Thorough auscultation of the heart and lungs, abdominal palpation, lymph node palpation, evaluation of mucocutaneous junctions, and assessment of the skin are necessary. Ancillary tests may be needed for more thorough evaluation, including electrocardiogram, complete blood count, serum biochemistry profile, urinalysis, radiographs or sonograms of the thorax or abdomen, and other special tests.

Initial Observation

The dog should be carefully observed at rest and at several gaits before in-depth examination and palpation to avoid artificially accentuating lameness with manipulation of the affected areas. If possible, observe the patient as it arises from a sitting or recumbent position. Often the lameness is more severe immediately after rising and then improves with ambulation; in some instances, the patient may not use the affected limb at all to help rise to a standing position.

With the patient standing, one should observe for weakness, limb trembling, asymmetry of regions of the limbs indicative of muscle atrophy, asymmetry of the head and neck, limb position, and conformation (Figure 10-1). It is common for standing animals to bear less weight on a limb afflicted with lameness. In this situation, the animal may not have the entire foot in contact with the floor. One method to assess the relative amount of weight placed on each limb is for the examiner to place the front feet or the rear feet on the palm of each hand and allow the animal to bear weight on the observer's hands rather than on the floor. This is a semiquantitative method of assessing weight bearing. Another method to assess weight bearing is to use bathroom scales, or more sophisticated devices described in Chapter 13, under each foot to determine the amount of weight placed on each limb at a stance. In many lamenesses there is also marked muscle atrophy of the affected

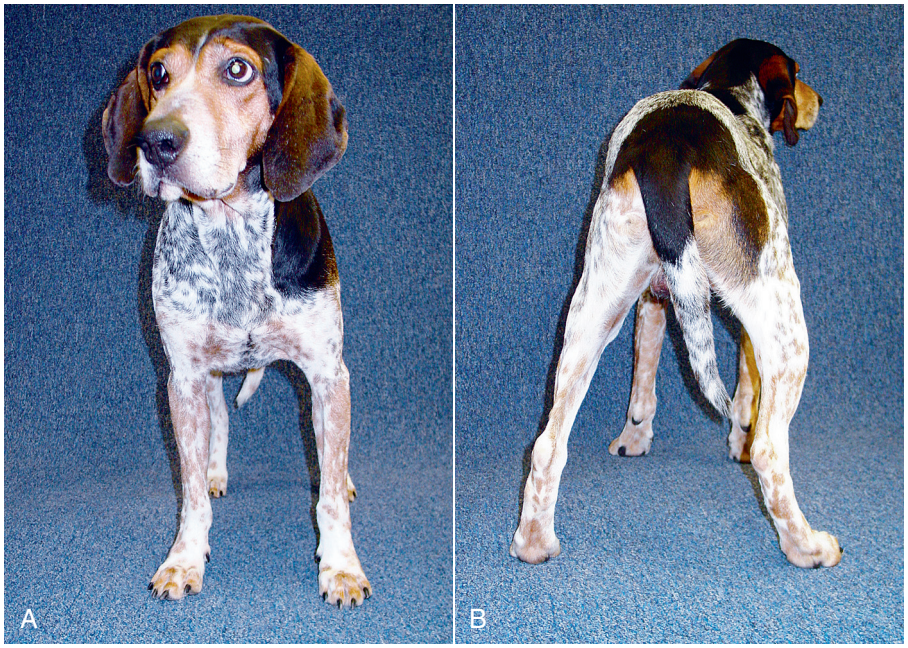


Figure 10-1 The dog should be observed from (*A*) the front and (*B*) the rear while standing for evidence of reduced weight bearing on an affected limb, turning the toes out, and not bearing weight symmetrically in comparison with the contralateral limb. Note the reduced weight bearing on the left rear limb indicated by less contact of the metatarsal pad with the ground and the more lateral position of the foot as compared with the other rear limb.



Figure 10-2 The dog should be observed going (*A*) away from and (*B*) toward the evaluator at both a walk and a trot. (*C*) The dog should also be observed from the side.

Box 10-1 Lameness Score (Applied Separately at a Walk and a Trot)

0: Normal
1: Slight, intermittent lameness
2: Obvious weight-bearing lameness
3: Severe weight-bearing lameness
4: Intermittent non–weight-bearing lameness
5: Continuous non–weight-bearing lameness

leg. In some chronic lamenesses there may be abnormal foot pad wear and unequal weight bearing that may be noticeable.

Gait

Gait evaluation is generally performed after observing the dog rising and at a stance. The patient's gait is best observed in a large, enclosed exercise area from a distance. In addition, it is beneficial to have an area in which observations may be made with the dog off leash. Gaiting begins with the handler moving the patient first at a walk, then at a trot. The patient should be evaluated while moving directly away from, and then toward the examiner (Figure 10-2). The patient should then be evaluated from the side at a distance (Figure 10-2C). Evaluation at a lope or gallop may be performed if the patient is able, but this gait is less useful for evaluation of most lameness because of the speed of limb motion and the fact that these gaits are asymmetric. It may be helpful to capture the gait with a video camera and then evaluate the gait in slow motion. The degree of lameness may be subjectively evaluated using a lameness scoring system to allow comparison of the lameness over time (Box 10-1).

Observation of the patient while it is walking is beneficial for those patients with severe lameness that may be unwilling to trot. Because the walk is a slow four-beat gait, each limb may be separately evaluated. This is particularly beneficial to help differentiate a lameness seen at a trot, in which a diagonal forelimb and hindlimb strike the ground at the same time. Observing at a walk with separate limb strikes helps to determine which diagonal limb is affected in cases of subtle lameness. In many cases, the head will nod up and down if a lameness is present. In general the head will nod up when the affected leg is placed on the ground if a forelimb lameness is present, and the head will nod down when the affected limb is placed on the ground if a hindlimb lameness is present. The reason for this is to reduce weight bearing and force on the lame limb by using the head and neck as a lever arm to shift weight from the painful limb to the opposite end of the body.

The patient should also be evaluated at a trot, which is a symmetric two-beat diagonal gait. Subtle lameness is generally easier to see at a trot because greater forces placed on the lame limb accentuate the lameness. Although moderate forelimb and hindlimb lameness may be differentiated at a walk because the pattern of footfalls is separated, differentiation of forelimb and hindlimb lameness is more challenging at a trot. The reason for this is that diagonal forelimbs and hindlimbs strike the ground at the same time. As an example, consider a head that nods up when the left forelimb strikes the ground. This may indicate a left forelimb lameness. However, it may also be indicative of a left hindlimb lameness, because as the left hindlimb strikes the ground, the head will nod down; as the right hindlimb and left forelimb strike the ground immediately after, the head will nod back up. Careful observation of other features of gait may help distinguish between forelimb and hindlimb lameness at a trot, in conjunction with other features of the physical examination. It may also be helpful to place each major joint through a full range of motion, and then reassess the lameness. Stresses placed on joints as they are flexed and extended may accentuate a subtle lameness.

In addition, other factors such as stride length, limb carriage, joint motion, and side bending of the spinal column are useful in evaluating lameness. The affected limb may have a shortened stride length and reduced

flexion and extension of the affected joints. Most dogs will walk with the normal limb centered under the body during the weight-bearing phase of gait on that limb, whereas a lame limb may be carried further eccentrically during weight bearing. The affected limb may also circumduct during gait. Animals with hip or stifle conditions may have increased lateral flexion of the spinal column toward the affected side in an attempt to advance the limb by using the back, especially if the stride is shortened on that side. In all cases, symmetry of movement should be evaluated. Abnormal proprioception, dragging of the nails, knuckling over on the dorsum of the paw, and hypermetria indicate neurologic disease.

When possible, the patient should be walked and trotted in a large circle in both directions. In many cases, lameness of the inside limb will be accentuated. Stairs and steps may also be helpful in observing lameness and subtle neurologic problems. Dogs may skip up or down steps with the affected limb rather than pushing off the limb to ascend or descend a flight of stairs, or may knuckle over on the dorsum of the paw of a limb afflicted with a neurologic condition.



Figure 10-3 Simultaneous palpation of both hindlimbs for evidence of muscle atrophy and asymmetry.

General Palpation for Symmetry and Atrophy

Following observation of the patient for lameness during ambulation, a brief examination is performed while the dog is still standing to assess symmetry and to assess relative muscle mass and the presence of muscle atrophy. Simultaneous palpation of both forelimbs and then both rear limbs will allow the examiner to detect subtle differences between limbs that are normally symmetric (Figure 10-3). Fractures and neoplasia of the musculoskeletal system are usually obvious. Individual regions are evaluated for swelling, abnormal shape, heat, and sensitivity and pain.

Beginning with the forelimb, the dorsal border of the scapula is palpated to be certain that one side is not higher than the other. The spine of the scapula is assessed for fractures and prominence of the spine of the scapula, which might indicate muscle atrophy if it is readily palpable and differs from the other side. The acromion and greater tubercle of the humerus are palpated next. The distance between the two landmarks and the amount of soft tissue between the two points should be equal on both sides. The shaft of the humerus is palpated next to locate any areas of swelling or muscle atrophy.

The elbow is a difficult joint to assess because many structures are present in a relatively small area, including the medial and lateral epicondyles of the humerus and the flexor and extensor muscles that arise from them, the head of the radius, the region of the medial coronoid process of the ulna, the joint capsule, and other soft-tissue structures in the area. The task is made easier by standing behind the dog and placing the thumbs of each hand on the lateral epicondyles. The fingers are then wrapped around the cranial aspect of the elbow, and the index fingers are placed in the region of the medial coronoid processes of the ulnas. The area of the medial coronoid process is firmly pressed to assess for pain and discomfort, which may indicate a fragmented medial coronoid process. Following palpation of this area, the cranial and caudal aspects of the medial joint are assessed for joint effusion, swelling, and increased or decreased soft-tissue mass. The caudolateral joint compartment is assessed next between the lateral epicondyle and the olecranon process for joint effusion or swelling, which might indicate osteoarthritis or an ununited anconeal process. This swelling is usually palpable caudal and parallel to the lateral epicondyle, and compression forces the effusion medially. By palpating the elbows simultaneously with the dog standing, subtle abnormalities may be detected by assessing the area for asymmetry. It may also be helpful in certain cases to pronate and supinate the distal extremity when evaluating each individual elbow. These maneuvers help to concentrate the forces in the medial or lateral joint compartment, accentuating any subtle discomfort.

The shafts of the radius and ulna are palpated from proximal to distal to assess the bones for any pain or swelling. The carpi and digits are usually best assessed with the dog in lateral recumbency, although the standing angle of the carpus should be observed to be certain that carpal hyperextension is not present.

The hindlimbs are evaluated next. The dorsal aspect of the wings of the iliums are located to be certain that one is not located further cranially or dorsally than the other, which might indicate a sacroiliac luxation. The gluteal muscles are assessed for atrophy throughout the ilium. The area between the greater trochanter and the tuber ischium is located and compared with the other side. Asymmetry may indicate the presence of muscle atrophy on the side that has less soft tissue between the two points, or a hip luxation if the distance between the two points differs. The thigh musculature is evaluated for atrophy. The stifles are simultaneously palpated in a manner similar to that of the elbows to assess for joint effusion, the presence of firm tissue on the medial aspect of the distal femur (often referred to as a *medial buttress*) that might indicate a chronic rupture of the cranial cruciate ligament, and the position of the patellae, which should be symmetric.

The tibias are assessed for any asymmetric swelling or pain, and the angle of the tarsus is observed to evaluate the integrity of the common calcaneal tendons. The tarsus and digits are best evaluated with the patient in lateral recumbency.

Orthopedic Examination

A systematic approach to the orthopedic examination is necessary to reduce the risk of missing a condition and to create an evaluation that can be consistently repeated. In many cases the contralateral limb may be used as a reference for comparison of limb circumference, range of motion, and sensitivity to palpation. Comparison with the contralateral limb may be especially helpful when a subtle condition exists and there is uncertainty regarding an abnormality. Concentrating on anatomy during the examination, and referring to an anatomy book as necessary, helps in confusing or unusual situations. The patient is usually examined in lateral recumbency, but may be evaluated while standing if it is anxious.

Rear Limbs

Evaluation of the rear limbs first may be safer for the examiner while the patient is becoming accustomed to the examiner. The phalanges and metatarsals of the distal limb are evaluated first. The toes are spread apart to evaluate the nail beds, webbing between the toes, and the pads for any infection, trauma, or cracks. The phalanges, interphalangeal joints, metatarsal bones, and metatarsophalangeal joints are individually palpated and assessed for pain, crepitus, swelling, fractures, luxations, or collateral ligament instability (Figure 10-4). The range of motion of the joints is also assessed. The area over the plantar sesamoid bones is palpated for pain and proliferative changes that might be associated with an old fracture or arthritis. Joint stability



Figure 10-4 The interdigital areas, pads, phalanges, and metatarsal bones should be palpated and the joints placed through a range of motion.

is further assessed by flexing and extending individual joints and placing varus and valgus stresses on the joints. It is helpful to be familiar with the origin and insertion of the interphalangeal ligaments to properly evaluate instability (Box 10-2).

The tarsus is a complex series of joints. It is first assessed for joint effusion and instability. Palpation of the dorsal aspect of the tarsocrural joint is especially rewarding in cases of joint effusion. The joint is placed through a full range of motion to assess the joint for limitations, crepitus, and pain. The examiner's finger is used to palpate the tarsal bones, with particular attention to the central, third, and fourth tarsal bones. The tarsus has short and long components of the medial and lateral collateral ligaments that are taut in different degrees of flexion and extension. To assess the long components of the collateral ligaments, valgus and varus stresses are applied while the tarsus is placed in full extension (Figure 10-5). Normally, there should be little varus or valgus movement with the hock extended. The short portions of the collateral ligaments are assessed by placing varus and valgus stresses on the tarsus with the tarsocrural joint flexed to 90 degrees. Assessment of these structures while the hock is flexed is more difficult because there is some normal motion in this position. After stabilizing the metatarsus with one hand and the tuber calcis with the other, dorsal and plantar stresses are placed on the hock to determine if subluxation is present or if there has been damage to the supporting plantar structures. With the stifle maintained in an extended position, the hock is flexed to evaluate the calcaneus and common calcaneal tendon. Normally, there should be little ability to flex the tarsus. Excessive flexion may indicate that there has been a fracture of the calcaneus or damage to the common calcaneal tendon. It is also helpful to

Box 10-2 Common Conditions of the Hindlimbs

Common Conditions of the Digits, Pads, and Feet
Paronychia
Lacerations of the interdigital skin or foot pads
Luxations of the interphalangeal joints
Luxations of the tarsometatarsal joint
Fractures of the metatarsal bones
Fractures of the phalanges
Fractures of the sesamoid bones

Common Conditions of the Tarsus
Osteochondritis dissecans of the talus
Fracture of the calcaneus
Fracture of the central tarsal bone
Fracture of the third or fourth tarsal bones
Luxations of the tarsus with damage to the short or long tarsocrural ligaments
Osteoarthritis
Disruption of the common calcaneal tendon
Rupture of the plantar ligaments

Common Conditions of the Tibia and Fibula
Fractures of the tibia and fibula
Panosteitis
Traumatic periostitis
Osteosarcoma

Common Conditions of the Stifle
Cranial cruciate ligament disruption
Osteochondritis dissecans of the femoral condyle
Injuries to the medial and lateral collateral ligaments
Medial and lateral patellar luxation
Medial and lateral meniscal damage
Fractures
Congenital absence of the patella
Caudal cruciate ligament disruption
Osteoarthritis

Common Conditions of the Femur
Fracture of the femur
Panosteitis
Osteosarcoma

Common Conditions of the Coxofemoral Joint
Hip dysplasia
Fracture of the acetabulum
Fracture of the femoral head or neck
Fracture and separation of the proximal capital femoral physis
Legg-Calvé-Perthes disease
Traumatic luxation of the coxofemoral joint
Osteoarthritis

palpate the insertion of the tendon on the calcaneus to rule out partial avulsion injuries, which may be manifested as a firm swollen area. It is valuable to compare the suspected limb with the contralateral limb to assess subtle findings.



Figure 10-5 The tarsus is evaluated for evidence of medial or lateral collateral ligament damage by applying varus and valgus stresses to the joint.



Figure 10-6 The tibia is palpated for evidence of pain, swelling, or inflammation.

Palpation of the tibia is relatively easy because the medial aspect has little muscle covering. The metaphyseal and diaphyseal regions are palpated for periosteal or bone pain, which might indicate hypertrophic osteodystrophy, panosteitis, fractures, neoplasia, or traumatic periostitis (Figure 10-6). It is helpful to palpate the medial and lateral malleoli distally and the head of the fibula proximally.

The stifle joint is frequently afflicted with orthopedic conditions. Sedation may be necessary in some larger patients that are tense. The joint is initially assessed for swelling. The tibial tubercle is located first, and the path of the patellar ligament is traced proximally. The joint is palpated medial and lateral to the patellar ligament. Normally, the ligament feels like a pencil and the medial and lateral edges can be distinctly felt, but with joint effusion, the patellar ligament is less distinct and fluid may be palpable in cases of moderate to severe joint effusion. The femoral condyles and the region of the trochlear ridges are palpated next. In some cases, osteophytes or joint capsule thickening may be palpable. Thickening of the medial

aspect of the distal femur is often present in dogs with chronic rupture of the cranial cruciate ligament. This is easily felt by placing the examiner's hand and fingers across the stifle so that the fingers rest over the medial surface of the joint. The other stifle may be simultaneously palpated to compare the two limbs. Swelling of the craniolateral joint in a young dog might indicate the presence of an avulsion of the long digital extensor tendon, although this condition is rare. The stifle is flexed and extended to evaluate for crepitus, grating, clicking, or snapping, which might indicate osteoarthritis, a damaged meniscus, or joint instability. The examiner's hand may be placed on the patella to assess crepitus. Often dogs with a partial rupture of the cranial cruciate ligament or chronic osteoarthritis secondary to cranial cruciate ligament rupture have marked pain with full stifle extension.

The patella is assessed for medial or lateral luxation. To test for medial patella luxation, the stifle is extended, the distal limb is internally rotated, and pressure is applied to the lateral aspect of the patella to try to displace it medially (Figure 10-7). While maintaining pressure, the limb is slowly flexed and extended to see if luxation occurs with the limb in a position other than full extension. Medial patella luxation is common in toy and miniature breeds and in larger breeds such as Labrador retrievers. In small dogs, the patella may be difficult to locate if patella ectopia exists. In these cases, the tibial tubercle is located and the path of the patellar ligament is traced proximally until the patella is located. To test for lateral patella luxation, the stifle is placed in extension or slight flexion while the distal limb is externally rotated. Pressure is applied to the medial aspect of the patella, trying to force it laterally. Although medial patella luxation is most common in all breeds, if a lateral patella luxation is present, it is most likely a large dog or a chondrodystrophic breed.

Instability of the medial collateral ligament is assessed with the stifle in extension. While holding the distal femur with one hand and the proximal tibia with the other, a valgus stress is placed on the tibia. In normal dogs, little motion occurs with this maneuver. Lateral collateral ligament integrity is assessed by holding the limb as if checking for medial collateral instability, but a varus stress is placed on the tibia.

Injury to the cranial cruciate ligament is common, and this structure should be carefully evaluated. Large dogs may be tense and require sedation for a thorough examination. The most common method of assessing rupture of the cranial cruciate ligament rupture is the cranial drawer test (Figure 10-8). To perform this test, place one index finger on the tibial tubercle and stretch the soft tissues caudally with the thumb until the head of the fibula is reached. Place the other index finger on the patella and stretch the soft tissues with the thumb until the lateral fabella is palpated. Now place the stifle in mild flexion and quickly and gently try to move the tibia cranially and caudally (slide the drawer open and closed). The examiner should avoid internally rotating the limb while performing the drawer maneuver. Because the cranial cruciate ligament also limits excessive internal rotation and hyperextension, in addition to cranial drawer, excessive internal rotation or



Figure 10-7 To evaluate the stifle for medial patella luxation, the stifle is extended, the tibia is internally rotated, and an attempt is made to push the patella medially out of the trochlear groove.



Figure 10-8 Cranial drawer motion is assessed by placing one forefinger on the patella, one thumb on the lateral fabella, the other forefinger on the tibial tuberosity, and the other thumb on the head of the fibula. An attempt is then made to slide the tibia cranially in relation to the femur, while holding the femur in a steady position.

hyperextension may be helpful in diagnosing injury to the ligament. In some cases, partial drawer may be detected. This may result from a partial cranial cruciate rupture, a torn meniscus that has become wedged between the tibia and femur, or a chronic cranial cruciate ligament rupture with periarticular fibrosis providing partial stability of the stifle. In some cases of partial cranial cruciate ligament rupture, there may be drawer motion in flexion but not extension.

An indirect method of evaluating the integrity of the cranial cruciate ligament is the tibial compression test (Figure 10-9). The stifle is flexed to an approximately 90-135-degree angle. One hand is placed on the stifle with the forefinger resting on the tibial tuberosity. The tarsus is slowly flexed with the other hand while keeping the stifle at a fixed angle. This causes tension in the gastrocnemius muscle and common calcaneal tendon that compresses the femur and tibia. In the absence of the cranial cruciate ligament, cranial movement of the tibial tuberosity in relation to the distal femur is detected.

Injury to the caudal cruciate ligament is rare and is usually due to trauma. Other structures are also commonly injured, such as the cranial cruciate ligament, the medial collateral ligament, or the medial meniscus. To assess the caudal cruciate ligament, the limb is held as for testing cranial drawer, but the tibia is displaced caudally rather than cranially. Diagnosis of tears of the caudal cruciate ligament can be difficult and requires excellent knowledge of the spatial anatomy of the stifle joint. The integrity of the medial and lateral collateral ligaments are assessed by holding the stifle in an extended position while placing valgus and varus stresses on the joint.

The femur is evaluated next. The quadriceps, biceps femoris, semimembranosus, and semitendinosus muscles are palpated for atrophy, swelling, and pain. Although muscle tears are rare in breeds other than sight hounds, the tensor fascia lata and gracilis muscles should be examined. The shaft of the femur is palpated between muscle bellies, being careful not to confuse bone pain with pain from pinching muscles (Figure 10-10). Bone pain and swelling in the distal femur may indicate osteosarcoma in a large breed, whereas pain in the diaphysis of the bone in a skeletally immature dog may indicate panosteitis.

The coxofemoral, or hip joint is also commonly afflicted with orthopedic conditions. The hip joint should be assessed for luxation in dogs with acute lameness of a rear limb as a result of trauma. To do this, fingers are placed on the tuber sacrale, greater trochanter, and tuber ischium (Figure 10-11). The greater trochanter should be below a line drawn from the tuber ischium to the tuber sacrale; if not, then a hip luxation should be suspected. Next, a thumb is placed in a notch between the greater

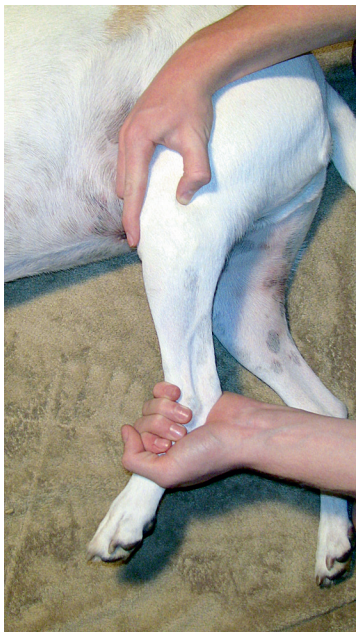


Figure 10-9 The tibial compression test is a method to indirectly create cranial drawer motion. To perform this maneuver, the stifle is placed in a slightly extended walking position and firmly maintained in this position. The examiner places a forefinger on the tibial tuberosity and the thumb and remaining fingers on the medial and lateral condyles of the femur. The other hand is used to flex the hock. The forefinger on the tibial tuberosity detects any cranial shift of the tibia in relation to the femur as a result of an insufficient cranial cruciate ligament.



Figure 10-10 The femur is palpated for any pain, inflammation, or swelling, while minimizing excessive compression of the muscles.

trochanter and the tuber ischium, and the femur is externally rotated. With a normal hip, the thumb will be displaced out of the notch as the femur is rotated. With a hip luxation, the thumb is not displaced because the greater trochanter is typically displaced cranially and dorsally and does not rotate caudally to displace the thumb from the notch. Another method of evaluating the possibility of a hip luxation is to measure the length of the affected leg. The affected leg will be shorter with the hip extended and will be longer with the hip flexed as compared with the normal contralateral leg. It should be noted that fractures of the femoral neck and capital femoral physeal fractures may give similar physical examination findings.



Figure 10-11 The relationships of the dorsal border of the wing of the ilium, the greater trochanter of the femur, and the ischiatic tuberosity are established. A triangle should be formed when these structures are outlined. In this photo, the thumb is on the tuber ischium, the forefinger is on the greater trochanter, and the middle finger is on the dorsal border of the wing of the ilium. If the greater trochanter is in a line with the dorsal wing of the ilium and the ischiatic tuberosity, a craniodorsal luxation of the coxofemoral joint may be present.

Next the hip should be placed through a range of motion, including flexion, extension, and rotation of the hip joint to determine if pain, crepitus, or decreased range of motion exist. In most cases, dogs have full hip flexion with little or no pain. Patients with osteoarthritis of the hip have pain with hip extension. Subluxation of the hip commonly occurs in young dogs with hip dysplasia. The Ortolani sign may be performed to determine whether subluxation of the coxofemoral joint is present (Figure 10-12). To perform this test, the dog is placed in lateral or dorsal recumbency. The palm of one hand is placed over the region of the sacrum to support the dog, and the fingers of that hand are placed on the greater trochanter and rim of the acetabulum. The femur is grasped with the other hand, and the femur is adducted and pushed dorsally. The examiner may feel the femur displace from the acetabulum. The femur is then slowly abducted while palpating for a change in the relation of the greater trochanter and acetabulum. If the hip was subluxated, a distinct "clunk" will be felt as the hip spontaneously reduces itself as the femur is abducted. An Ortolani maneuver may be performed in a standing dog by

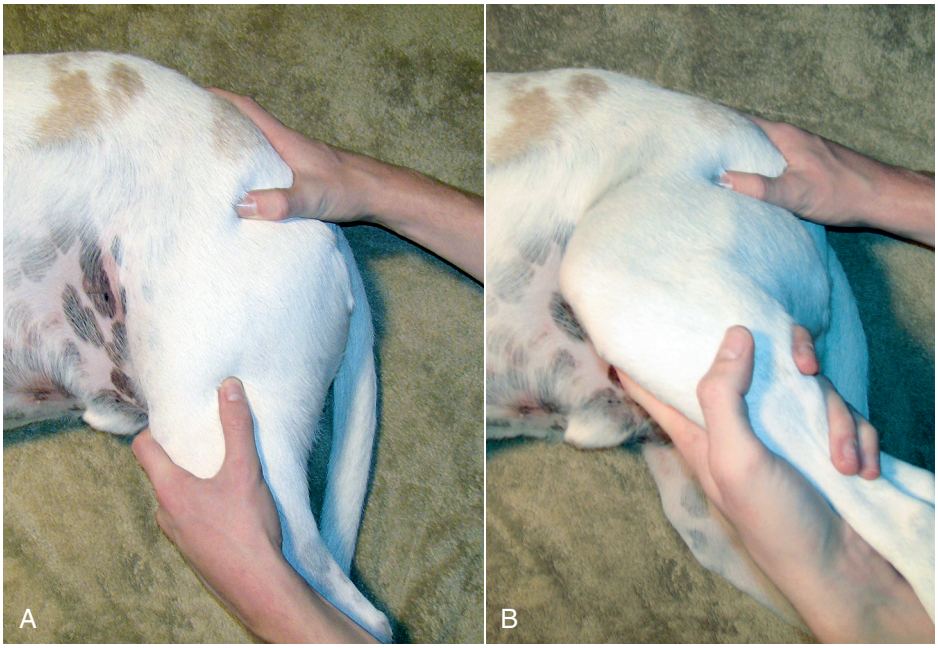


Figure 10-12 **A,** To perform the Ortolani test, the patient is placed in lateral recumbency, the palm of one hand is placed over the area of the pelvis, the thumb or forefinger of that hand is placed on the greater trochanter, and the femur is adducted while the femur is pushed dorsally with the other hand. **B,** The femur is then abducted, and if subluxation of the hip has occurred, the reduction will be felt with the thumb or forefinger on the greater trochanter as it is reduced.



Figure 10-13 The phalanges are flexed and extended to determine if any pain or decreased range of motion is present. Here, the digits are flexed as a group.

placing the dog's pelvis and sacrum against the examiner's chest and using one hand to create a dorsal force while adducting the hip.

Forelimbs

Evaluation of the digits, pads, interdigital skin, metacarpal bones, phalangeal joints, and metacarpophalangeal joints is similar to that conducted in the rear limbs (Figure 10-13 and Box 10-3). Careful digital palpation on the dorsal surface of these bones may reveal point pain or tenderness often associated with stress periostitis.

The carpus is evaluated next. The antebrachiocarpal, middle carpal, and carpometacarpal joints are assessed for effusion. The areas of each carpal bone are individually palpated for pain, swelling, and discomfort that may indicate chip fractures or other injuries. The carpus is placed through a complete range of motion to assess for restricted motion, pain, or crepitus. Normally, the carpus should flex so that the pads on the bottom of the foot contact the caudal aspect of the antebrachium. The carpus should extend approximately 10 to 20 degrees beyond vertical (overextension). The collateral ligaments of the carpus are evaluated by placing varus and valgus stresses with the carpus in full extension. There is normally more varus and valgus motion in the carpus than in the other hinge-type joints. The carpus should be placed in a normal walking position and pressure placed on the foot to evaluate for carpal hyperextension syndrome, which is associated with damage to the palmar fibrocartilage and intercarpal ligaments (Figure 10-14). In early hyperextension injuries, there may be pain and reduced flexion of the carpus. The accessory carpal bone should be evaluated for fractures or changes associated with chronic osteoarthritis.

The radius and ulna are assessed for angular limb deformities, and their entire lengths are palpated for pain and swelling. The examiner should palpate the medial and lateral styloid processes. Swelling and pain in the metaphyseal region may indicate hypertrophic osteodystrophy in a young, large-breed dog. Pain and swelling in this region in an older dog may indicate the presence of

Box 10-3 Common Conditions of the Forelimbs

Common Conditions of the Digits, Pads, and Feet

Paronychia
Lacerations of the interdigital skin or foot pads
Luxations of the interphalangeal joints
Luxations of the carpometacarpal joint
Fractures of the metacarpal bones
Fractures of the phalanges
Fractures of the sesamoid bones
Stress periostitis
Periosteal bone formation associated with hypertrophic osteopathy

Common Conditions of the Carpus

Fractures of the accessory, radial, ulnar, or numbered carpal bones
Carpal hyperextension injury
Flexural contracture of the carpus
Luxation of the antebrachiocarpal joint
Collateral ligament injuries
Stress tendinitis of the ulnar lateralis
Osteoarthritis

Common Conditions of the Radius, Ulna, and Elbow

Fragmented coronoid process of the ulna
Ununited anconeal process
Osteochondritis dissecans of the medial aspect of the humeral condyle
Incongruity of the elbow joint
Fracture of the lateral and medial condyle of the humerus
Luxation of the elbow joint, usually lateral
Panosteitis
Hypertrophic osteodystrophy
Osteoarthritis
Fracture of the radius and ulna

Common Conditions of the Humerus

Fracture of the humerus
Panosteitis
Osteosarcoma

Common Problems of the Scapulohumeral Joint

Avulsion fracture of the supraglenoid tubercle
Luxation of the scapulohumeral joint
Fracture of the scapulohumeral joint
Biceps tenosynovitis
Medial glenohumeral ligament instability
Mineralization of the supraspinatus tendon
Contracture of the infraspinatus muscle
Osteochondritis dissecans of the caudal head of the humerus
Osteoarthritis



Figure 10-14 The carpus is placed through a range of motion. With the limb placed in a walking position, dorsal pressure is placed on the bottom of the foot to test for carpal hyperextension syndrome.



Figure 10-15 The radius and ulna are palpated. If pain is elicited in the midshaft of the radius in a skeletally immature large-breed dog, panosteitis is suspected.

osteosarcoma. Lameness and pain on palpation of the diaphyseal region of the radius or ulna in a young large-breed dog may indicate panosteitis (Figure 10-15).

The elbow is a difficult joint to assess because three bones are articulating together, and many soft-tissue structures originate in the region of the elbow. It is helpful to simultaneously palpate both elbows to detect subtle differences between them, such as mild joint effusion (Figure 10-16). The range of motion in flexion and extension is determined, and the joint is evaluated for crepitus. Any loss



Figure 10-16 Simultaneous palpation of both elbows allows for the detection of subtle abnormalities, such as mild joint effusion. The placements of hands and fingers are essentially mirror images of each other on the right and left elbows. Effusion of the caudolateral compartment in a 6-month-old large-breed dog may indicate the presence of an ununited anconeal process, whereas effusion of the craniomedial joint compartment may indicate a fragmented medial coronoid process or osteochondritis dissecans of the medial condyle of the humerus.

of motion, especially flexion, almost always indicates the presence of osteoarthritis. The medial and lateral humeral epicondyles and heads of the radius and ulna are palpated. The collateral ligaments of the elbow joint are evaluated with the elbow fully extended. The distal extremity is grasped and varus and valgus stresses are applied to evaluate the collateral ligaments. The elbow and carpus are then flexed to 90 degrees and the distal limb is internally and externally rotated. With this maneuver, it is possible to internally rotate the distal extremity and apply digital pressure on the medial portion of the joint. In many dogs with medial coronoid problems, pain may be elicited. Swelling in the caudolateral aspect of the elbow may indicate joint effusion associated with an ununited anconeal process, while swelling on the medial aspect of the elbow joint may indicate osteochondritis dissecans of the medial humeral condyle or a fragmented medial coronoid process of the ulna. With digital pressure, the effusion may be displaced, but the effusion reappears after the digital pressure is released.

The humerus is palpated for swelling and pain that may result from panosteitis or neoplasia. Caution should be exercised in interpreting pain when palpating the region of the radial nerve (Figure 10-17). Direct pressure on the



Figure 10-17 Caution should be exercised when palpating the distal humerus to be certain that compression of the radial nerve by the examiner is not causing pain and discomfort. The general area of the radial nerve is indicated by the finger.

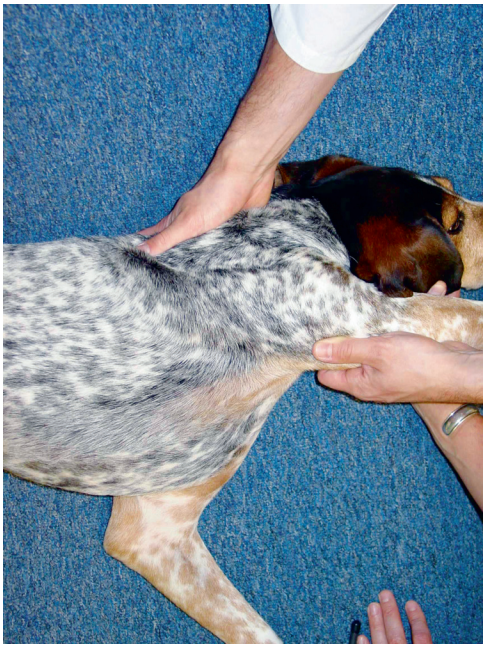


Figure 10-18 Pain caused by extension of the shoulder joint in a skeletally immature large-breed dog is consistent with osteochondritis dissecans of the caudal head of the humerus.

nerve may elicit a painful response unrelated to any pathologic finding of the humerus. Anatomically, an imaginary line drawn along the dorsal border of the antebrachium and continued to the humerus with the elbow held in mild flexion will approximate the location of the radial nerve as it crosses over the humerus.

The scapulohumeral or shoulder joint should be flexed and extended. The relationship of the acromial process to the greater tubercle of the humerus should be palpated. An abnormality of this relationship may indicate a shoulder luxation, either medial or lateral. In the absence of trauma, pain on extension of the shoulder joint of a young dog is almost pathognomonic for osteochondritis dissecans of the caudal aspect of the humeral head (Figure 10-18). Similarly, pain may be elicited in a dog with osteochondritis dissecans if the shoulder joint is simultaneously flexed and internally rotated. Pain may be elicited with simultaneous flexion of the shoulder joint and extension of the elbow if biceps tenosynovitis is present (Figure 10-19). This maneuver places increased stress on the biceps tendon as it traverses the bicipital groove in the proximal humerus. Direct palpation of the biceps tendon in this region, located cranial and medial to the greater tubercle, may also elicit pain.

In addition, varus and valgus stresses should be applied to the shoulder to check the collateral support. Increasingly, medial glenohumeral ligament instability is being recognized. The examiner uses the thumb and forefinger of the same hand to grasp the acromion process and place



Figure 10-19 Pain caused by shoulder flexion and simultaneous extension of the elbow is likely due to biceps tenosynovitis. Direct palpation of the region of the biceps tendon just medial to the greater tubercle of the proximal humerus may also be painful.

medial pressure on it to prevent movement of the scapula.[1] The examiner then grasps the limb at the level of the distal humerus using the opposite hand. With the elbow and shoulder held in extension, the shoulder joint is abducted to its physiologic limit (Figure 10-20). Normal dogs have an abduction angle of 30-35 degrees, whereas dogs with medial glenohumeral instability have increased abduction



Figure 10-20 Shoulder abduction is performed to detect the presence of medial glenohumeral instability. Note that the shoulder and elbow joints are held in extension while the shoulder is abducted.

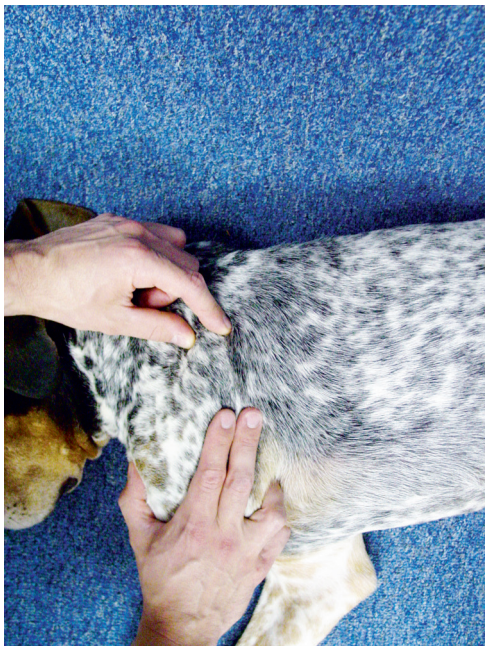


Figure 10-21 Palpation of the scapula is performed to evaluate for fractures or, rarely, luxation of the scapula.

and usually pain at the end range of abduction. Dogs with chronic lameness of a forelimb, unrelated to medial glenohumeral instability, with muscle atrophy may have increased shoulder abduction, but do not usually have pain at the end range of abduction. In some dogs with chronic scapulohumeral instability, it is possible to elicit a drawer motion of the humerus in relation to the scapula.

The scapula should be palpated for fractures or pain, especially the spine of the scapula (Figure 10-21). The body of the scapula should be moved in a dorsal direction to determine if a scapular luxation exists, although this is a rare condition.

Other Tips

In general, the best systematic approach to the orthopedic examination is to examine the normal limbs first and the affected limb last. In many cases subtle problems in other limbs may be masked or not detected if attention is initially placed on the most obvious problem. Also, many animals become tense throughout the body if they experience discomfort as a result of palpating a painful area on the affected limb, and a thorough, accurate examination may no longer be possible. Tranquilization and sedation should be avoided until after the initial examination has been completed so that discomfort during palpation is not masked. Similarly, it is best to avoid antiinflammatory drugs for 24 to 48 hours before examination. Diagnostic joint blocks should also be considered. These can be helpful in prioritizing problems if more than one joint is involved, such as in osteoarthritis of the tarsocrural joint and chronic rupture of a cranial cruciate ligament. Tranquilization may facilitate examination of large, athletic dogs in which muscle tone or an anxious patient results in difficulty in assessing certain aspects of the examination, such as an Ortolani sign or cranial drawer motion.

Arthrocentesis

In some cases, arthrocentesis of one or more joints may be performed by a veterinarian. The joint should be identified and will typically have some effusion or an increased quantity of synovial fluid. The area should be clipped and prepared using aseptic technique. A 20- or 22-gauge needle and 3-mL syringe are used. The affected joint should be flexed and extended several times before arthrocentesis. This helps to dislodge any bacteria from the synovium if an infection is suspected. The needle is inserted without the syringe attached, and the veterinarian waits for synovial fluid to drop into a small EDTA tube. The needle should be positioned downward to take advantage of gravity to encourage the flow of synovial fluid. This avoids direct aspiration of the joint and iatrogenic hemorrhage. If no synovial fluid drips from the needle, then the syringe is attached and gentle aspiration is attempted. The fluid should be cultured if sepsis is suspected. Two samples are obtained, one for immediate culture, and the other for culture in blood culture media for 24 hours. This will allow the recovery of relatively low numbers of bacteria to be detected. In many cases, only several drops of fluid may be obtained, precluding a full cell count and evaluation. However, cytology may be performed to determine relative cellularity and the distribution of cell types. Synovial fluid analysis is most useful to differentiate inflammatory and noninflammatory joint disease. Table 10-1 indicates the most common general joint conditions and the characteristics of synovial fluid analysis.

Table 10-1 Synovial Fluid Changes in Canine Arthritis

Condition	Nucleated Cells/mm^3	Differential Mononuclear Cells	Differential Neutrophils
Normal	250-3000	94-100	0-6
DJD	1000-5000	88-100	0-12
Erosive (rheumatoid-like)	8000-38,000	20-80	20-80
Nonerosive	4400-371,000	5-85	15-95
Septic	40,000-267,000	1-10	90-99

DJD, Degenerative joint disease.
From Piermattei DL, Flo GL: Brinker, Piermattei, and Flo's handbook of small animal orthopedics and fracture repair, ed 3, Philadelphia, 1997, WB Saunders.

Neurologic Examination

Dogs suspected of having a neurologic condition should have a complete neurologic evaluation. In addition, during evaluation of small animals for lameness or other musculoskeletal abnormalities, the neurologic system must always be examined because some "lamenesses" are actually neurologic conditions. A complete neurologic examination should be performed to help determine the contribution of the nervous system to the condition being evaluated, and to help identify other problems that may be occurring concurrently. In addition, a neurologic evaluation should be performed in trauma patients, and owners should be made aware of conditions before undertaking any treatment of traumatic musculoskeletal conditions. A neurologic examination should be performed in a systematic, sequential manner. By always performing a neurologic evaluation in a consistent fashion, the examiner can ensure that all aspects of the nervous system are evaluated and that nothing is omitted. The neurologic examination should always be performed in a calm, quiet place with minimal distractions. It is important that the patient not be sedated or tranquilized before the examination to help minimize erroneous interpretations of pain sensation or reflexes.

General Neurologic Examination

The neurologic examination begins when an animal is first presented to the hospital. The general appearance of the patient may initially be evaluated at the time the history is taken, even before a physical examination is performed. Behavior, posture, and voluntary movements should be noted as the animal is allowed to freely move around the examination room. During this time, the interactions between the patient and owner should be observed, as this can give valuable information about the patient's mentation. An animal's behavior may be normal, aggressive, excited, apathetic, or depressed. A change in mentation should be noted because this may indicate abnormal brain function or other problems. Posture and voluntary movement may also indicate abnormal forebrain or brainstem function, but may also indicate a spinal cord lesion or other neurologic conditions. For an animal to have normal posture, normal peripheral sensory inputs must be processed in the higher centers of the brain including the cerebrum, the cerebellum, and the brainstem. Conditions such as head tilts, tremors, or falling to one side may indicate a problem in any one of these locations.

Nonambulatory animals may be paretic or paralyzed. Either condition causes obvious and markedly abnormal posture in patients. Paretic animals cannot walk without assistance, and although patients can still voluntarily move their legs, limb motion is weak. A lesion causing paresis is considered less severe than one causing paralysis in which voluntary movement is no longer possible. Although paresis may be caused by many conditions, it is often a sign of a less severe lesion than would be seen in a paralyzed animal. If a paretic lesion worsens, the condition often progresses to complete paralysis. The importance in differentiating paresis from paralysis comes into play when determining prognosis. When examining quadrupeds with spinal lesions, the examiner should keep in mind that paresis and paralysis do not always affect all four limbs equally. When an animal is unable to use or move its limbs, it is said to be *paralyzed.* When an animal is unable to walk because of paresis in all four limbs, the term *tetraparesis* is used. *Hemiparesis* and *paraparesis* are terms used to describe paresis on one side of the body or of the hindlimbs, respectively, while other limbs have normal function. These presentations are typically seen when a lesion affects nerves to a particular limb without affecting the innervation of other limbs, or when a lesion affects only one side of the spinal cord. A typical example is a thoracolumbar spinal fracture leading to paraparesis or paralysis of both hindlimbs while forelimb function remains unaffected.

After the history is recorded and the general appearance of the animal is evaluated, the patient's gait should be evaluated if the patient is capable of ambulating and if trauma to the vertebral column is not present. A harness should be used for any patient in which head or neck lesions are suspected. In preparation for this aspect of the evaluation, a handler is instructed to gait the animal at a

walk and a trot. Although an ataxic or circling animal may not need to be trotted, other conditions such as hypometria or hypermetria may not be evident until the speed of the gait is increased. Gait evaluation should always be performed on a surface with adequate traction and should involve evaluation while the patient ambulates toward and away from the examiner. Several passes at a walk and trot should be performed to detect any minor deficits that may be present. Special attention should focus on each limb and not just the limb or limbs in question to help ensure that all four limbs are properly examined. Some patients that have severe ataxia or a tendency to circle can be difficult to walk in a straight line. It may be beneficial in these patients to allow them to ambulate freely around a large confined space to more adequately characterize their gait.

After behavior, posture, voluntary movements, and gait have been assessed, the physical examination is initiated. Palpation allows the examiner to inspect muscles, bones, and other soft tissues for subtle abnormalities. Palpation is performed in a systematic way beginning at the head, moving down the neck, and proceeding to the trunk and extremities. Again, by following the same procedure for each patient, the examiner minimizes omissions and is able to perform a consistent evaluation. One side of the body is compared with the other to evaluate symmetry. In patients with one side or limb more severely affected, it may be beneficial to palpate and examine the more normal side or limb first. Careful attention should be paid to muscle size, tone, and strength. Deviation of joints or limbs, abnormal body carriage, or worn toenails could indicate a neurologic problem. Because intervertebral disk herniation and other forms of spinal cord trauma may be painful, the presence of pain during physical examination not only may help identify possible differential diagnoses for a condition, but also may help to localize the lesion.

Spinal Reflexes

Spinal reflexes are used to test the sensory and motor components of a reflex arc. In general, reflexes may be (1) decreased or absent (hyporeflexia), indicating either partial or complete loss of sensory or motor nerve function to a particular region of the body (lower motor neuron [LMN] disease); (2) normal, indicating that no neurologic abnormalities exist within the reflex arc; or (3) increased or exaggerated (hyperreflexia), indicating that the inhibitory neurons from the brain and spinal cord cranial to the LMNs have been affected (upper motor neuron [UMN] disease). In general, spinal reflexes are graded using the following scheme: absent (0); decreased (+1); normal (+2); exaggerated (+3); or very exaggerated or clonus (+4).

Myotatic Reflexes

Myotatic reflexes are stretch reflexes that cause muscle contraction after the muscle is stretched. These are local reflex arcs that are used for body posture and movement and do not rely on cerebral input for function. The reflex arc consists of a sensory neuron that responds to the stretch of a muscle, and a motor neuron that in turn causes muscle contraction. In general, if only one limb is affected, a peripheral nerve injury is suspected, whereas a spinal cord lesion usually produces bilateral deficits. The most reliable myotatic reflex is the patellar reflex. This reflex is performed with a relaxed patient in lateral recumbency and the pelvic limb held in a slightly flexed position. With the knee supported, the patellar ligament is briskly tapped with a pleximeter (Figure 10-22). As long as the leg is not already in full extension, suddenly stretching the patellar ligament evokes a reflex arc that causes sudden extension of the stifle. Typically, if this reflex is diminished or absent, an LMN lesion is localized to spinal cord segments L4 to L6 (femoral nerve) or to peripheral sensory or motor nerves. If hyperreflexia is present, a UMN lesion involving the spinal cord cranial to L4 is suspected.

The cranial tibial reflex is tested with the animal in the same position as for the patellar reflex. The pleximeter is used to strike the muscle belly of the cranial tibial muscle, eliciting flexion of the hock. This reflex is localized to the L6 to L7 segments of the spinal cord (peroneal branch of the sciatic nerve). The gastrocnemius reflex can then be tested while the animal is still in lateral recumbency. The hock can be slightly flexed, and either the gastrocnemius tendon can be struck or the muscle belly can be struck eliciting extension of the hock. This reflex involves the



Figure 10-22 A patellar reflex is elicited with the patient relaxed and in lateral recumbency by tapping the patellar ligament with a pleximeter.

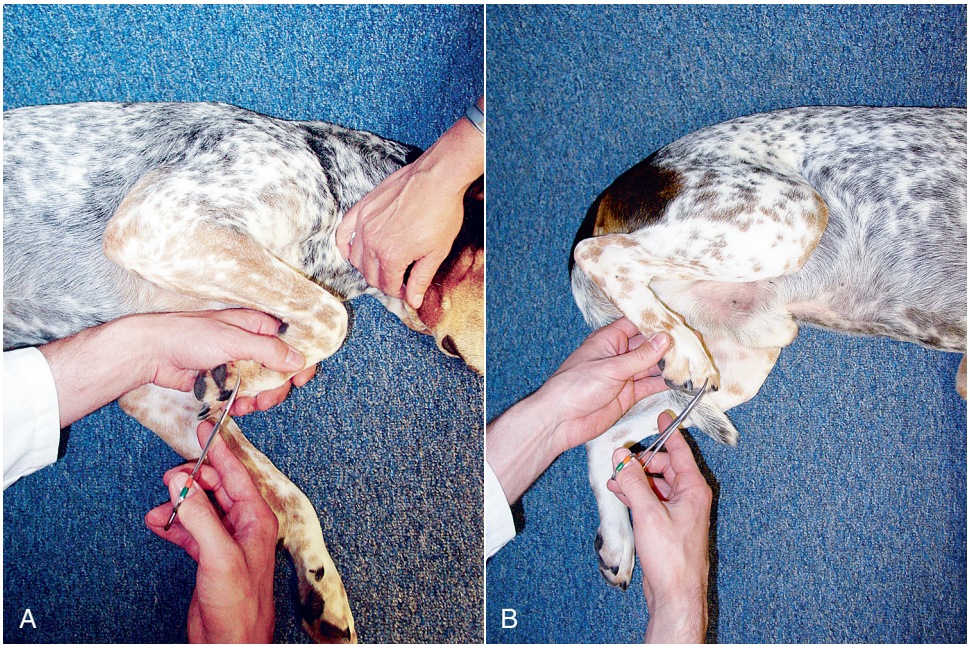


Figure 10-23 Eliciting a flexor reflex of a forelimb (*A*) and a hindlimb (*B*). Note flexion of the shoulder, elbow, and carpus in the forelimb, and the hip, stifle, and hock in the rear limb.

L7-S1 segments of the spinal cord (tibial branch of the sciatic nerve). The cranial tibial and gastrocnemius reflex are much less reliable than the patellar reflex. If these reflexes are present, the reflex arc is intact. However, the inability to obtain a reflex does not necessarily indicate that there is no intact pathway.

In the thoracic limb, the reflexes tested include the extensor carpi radialis, triceps, and biceps reflex. For the extensor carpi radialis reflex, the limb is held with the elbow in slight flexion and the carpus slightly flexed as well. The muscle belly is struck, which should cause extension of the carpus. This reflex is localized to spinal cord segments C7-T1 (radial nerve). The triceps reflex also tests these segments. The limb is held in the same position and the triceps tendon is struck just proximal to its insertion, resulting in contraction of the muscle. Finally, the biceps reflex is tested by extending the elbow while palpating the biceps tendon just proximal to its insertion. The pleximeter is used to strike the examiner's finger, resulting in contraction of the biceps muscle. This reflex involves the segments from C6-C8 (musculocutaneous nerve). Myotatic reflexes in the thoracic limbs can be difficult to interpret, as many normal animals have decreased responses. The flexor reflex gives the most information about nerve function in this area.

Flexor Reflexes

Flexor reflexes are used to evaluate nerve function in both the thoracic and pelvic limbs. These reflexes are useful in the normal animal to help prevent injury from noxious stimuli and to allow withdrawal of the limb away from a noxious stimulus. Briefly, the animal is positioned in lateral recumbency and the limb is positioned in a neutral position. The animal should be relaxed and comfortable. A noxious stimulus is applied to the foot, stimulating sensory neurons (Figure 10-23). Only the amount of stimulus required to elicit a response should be used. Initially, the skin on a toe or between toes is pinched. If no response is elicited with a mild stimulus, a hemostat placed across the nail bed of a toe is used before concluding that there is a negative response. Both medial and lateral aspects of the foot should be tested. The motor pathways of flexor reflexes are more complex than those of myotatic reflexes because motor neurons for all flexor muscles in the affected limb are activated and the entire limb is flexed. An example is a flexor reflex of the pelvic limb of a dog, which causes flexion of the hock, stifle, and hip.

Autonomous Zones

Because specific peripheral sensory nerves innervate various portions of the limbs, the peripheral nerves and their spinal cord segments may be evaluated to determine the level of injury. In the thoracic limb, the dorsal surface of the foot is innervated by the radial nerve (C7-T1), the medial surface of the antebrachium is innervated by the musculocutaneous nerve (C6-8), and the caudolateral surface of the foot is innervated by the ulnar nerve (C8-T2). In the pelvic limb, the most medial toe is innervated by the saphenous nerve, a branch of the femoral nerve (L4-L6), the plantar surface is innervated by the tibial

branch of the sciatic nerve (L7-S1), and the rest of the foot is innervated by the peroneal branch of the sciatic nerve (L6-L7). Variable responses to noxious stimuli in different regions of the limb help to localize the lesion to a particular region.

Crossed-Extensor Reflex

The crossed-extensor reflex is a normal reflex in a standing animal. The reflex causes extension of one limb when the contralateral limb is flexed. In a recumbent animal, this reflex is normally inhibited by descending inhibitory pathways. To elicit the reflex, the animal is placed in lateral recumbency, and the flexor reflex is evoked from the upper limb. Extension of the contralateral limb while the patient is in lateral recumbency signals a lesion in the inhibitory pathways or pathologic condition of the UMN.

Perineal Reflex

The perineal reflex is elicited by tactile stimulation of the perineal region. It is important to test both the left and right side, as some lesions may cause only unilateral signs. The normal response of this reflex consists of contraction of the anal sphincter (anal winking) and ventral flexion of the tail. The sensory and motor components of this reflex are innervated by the pudendal nerve; thus an absent or decreased response indicates a lesion of the pudendal nerve or sacral spinal cord segments S1-S3. Lesions in the sacral spinal cord or pudendal nerves also often result in decreased urethral sphincter tone and an LMN bladder. This reflex can also be noted when taking the temperature of the patient during the initial physical examination.

Cutaneous Trunci Reflex

The cutaneous trunci reflex (sometimes called the *panniculus reflex*) is a relatively complex reflex elicited by applying a noxious stimulus to the skin, which stimulates the superficial spinal nerves innervating a particular region or autonomous zone (dermatome) of the dermis and elicits a motor response, which is seen as a skin twitch. Typically, fingers or hemostats are used to pinch the skin and dermis just lateral to the dorsal midline, beginning in the area of the sacrum and moving cranially until a response is elicited. Where this response starts varies between patients, but usually is in the mid to caudal lumbar region. When the cutaneous trunci reflex is stimulated, a reflex arc is initiated, with the afferent nerves of that particular region of the skin stimulated and a signal transmitted to the spinal cord. The signal is transmitted cranially to the C8-T1 spinal cord segments, where the nerves synapse with the efferent nerves of the lateral thoracic nerve. The impulse causes contraction of the cutaneous trunci muscles to elicit a skin twitch. Spinal nerve roots to autonomous zones travel from cranial to caudal; therefore this reflex usually tests spinal cord segments or nerves approximately two vertebral bodies cranial to the noxious stimulus. Thus a deficit in the cutaneous trunci response at a specific location indicates a spinal cord lesion approximately two vertebral bodies cranial to the site of the skin pinch. The cutaneous trunci reflex is most reliable in the middle of the back. Testing over the cervical, caudal lumbar, and sacral regions is far less reliable.

Sensation

The presence or absence of sensation in each appendage must be assessed. Unless peripheral nerve injury is suspected, pain sensation is usually absent only when voluntary motion is also absent. This typically occurs in the presence of severe spinal cord injury. In general, if an animal can move its limbs, sensation is usually present. In paralyzed animals the presence or absence of pain sensation is very important because absence of deep pain sensation may indicate severe and often irreversible spinal cord damage.

Deep pain sensation is assessed concurrently with the flexor reflexes. Animals with severe damage to the spinal cord cranial to the nerve cell bodies and peripheral nerves of the tested flexor reflex may still have an intact flexor reflex, but may not transmit the signal to the brain for the interpretation of pain. Intact pain perception is indicated by a cerebral response to the pain, such as vocalization, turning the head toward the stimulated limb, or an attempt to move away from the stimulus. In very stoic patients, the response may be seen as a cessation of panting or movement of the eyes. The absence of deep pain perception usually indicates severe spinal cord damage, but it does not indicate whether the lesion is UMN or LMN. The presence of a hyperreflexic or hyporeflexic flexor reflex indicates whether the lesion is UMN or LMN. It must be emphasized that a patient may still retain a positive flexor reflex, but have loss of deep pain sensation. This situation occurs relatively commonly in dogs with serious intervertebral disk extrusion in the thoracolumbar region. Therefore a conscious response must be obtained to determine if the pain pathway is intact.

Postural Reactions

Postural reactions are used by animals to maintain an upright position and normal body posture. Under normal circumstances, an animal knows the position of each limb in space and will appropriately shift its weight to avoid falling. Postural reactions involve spinal reflexes, as well as input from higher centers, such as the brain and spinal cord, to maintain proper body position. Several postural reactions should be tested during a complete neurologic examination. Although these tests involve different limbs, all test the integration of brain, spinal cord, and peripheral nerves. Abnormal postural reactions indicate a neurologic abnormality, but provide the examiner with limited information regarding the specific location of the lesion. Other portions of the neurologic examination will help to localize



Figure 10-24 Hopping performed to assess postural reactions in a forelimb.



Figure 10-25 Wheelbarrowing to assess proprioceptive function of the forelimbs.

the lesion. Common postural reaction tests are described in the following sections.

Hopping

Hopping may be performed on thoracic and pelvic limbs. The animal is supported, and weight is placed on one limb with the other limbs supported so they bear no weight (Figure 10-24). The animal is then moved laterally in a smooth, quick fashion. The animal should hop on the limb to prevent falling and collapse. Caution should be used in animals with muscle weakness because these animals might have a normal postural reaction to hopping, but may not have the muscle strength to support their weight. In these situations, the animal may be supported to allow hopping, even if it is weak. In larger dogs hopping may be tested by supporting a thoracic limb completely and hopping the animal in the thoracic region using the pelvic limbs as a pivot. This technique allows for adequate testing of the response, without the examiner having to fully support the patient's weight. This can be repeated in the pelvic limbs, using the thoracic limbs as the pivot point.

Wheelbarrowing

Wheelbarrowing is performed by lifting the caudal abdomen and pelvic limbs of the dog and allowing weight bearing on the forelimbs (Figure 10-25). In this position, a dog with normal neurologic function should be able to move forward as the dog is pushed forward. Muscle weakness may affect wheelbarrowing and postural reactions, requiring some support.



Figure 10-26 Hemiwalking.

Hemiwalking

The hemiwalking test is performed by lifting ipsilateral forelimbs and hindlimbs of the patient, forcing weight bearing on the other two contralateral limbs (Figure 10-26). The dog is moved laterally and should be able to walk on two legs without falling. Weak animals may require some support.

Conscious Proprioception

Conscious proprioception is used to test an animal's awareness of limb position in space without visual input. It is unnatural for a dog to walk or stand on the dorsum of the paw. To test conscious proprioception, a knuckling test is



Figure 10-27 Conscious proprioception is evaluated by knuckling over the dorsum of the paw. A neurologically normal patient will quickly right the paw to a proper position.

performed on each of the four limbs. With the animal supported, the paw is picked up and turned over so that the dorsum of the paw contacts the ground (Figure 10-27). Generally, normal animals immediately pick up the paw and place it into a normal position. Normally, a patient will not stand on the dorsum of the paw to support weight. If the paw is not properly replaced, a neurologic deficit is usually present. In general, animals with mild neurologic lesions may have proprioceptive deficits, but may not show other obvious abnormal signs; in some cases, motor activity may remain relatively normal until the lesion progresses.

Tactile Placing

Tactile placing tests are performed by covering the eyes, lifting the dog off of the ground, and bringing the forelimbs toward a table or stable surface. After sensing the surface, the normal response to a tactile placing test is to immediately place both forelimbs onto the surface in a weight-bearing position. A normal tactile placing response relies on normal sensory and motor pathways of the thoracic limbs. Also, normal cerebrocortical function is required to receive and process the information.

Visual placing tests are performed in the same manner as a tactile placing test except that the patient's eyes are not covered. During this test, it is normal for a suspended animal to reach for a surface even before touching it. Although this test still relies on a normal cerebral cortex and intact motor function to the thoracic limbs, sensory input is via the visual pathways rather than the tactile sensation of the paw.

Cranial Nerve Examination

A brief cranial nerve examination should be performed during a neurologic evaluation. Animals possess 12 cranial nerves (Table 10-2). These nerves are mainly involved with functions of the head, and abnormal function may indicate a brain lesion. As with other neurologic tests, cranial nerve examination should be performed in a systematic manner and usually does not add more than a few minutes to the examination. Cranial nerve examination is briefly discussed; the nerves evaluated are indicated in parentheses after each portion of the test.

The patient's head is inspected for muscle atrophy and abnormal jaw tone (cranial nerves VII [facial nerve] and V [trigeminal nerve], respectively). While the eyes are covered, the skin around the ears, eyes, and nose is stimulated to elicit a twitch or other facial movement (cranial nerves V and VII). The lateral and medial canthus of each eye is stimulated to elicit a blink response. A menace response is elicited by quick hand movements toward each open eye. Caution must be used in this interpretation, as an air current created by the examiners hand has the potential to stimulate a response as well as if the examiner unintentionally touches the whiskers of the patient. In adult animals, anticipation of ocular injury should cause momentary closure of the affected eye (cranial nerves II [optic nerve] and VII). The menace response is a learned response and is generally not able to be elicited until approximately 10-12 weeks of age. A pupillary light response is tested by shining a bright light into each eye while monitoring for pupillary constriction (cranial nerves II and III [oculomotor nerve]). Direct and consensual pupillary light responses should be tested. To elicit a physiologic nystagmus response, the patient's head is moved from side to side. The fast phase of the nystagmus should be in the direction of the movement (cranial nerves III, IV [trochlear nerve], and VI [abducens nerve]). The mouth is then opened and the gag reflex is tested (cranial nerves IX [glossopharyngeal nerve] and X [vagus nerve]). At the same time, symmetry of the tongue should be observed, which also can occur when the patient licks (cranial nerve XII [hypoglossal nerve]). Palpation of the neck muscles for the presence of atrophy helps to evaluate cranial nerve XI (accessory nerve). The sense of smell may be tested by monitoring for a response to a food odor such as a favorite treat positioned near the nose (cranial nerve I [olfactory nerve]), although this reflex is rarely tested.

Interpreting the Neurologic Examination

After the neurologic examination is complete, the results of each individual test should be reviewed to help determine the presence or absence of a neurologic abnormality and to help the examiner to localize the lesion. In general,

Table 10-2 Cranial Nerves and Their Functions

CN I: Olfactory nerve	Sensory pathway for the sense of smell
CN II: Optic nerve	Sensory pathway for vision and pupillary light reflexes
CN III: Oculomotor nerve	Motor pathway to all extrinsic ocular muscles except the lateral rectus, retractor bulbi, and dorsal oblique muscles
	Motor pathway for pupillary constriction
CN IV: Trochlear nerve	Motor pathway to the dorsal oblique muscle of the eye
CN V: Trigeminal nerve	Motor pathway to masticatory muscles
	Sensory pathway to the mouth and face
CN VI: Abducent nerve	Motor pathway for the lateral rectus and dorsal oblique muscles of the eye
CN VII: Facial nerve	Motor pathway to the muscles of facial expression
	Sensory pathway for taste to the rostral two thirds of the tongue and the palate
CN VIII: Vestibulocochlear nerve	Vestibular portion—Proprioceptive role for equilibrium and posture
	Cochlear portion—Sensory role in hearing
CN IX: Glossopharyngeal nerve	Sensory and motor innervation for swallowing and the gag reflex
	Sensory pathway for taste to the caudal third of the tongue
CN X: Vagus nerve	Sensory and motor innervation for swallowing, the gag reflex, laryngeal function, and vocalization
CN XI: Spinal accessory nerve	Motor pathway to the muscles of the neck
CN XII: Hypoglossal nerve	Motor innervation to the muscles of the tongue

CN, Cranial nerve.

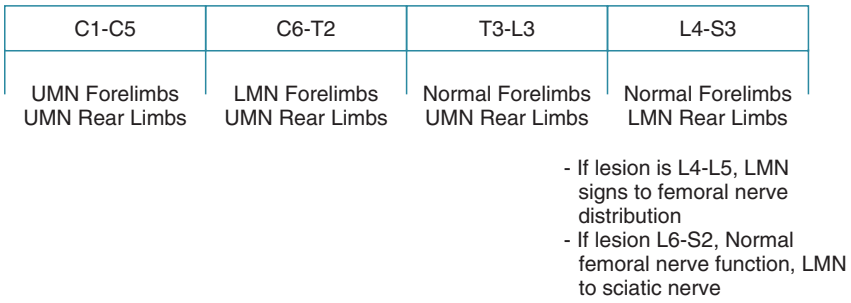


Figure 10-28 Appearance of upper motor or lower motor neuron signs associated with lesions of various spinal cord segments or their nerve roots.

animals with progressive neurologic disease initially have decreased proprioception, followed by loss of motor function, and, in severe cases, a patient may progress to loss of superficial and finally, deep pain. If patients recover, pain sensation generally returns first, followed by return of some motor function, and with near-complete recovery, proprioceptive function returns last.

Interpretation of the neurologic examination is important to localize the lesion so that additional diagnostic tests can be performed to evaluate a particular area. The hallmarks of LMN signs are hyporeflexia, weakness, and reduced resting tone of muscles, and a decreased ability to generate tone with limb movement. The indications of UMN signs are hyperreflexia, maintenance of strength, increased resting tone of muscles, and spasticity of the limbs.

Lesions of spinal cord segments or nerves of C1-C5 result in UMN signs to all four limbs (Figure 10-28). In some cases, the clinical signs may be more obvious in the hindlimbs because the spinal tracts to these limbs are located more peripherally in the ventral spinal cord, where compression by a space-occupying mass, such as a protruding disk, may occur. Lesions of spinal cord segments or nerves of C6-T2 will cause LMN signs to the forelimbs and UMN signs to the hindlimbs. The forelimbs will be normal if the lesion is caudal to T2. Lesions between T3 and L3 will result in UMN signs to the hindlimbs. Lesions located from L4 to S3 will result in LMN signs to the hindlimbs.

Ancillary Tests

Thorough orthopedic and neurologic examinations help to isolate the area of interest to a smaller region. This ultimately saves time and expense and will likely result in a more accurate diagnosis. Ancillary tests to aid

in a definitive diagnosis include radiographs, magnetic resonance imaging (MRI), computed tomography (CT), myelography, electrodiagnostics, and nuclear medicine studies.

Radiographs are the most commonly used test. It is important to always take at least two views at 90-degree angles. Most commonly, lateral and cranial-caudal views are obtained of the region of interest. Comparison films of the opposite limb may be useful to evaluate subtle lesions. Occasionally, stress views of the tarsus or carpus may be helpful. Specialty clinics and university teaching hospitals may have the capability to perform advanced diagnostic tests such as MRI or CT. These studies may give enhanced resolution and information, particularly of soft tissue structures, and of the three-dimensional nature of conditions. Nuclear studies may indicate regions of inflammation and metabolic activity in soft tissues or bone, but are available in only a few locations.

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11 Canine Locomotion Analysis

Robert L. Gillette and T. Craig Angle

It is important to understand the internal and external factors of locomotion. A complete clinical assessment and rehabilitation program depend on an in-depth knowledge of the biomechanics of locomotion. Biomechanics is the study of the mechanics of a biological system and includes kinematic and kinetic principles. Kinematics refers to motion, whereas kinetics involves the forces associated with motion. Locomotion has been compared with a symphony orchestra in that "all parts must blend into a harmonious pattern—from the gentle sway of the head and tail for balance to the coordinated efforts of each limb and muscle to accomplish its special function. Conversely, also like an orchestration, if all movements are not attuned to the whole, a major fault should be evident."[1]

Locomotion

Previous biomechanical research has enhanced our understanding of canine locomotion and thus warrants an in-depth description.[2] The two primary types of locomotion are gait and nonrepetitive motions. Gait is a series of repetitive motions that include walk, trot, pace, gallop, and swim. Nonrepetitive motions do not occur in sequence and include single events such as jumping, movement initiation, and sitting. Nonrepetitive motions have received some focused research attention and are important to understand for clinical evaluations because forces and structural stresses can be at their greatest levels during these motions. By understanding the dynamics of gait and nonrepetitive motions, clinicians can better diagnose and treat injuries and diseases.

Locomotion requires a synergistic effort of the bones, joints, and neuromuscular systems to move, stop, and stabilize the body in the presence of sensory input. Locomotion depends on sensory input, and the sensory input determines how various static and dynamic responsibilities of the musculoskeletal systems are assigned. The musculoskeletal system is constantly distributing body weight, absorbing forces, maintaining posture, maintaining balance, and transferring the center of gravity over the base of support. The ability to properly carry out these static and dynamic responsibilities is what produces efficient and fluid locomotion. Any dysfunction in one joint or segment of the skeletal system or any disruption of the neuromuscular system may result in an inability to carry out certain dynamic and static responsibilities. Asymmetries may develop between the angular displacements of joints, which produce discrepancies in timing and displacement of the limbs. This may lead to inefficient locomotion and/or lameness.

The skeletal segments and joints of the quadruped are repetitively accelerated, decelerated, and loaded with various force magnitudes. Depending on the geometry of the articular surface and the tissues surrounding a joint, and the load involved, each joint moves through a specific range of motion. There is a coordinated activation of neuromuscular components to rhythmically act on skeletal segments to produce fluid and symmetric motion about each joint. Neuromuscular components must produce concentric, eccentric, and isometric muscle contractions in a synergistic fashion. These muscle contractions must also be at optimal lines of action to apply forces to skeletal structures for efficient propulsion and force absorption. Skeletal structure position is directly influenced by the neuromuscular system and must maintain optimal joint angles and joint velocities to facilitate locomotion. Any alteration in motion of one joint by external devices, disease, injury, inflammation, or surgery may disrupt the flow and fluidity of locomotion.[3] Also, joint motion can be indirectly altered psychologically by stimuli such as pain. All of the aforementioned alterations can be present in any quadruped, such as the dog.

Locomotion can be affected by the internal factors previously mentioned and by certain external factors. First, movement can be affected by the textural properties (e.g., vegetation and soil content) of the surface. A hard, soft, smooth, rough, and/or slippery surface affects movement by influencing traction and sensory input. Second, the angle or incline of a surface and the direction (i.e., up or down the incline) in which the dog is traveling affects movement and locomotion. Third, the curvature of a path such as running around a turn can directly influence the forces experienced by the dog. A curvilinear path subjects the dog to centrifugal and centripetal forces that the dog must withstand and compensate so as to stay balanced and continue with a sound gait. Fourth, a fluid environment can

affect movement by applying resistance to motion and buoyancy on the body. Finally, on a treadmill, the base of support passes under the center of gravity, whereas the center of gravity passes over the base of support overground. The specific way in which a treadmill affects neuromuscular patterns has not been studied, but its opposition in relation to support during overground locomotion is worth pointing out. Thus, there are both internal and external factors to consider when evaluating locomotion.

Gait

The gait describes a particular series of joint, segment, and whole-body movements used for locomotion. The temporospatial dynamics of each gait determine its type such as a swimming stroke,[4] walk, trot, pace, or gallop. The temporospatial dynamics of a gait can be influenced by surface properties, surface incline, ground movement (i.e., treadmill), submersion in water, and whether or not the dog is traveling in a linear or curvilinear path.

A gait is made up of a series of repeated strides. Within a stride, each limb goes through a step cycle. A step cycle includes a stance phase and a swing phase (Figure 11-1). The stance phase is the period in which the foot is in contact with the ground. The first part of the stance phase occurs when the braking forces take place as a result of surface contact. This is followed by the second part of the stance phase in which there is a period of propulsion. The swing phase is the period in which the foot is in the air. It is divided into three parts. The leg first swings backward as a result of the propulsive action; the muscles then swing it forward for locomotion, and then bring it backward and down as it returns to the ground. Each one of these phases applies different forces on the limb's bony structure and musculature. Variations in these phases help the clinician when diagnosing lameness.

A stride is defined as the cycle of body movements that begins with the contact of one foot and ends when that foot again contacts the ground. There are various forms of gait that are defined by how the body moves during each stride.[5,6] Symmetric gaits include the walk, trot, and pace. The trot is a two-beat diagonal gait in which the movements of one stride have the diagonal fore and hind limbs in support followed by the other two limbs in support (Figure 11-2). In this gait, the right side and left side of the body move symmetrically, or the two sides mirror each other.

Asymmetric gaits include the rack, gallop or canter. In asymmetric movement the sides move differently. The gallop is the gait typically used by dogs that are running at high speeds.[7] It has two support phases and two flight phases in each stride (Figure 11-3). The gallop can have either a right or a left lead. In left-lead dogs the feet contact the surface in a clockwise fashion. The left front leg comes into contact first, followed by the right front foot in the front support phase. The dog then leaves the ground in the front flight phase. The next foot to make contact is the right rear foot, followed by the left rear foot in the rear support

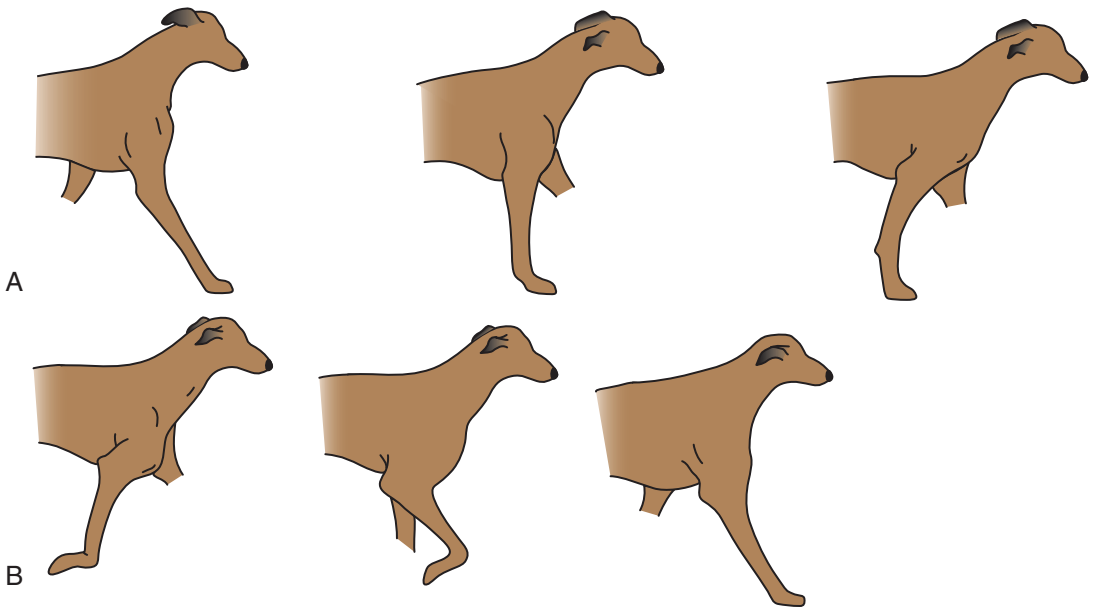


Figure 11-1 **A,** The stance phase. Braking forces occur at initial impact and continue until the center of gravity transfers over the fulcrum. Propulsive forces predominate for the remainder of the stance phase until the foot leaves the surface. **B,** The swing phase. Initially the leg swings caudally after the foot leaves the ground. It then swings cranially, until it begins the last caudal swing, which ends when the foot touches the surface.

phase. The dog then propels forward, leaving the ground a second time in the rear flight phase, after which the sequence is again repeated. A right-lead dog goes through the same sequence but contact is in a counterclockwise rotation. This gait is described as a double suspension-rotary gallop. The canter is slower than the gallop and is a three-beat gait, characterized by a "lead" forelimb, with two limbs moving separately and two as a diagonal pair. The pattern of footfalls with a dog cantering in a left lead is right hind, left hind and right fore together, followed by the left forelimb reaching furthest forward. The opposite series of footfalls would occur when a dog is cantering on the right lead. There is only one flight phase in the canter when all four limbs are off the ground, and it occurs just after the forelimbs leave the ground. The canter, sometimes called a lope, is an endurance gait and is sometimes used by sled dogs. Some of the larger dogs that have a wider body structure, such as the Rottweiler or Saint Bernard, use the canter as their preferred running gait.

Nonrepetitive Motions

Little research exists evaluating nonrepetitive motions. Therefore there have been no scientifically accepted definitions of the movements (e.g., jumping and movement initiation). One paper describes jumping[8] and another describes movement initiation.[9] Understanding these movements is important; for example, movement initiation is the movement that is used to begin all gaits. The forces of nonrepetitive motions are often higher than most normal gaits such as the trot, and it is possible that noncontact injuries may occur during nonrepetitive motions. The motions yield not only high forces but also high rates of force application to musculoskeletal tissues. Nonrepetitive motions generally involve a wide range of joint motion and segmental alignments with high force outputs and impacts. From a clinical standpoint, it is important to understand these movements because they can cause injuries or can be used to reveal injuries that might not be noted on a routine clinical exam involving a trot.

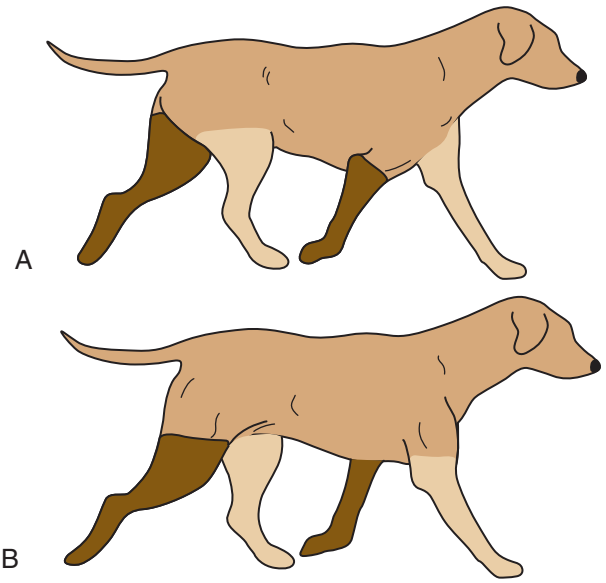


Figure 11-2 The trot. The limbs in light brown are in the stance phase, while the dark brown limbs are in the swing phase.

Structural Definitions

Movement is balanced throughout the whole body in a normal healthy animal. In the neuromusculoskeletal system, the two sides of the body mirror each other and are symmetric. An understanding of body symmetry is the key to analyzing gait abnormalities and lameness. Whether palpating the dog during a physical examination or observing the dog's movement, the right side of the body should be symmetric to the left side.

The dog's structure consists of axial and appendicular sections, which are divided into segments when analyzing motion. The axial vertebral column is made up of many joints and is divided into anatomic segments. The cranial segment is the head, followed caudally by the neck (cervical), thoracic, abdominal (lumbosacral), and tail segments. The appendicular segments are the front legs and the back legs. These are subdivided into smaller segments by the joints: shoulder, elbow, carpus, hip, stifle, tarsus, and phalanges. Locomotion as a whole is a result of the individual movements of these segments. How each of the segments and joints is used is determined by two factors. The first factor is the relationship of the dog's center of gravity to the surface, and the second is the type of movement that the body is performing.

Conformation

A major factor that affects how the body moves and receives forces is conformation. Conformation is the symmetry, size, and shape of various body regions relative to each other or in general appearance. Improper body design predisposes a structure to injury. Gait analysis reveals certain characteristics of a dog's conformation. This plays an important role in breeding programs. Sound movement contributes to the health and normal life span of all dogs.[10] Locomotion can vary for different breeds,[6] but in a

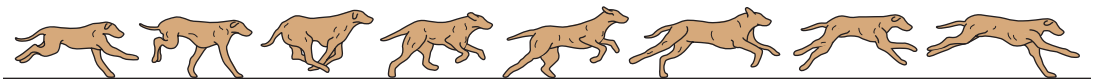


Figure 11-3 The gallop, with the dog in the right lead.

symmetric gait, like the trot, the movements of one side should still mirror the movements of the other side. A conformational abnormality usually creates an abnormal gait. Any abnormal gait should first be assessed for a conformational cause. Lameness of this type may not be related to pain but to a structural fault. Conformational abnormalities that can lead to musculoskeletal problems should be eliminated from breeding programs. If incorrect movement, or lameness, is not a result of compensation because of conformational abnormalities, other factors must be influencing the movement, such as an injury.

Center of Gravity

In the dog, 60% of the dog's weight is carried by the front limbs and 40% of the weight is carried by the back limbs while standing.[7] This results in the center of gravity (COG) being at the mid-chest level behind the scapula in the dog. The placement of the COG results in the front legs being associated more with braking forces, whereas the rear legs are more associated with propulsive forces on a level surface. Moving uphill shifts the balance of forces to the back legs and moving downhill shifts the forces to the front legs. An uneven or varying surface produces a variety of force alterations that depend on the body's position. For this reason, all general analysis should be performed on a level, even surface that provides proper traction.

Impact Forces

How a dog runs determines how the body receives the forces of surface impact. A dog that uses the double-suspension rotary gallop has high-impact forces on the front legs as they contact the ground. This is because as the limbs contact the ground, the entire weight of the body as well as the added braking and propulsion forces must be absorbed by the limbs. These forces have a greater effect on the lead leg than the non-lead leg. In the breeds that use the canter when they run, the impact forces are distributed differently. The forces of impact are distributed more evenly between the front and back legs.

Locomotion Analysis

In the past two decades, technologic advances in computer-assisted gait analysis have aided our ability to quantitatively define temporospatial gait characteristics. This has assisted researchers and clinicians in gaining a better understanding of canine locomotion. Subjective evaluation of canine gait has been used for many years. However, our ability to perceive minute details during the gait cycle can be very difficult, and in some respects, impossible, even for the most talented gait specialist. During a subjective evaluation, a clinician is only able to perceive a few kinematic variables at a time, but a kinematic or kinetic analysis system can capture, analyze, and store hundreds of observations per second. In addition, clinicians have the ability to view an animal in motion, but cannot determine the forces involved in gait and identify specific neuromuscular activity; therefore, we must employ kinematic and kinetic analysis tools for a more complete description of gait.[11]

Subjective Kinematic Analysis

Kinematic analysis is one of the oldest methods to evaluate movement of the musculoskeletal system. In 1888, Muybridge used stroboscopic photography to show the stride of racing greyhounds and hound dogs running at various speeds.[12] Subjective kinematic analysis is the most common diagnostic tool to assess lameness. The clinician evaluates the dog by observing gait and/or nonrepetitive motions and looks for abnormal kinematics. When analyzing movement, a clinician should define the mechanical system under analysis. The mechanical system can be the dog as a whole or a specific joint or limb.

To describe the components that make up a gait or motion, the movements of the joints, individual segments, and body as a whole are observed. These are interpreted in terms of time, distance (i.e., degrees or meters), speed, consistency, and accuracy. The trot, which is a symmetric gait, provides a good visual presentation of movement for the clinician to diagnose lameness. If a gait abnormality is present, the movements of one side are different from the other side.

The observations should be conducted before any physical palpation. First observe the animal while it is at rest, looking for conformational abnormalities or abnormal stances. For example, does the dog seem to hold one leg up or put most of its body weight on a particular leg? After these observations are noted the animal is analyzed while moving. If able, trot the patient in a straight line toward and away from the clinician. Next it should be assessed moving in a straight line from the right side and then the left side. Then it should be observed moving in a circle, clockwise then counterclockwise.

Movements of the body as a whole should be assessed for any major gait deficits first. Then the step cycle of each leg should be evaluated to determine which phase of the cycle is deficient. If the lameness appears at foot contact only, or the stance phase is shortened, the lameness can be associated with impact or support. An example of a structure that presents with this type of gait abnormality is the paw. The tissues in this area are the pads and skin, bones, ligaments, joint capsule, and tendons. The entire limb should be examined using physical palpation to locate which tissues are involved.

Most obvious abnormalities can be detected with subjective gait analysis. A dog with a lesion causing severe sharp constant pain carries the limb and keeps the weight off it. A dull aching pain produces a limp during gait. A lesion that produces a small pain that occurs in certain phases of locomotion allows the dog to adjust its gait for

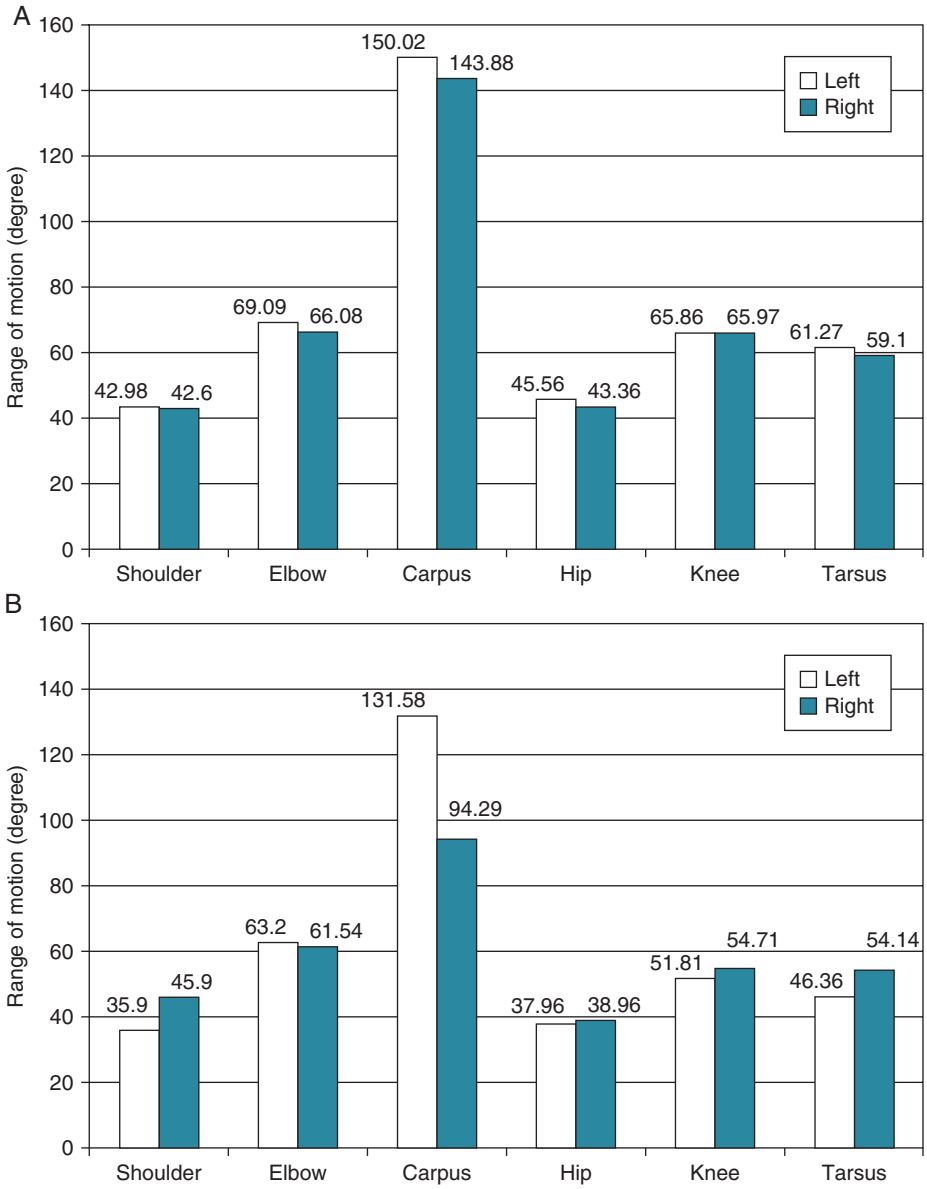


Figure 11-4 **A,** The chart shows actual range-of-motion values of sound dogs during a trot. **B,** The chart shows the actual range-of-motion values of a dog with restricted carpal movement.

relief. The quadruped has the ability to minimize pain by altering movement in such a way that the abnormality may be unnoticeable.[13] This altered gait can lead to subsequent compensatory problems.

The normal trot is a balance of movement by the whole body (Figure 11-4, *A*). Minor pain can create an imbalance in the system. For example, synovitis of the right carpal joint affects not only the joint of injury, but may also affect the left hip and left tarsus (Figure 11-4, *B*). An altered gait affects how the forces of movement act on the musculoskeletal structure. In addition to appendicular changes, the axial skeleton is affected. It is the frame through which this alteration is transferred. This can initiate two problems, one biomechanical and the other psychological. Biomechanically, if a condition is untreated the compensatory movements can lead to other injuries in the back or limbs. Psychologically, chronic pain can lead to behavioral problems.

Quantitative Kinematic Analysis

Quantitative kinematic analysis quantifies the positions, velocities, accelerations, and angles of anatomic points, segments, and joints in space. Kinematic analytic systems track anatomic markers through different planes of space and time and reveal their respective x, y, and z positional values. From the positional values, software programs can

use mathematical formulas to calculate linear or angular velocities and accelerations; therefore these systems assign numeric values to motion for quantitative gait analysis.[11] Some academic institutions currently have laboratories that can perform motion or gait analysis. These laboratories usually are equipped with a three-dimensional motion analysis system used for research or clinical analysis. Information about the animal's motion can assist in lameness diagnosis, assess rehabilitation progress, and possibly assess athletic potential. Veterinary medicine is just beginning to evaluate how motion analysis can raise the standards of clinical care. This is a rapidly growing field that will extend the boundaries of knowledge of patients' neuromusculoskeletal systems.

Traditionally these systems are too costly for use in most general practices and are relatively time-consuming to operate. An alternate method is two-dimensional gait analysis.[14] Reflective markers are placed over anatomic points (Figure 11-5). The subject is then trotted with its right side to the camera and then with its left side to the camera. The movement is filmed by a video camera system. The video clip is entered into a computer and each of the reflective markers are digitized and recorded. The points generally digitized are the lateral aspect of the distal fifth digit of the front paw, the carpus, the elbow, the acromion/greater tubercle of the humerus, the dorsal aspect of the scapular spine, the lateral aspect of the distal fifth digit of the rear paw, the tarsus, the stifle, the greater trochanter of the femur, and the dorsal iliac crest. Both sides of the body are digitized. These data points are used to quantify segmental and linear movement (Figure 11-6).

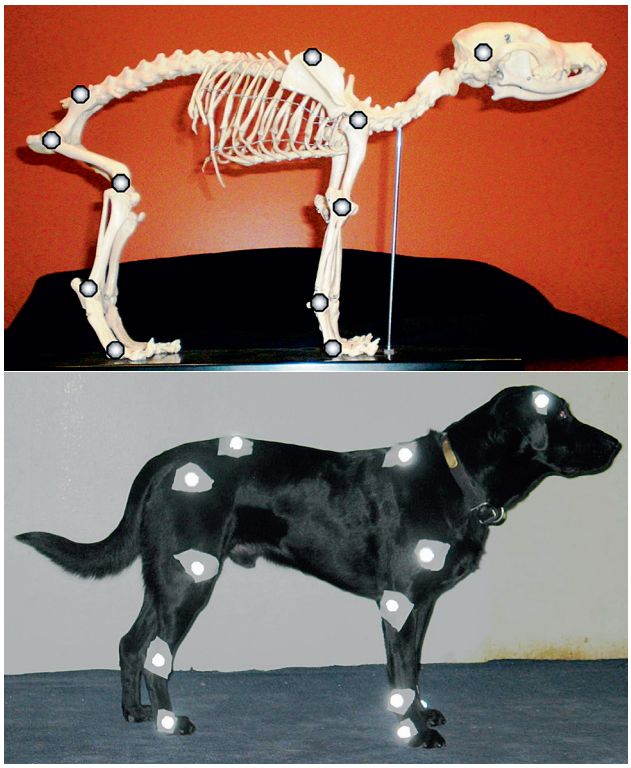


Figure 11-5 Reflective markers placed on the dog before filming. Markers are seen during locomotion.

The clinician can evaluate the data from both sides for asymmetry. When assessing gait and joint movement at the trot, any differences between the two sides may be used to evaluate lameness. When using proper methodology, two-dimensional gait analysis is valid and reliable. It requires only one video camera. It is important to only evaluate the gait cycle directly in front of the camera. Two-dimensional analysis systems are more economical than three-dimensional systems, allowing more practitioners an opportunity to use this tool.

In addition, filming outside of the laboratory is possible. Videotapes may then be evaluated by a laboratory, allowing further benefits of computer-assisted video gait analysis.

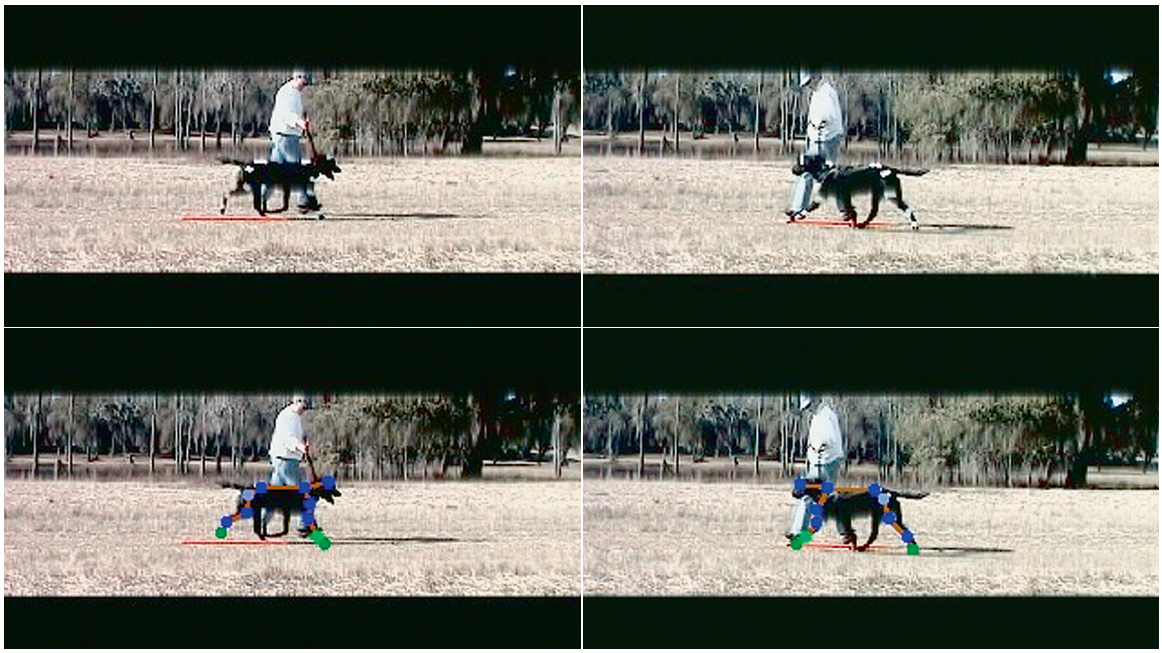
Future kinematic studies should include obtaining more information on different breeds. This may help define conformation standards for breeding purposes. Increasing the number of dogs analyzed with diagnosed musculoskeletal pathologies helps to determine parameters that are associated with those specific etiologies. After these parameters are determined, these methods may be used to quantify response to treatment.

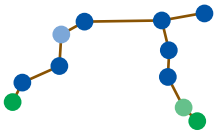
Professionals, trainers, and owners should be educated on the use of computer-assisted video gait analysis. The more information that is distributed the greater the potential to benefit from the knowledge gained about animal locomotion.

Kinetic Analysis

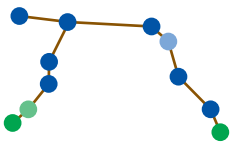
Kinetic (force) analysis quantifies the forces of locomotion by using measurement systems to evaluate kinetic variables, including peak vertical, braking, propulsive, and horizontal forces; vertical, braking, and propulsive impulses; strain within various tissues; rates of loading; temporal gait characteristics; and pressure distributions of the paw. There are many different methods and systems used to evaluate kinetic variables. Most of the systems used are ground-based (e.g., force plate and force mats) kinetic systems and measure orthogonal ground reaction forces (i.e., mediolateral force, craniocaudal force, and vertical force) resulting from paw impact during the stance phase of gait. Other systems, such as strain gauges, measure strain of different tissues when stress is applied during gait.[11]

The force plate can be used to measure ground reaction forces (GRFs) exerted on the limbs. The forces are measured in three orthogonal components: the vertical (z) plane, cranial-caudal (y) plane, and medial-lateral (x) plane (Figure 11-7). The force plate is usually embedded in a pathway for the test subjects to pass over. As the paw





	Maximum	Minimum	Range
Shoulder R	151.228	110.243	40.986
Shoulder L	152.974	118.156	34.818
Elbow R	149.462	75.421	74.040
Elbow L	155.255	95.657	59.598
Carpus R	250.092	79.376	170.716
Carpus L	228.575	106.408	122.168
Hip R	143.813	108.155	35.658
Hip L	146.587	106.966	39.621
Knee R	162.950	99.115	63.835
Knee L	157.884	103.896	53.988
Tarsus R	151.265	99.403	51.861
Tarsus L	161.181	98.810	62.372



A

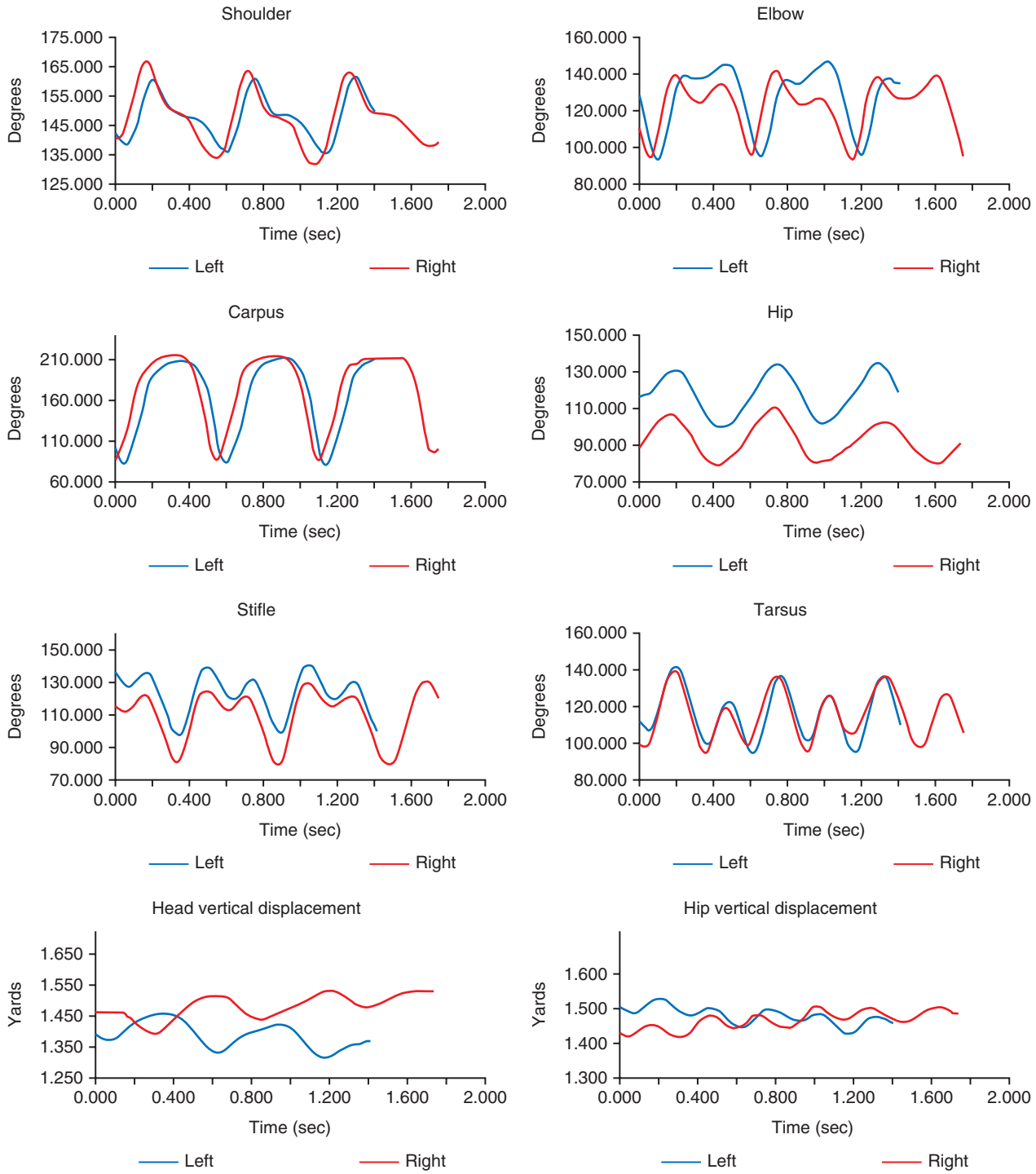
Figure 11-6 Video capture of gait from a dog using a 2-D video capture system. Kinematic data may be acquired from dogs working in the field.

Continued

comes into contact with the plate, GRFs are measured. The plate is connected to a computer that acquires the data for analysis. The data can be used to assess how the forces of the body are transferred through the limb. For analysis, the data from one limb can be compared with data collected over time or other limb data from the subject.

The measurements are made and read as force exerted over time. The contact time depends on the velocity of the subject. As the velocity increases, the contact time decreases. For example, the paw contact time for the walk is longer than the contact time in the trot. The walk has a biphasic *z*-force for each limb strike, which is similar to that of humans. The first peak is associated with the braking forces that occur at initial impact, and the second peak is a result of the propulsive forces. This is also seen in the *y* plane, with an initial negative force from the braking phase, and then a positive force from propulsion. The *x*-forces play a minimal role in gait evaluation. The biphasic *z*-force is not seen in the trot, presumably because of decreased contact time and the lack of a heel strike and toe-off in the dog. There is a single loading peak for each limb strike in the *z-plane* (Figure 11-8, *A*). The *y-forces* are biphasic, with braking and propulsive components (Figure 11-8, *B*). The contact time of the walk is longer than that of the trot, but the *z*-force of the trot is twice the force of the walk, as a result of the increase in velocity, and the limb support differences between the walk and the trot.

A technique has been reported for measuring ground contact pressure on individual paw pads of dogs.[15] The force plate measures the ground reaction forces transferred through the entire paw. To assess how the paw receives the GRFs, each individual digit may be assessed. This is done



B

Figure 11-6, cont'd Symmetry of Joint Motion may be evaluated with Kinematic data acquisition systems. For example, the right elbow, tarsus, and stifle joints have reduced ROM compared to the left side.

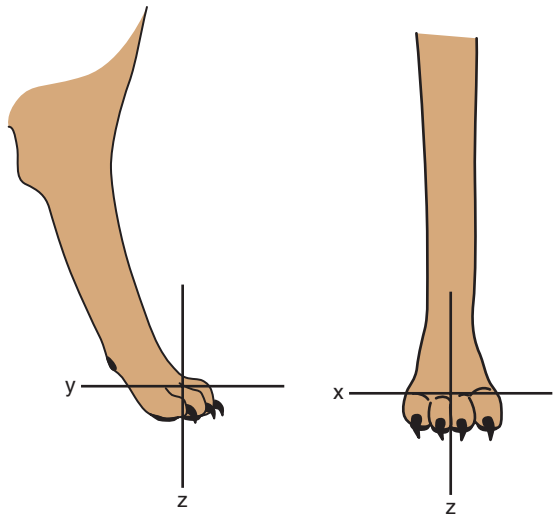


Figure 11-7 Ground reaction forces act on the foot in three orthogonal planes (x, y, and z).

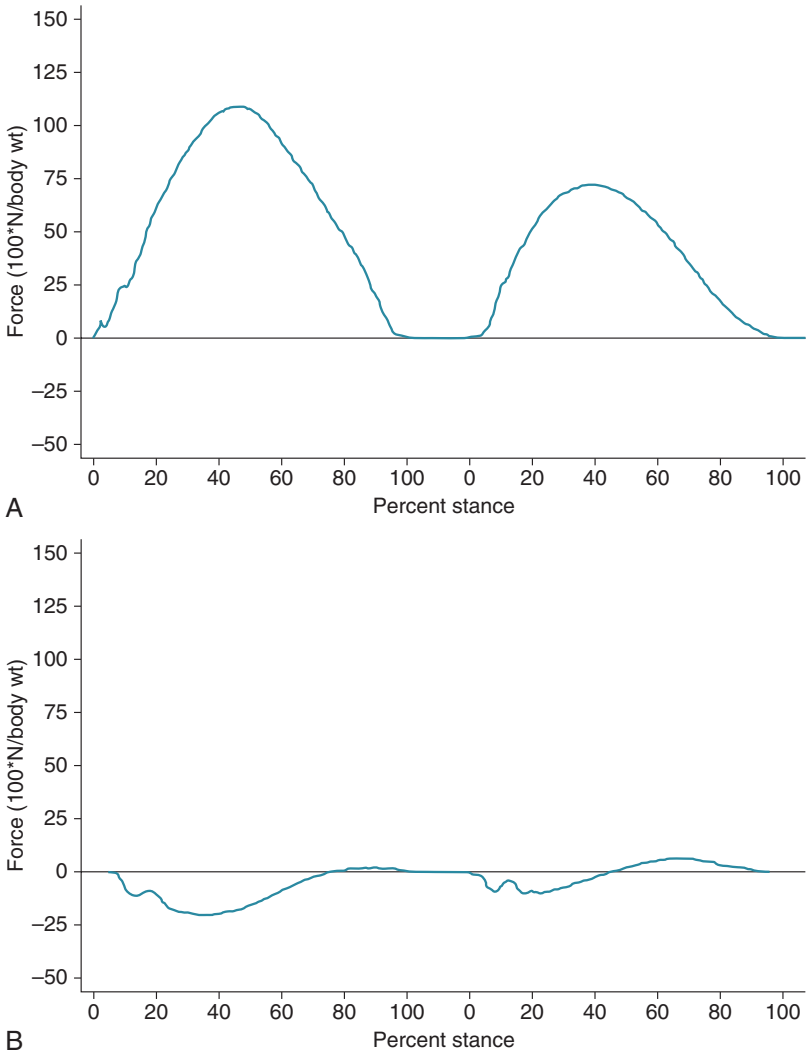


Figure 11-8 **A,** Force plate data showing measurement of the *z* force during surface contact at the trot. **B,** Measurement of *y* force taken during the trot.

by attaching a force-sensing resistor to each individual weight-bearing pad. One unexpected finding in one study was that the greatest pressures were on digital pads 3 and 5, and pads 2 and 4 had the least amount of pressure.[15] It had previously been thought that adjacent digits 3 and 4 were the pads of greatest pressure. This technique of measurement can be used for evaluating bandaging methods, prosthetic devices, and surgical manipulations for alleviating pressures.

Pressure Measurement Systems

Pressure mat systems are made up of many pressure sensors. When a dog walks over the mat, visual graphics of pressure distributions of the paw appear on the computer screen. These systems quantify high and low pressure areas of the paw, relative or actual vertical forces involved in the stance phase, and temporal characteristics of the stance phase. Pressure mats come in different sizes. Pressure mats in combination with a laptop computer provide a convenient portable kinetic data collection system.[11]

Summary

Locomotion analysis is a helpful analytic tool for the clinician, technician, handler, or owner. It is a good indicator of the status of the musculoskeletal system. The evaluator should have a good understanding of the individual components of locomotion, anatomy, conformation, and the influence of external factors. It is important to remember that center of gravity and conformation influence how ground reaction forces interact with the body. New technologies have advanced our ability to evaluate motion and forces. Veterinarians, physical therapists, breeders, owners, and trainers all can potentially benefit from the recent advances in locomotion analysis. Improved capabilities for data collection, storage, and analysis, in combination with advances in personal computer technology, have provided an opportunity for these analytic techniques to be used in the respective areas of interest. The more information that is gathered and distributed, the greater the potential to benefit from the knowledge gained regarding animal locomotion.

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12 The Physical Rehabilitation Evaluation

David Levine, Denis J. Marcellin-Little, Marti Drum, and Cassy Englert

Physical therapy is a profession with an established scientific basis in humans and companion animals. It has numerous clinical applications in the restoration, maintenance, and promotion of optimal physical function.[1] It is beneficial in helping people recover from anterior cruciate ligament reconstruction, fracture stabilization, joint surgery, spinal surgery, and many other injuries or diseases.[2-5] It also improves function in a variety of patients with soft tissue injuries, osteoarthritis, total joint arthroplasty, stroke, and chronic lower back pain throughout their lives.[6-10]

In providing physical therapy the goal is to restore, maintain, and promote optimal function, optimal fitness, wellness, and quality of life as they relate to movement disorders and health. In dogs, this may include treating patients during their recovery from orthopedic surgical procedures, such as femoral head ostectomy, monitoring weight loss programs, strengthening specific muscle groups, and helping to manage chronic conditions, such as osteoarthritis, or progressive conditions, such as degenerative myelopathy. A major emphasis is to prevent or minimize the onset, clinical signs, and progression of impairments, functional limitations, and disabilities that may result from diseases, disorders, conditions, and injuries. Examples in people include designing and delivering treatment programs for patients with problems such as pneumonia, multiple sclerosis, diabetes, cerebral palsy, lower back pain, or frozen shoulders.[1,11-14]

Treatment Philosophy

Physical therapists use a variety of treatment interventions such as stretching, targeted massage, passive range of motion, and joint mobilization. They also use electrical and thermal modalities and therapeutic exercises to help patients reach their goals. These treatments work synergistically to effectively achieve the therapeutic goals. When designing a treatment plan, the therapist should be aware of the scientific evidence supporting the use of each modality and exercise for the problems being treated. For example, when treating postoperative edema, ice has been proved to be beneficial,[15] whereas certain other therapies are unproven. The therapist should integrate the individual treatment plan with established perioperative and postoperative pain management protocols. Although the clinical signs present in many dogs with orthopedic or neurologic problems may improve over time, such as after fracture repair, a well-designed physical rehabilitation program may accelerate the recovery, may prevent permanent disability, and may help prevent future re-injuries.

Patients with movement disorders, weakness, pain, and limited endurance are candidates for physical rehabilitation. Examples of conditions include dogs recovering from orthopedic or other surgeries, dogs with osteoarthritis, tendonitis, or other soft tissue injuries. After a medical diagnosis is available, the therapist evaluates several aspects of the patient's health, particularly the health of the cardiopulmonary, neurologic, orthopedic, and integumentary systems. The more specific the medical diagnosis, the more directed will be the care. For example, the medical diagnosis for a patient may be osteoarthritis of the elbow and the physical rehabilitation diagnosis for that patient may be limited flexion and extension with cranial and caudal joint capsule tightness. These factors may limit function in terms of gait; improving elbow range of motion through specific treatment interventions may improve the functional status of the patient. In the practice of physical therapy for humans, the areas evaluated include aerobic capacity, balance, arousal, cognition, environmental barriers, ergonomics, posture, gait, pain, range of motion, prosthetic requirements, and assistive and supportive devices. Most of these parameters may be evaluated in dogs.

Physical rehabilitation in veterinary medicine follows the same principles. The therapist collects functional information by evaluating the dog's physical fitness, orthopedic, and neurologic health. This may be done in conjunction with or after the veterinarian's evaluations. There is overlap in these evaluations, but although the veterinarian evaluates the patient to obtain a medical diagnosis and to prescribe medical or surgical treatment plans, the therapist evaluates the patient to create a physical rehabilitation treatment plan. This evaluation includes the assessment of muscle mass, joint motion, joint stability, and pain. For example, loss of range of motion may be present with elbow dysplasia, whereas loss of sensation and muscle atrophy may be observed with radial nerve injuries.

Physical rehabilitation plays an important role in prevention of and recovery from injury. Educating owners in proper warm-up and cool-down techniques, for example, may help prevent orthopedic injuries during training or competition. Owners may have a substantial emotional and financial investment in their dogs and are generally very willing to assist in carrying out rehabilitation programs when properly instructed.

Physical Rehabilitation Evaluation

The physical rehabilitation evaluation involves the evaluation of active motion during various gaits and while performing specific activities, such as stair climbing. The dog should be evaluated at a walk, trot, and gallop if the patient is able and it is safe to do so. The evaluation also includes the assessment of function at rest, including posture, balance, and strength. Balance may be assessed by performing various perturbation maneuvers such as pushing the dog off balance and assessing the dog's response. Balance is further tested by evaluating overall coordination, gait, and whether or not the patient falls. Proprioception during ambulation is tested by evaluating toe dragging, knuckling, awkward stepping, and limb carriage and placement. Strength is tested by evaluating muscle mass and the ability to perform normal activities. For example, hip extensor strength may be assessed by observing the dog when it rises. The evaluation continues with assessment of pain and motion of joints. The therapist evaluates discomfort and the condition of injured and abnormal tissues, such as excessive laxity or restriction of motion as a result of scar formation (Table 12-1). The therapist collects this information to understand how these limitations affect function and how they may be targeted during therapy.

To help determine the cause of articular, muscle, or connective tissue restrictions in motion, the examiner assesses the *end feel*. The end feel is the sensation imparted to the examiner's hands at the end of the range of motion of the tissue being examined (Figure 12-1).[16] The normal end feel for most joints is imparted by the joint capsule and is a reflection of the slight elasticity of that capsule. Abnormal end feels are described in Table 12-2.

A typical rehabilitation evaluation form (Figure 12-2) allows the evaluator to easily document the pertinent parts of the evaluation and be able to refer back to them as treatment progresses. It may also be used as a form of communication between the rehabilitation practitioner and the referring veterinarian (RDVM).

Rehabilitation Candidates

All dogs that have neurologic or orthopedic problems are candidates for rehabilitation, particularly hunting and working dogs and those dogs that perform strenuous physical activities, such as agility, racing, field trials, police activity, and Schutzhund. Rehabilitation also applies to

Table 12-1 Specific Tissue Assessments during the Physical Rehabilitation Evaluation

Tissue	Parameters	Anomalies	Potential Causes
Muscle	Mass/size	Atrophy	Denervation, disuse, contractures
		Hypertrophy	Increased training and conditioning
		Increased	Edema, hematomas, neoplasia
	Tone	Increased	Spasm, guarding, denervation (upper motor neuron)
		Decreased	Denervation (lower motor neuron)
	Pain	Acute pain	Tear, myositis, spasm
		Chronic pain	Tear, contracture, spasm, referred neurogenic pain
Tendon	Pain	Acute pain	Tendonitis, strain
		Chronic pain	Tendonitis
	Tension	Increased	Adhesion, contracture
Joints	Motion	Loss	Contracture, periarticular fibrosis or adhesions, effusion
		Abnormal	Subluxation, luxation
	Pain	Acute pain	Subluxation, luxation, infection
		Chronic pain	Osteoarthritis
Ligaments	Stability	Decreased	Sprain, rupture
		Increased	Adhesion, contracture
	Pain	Acute pain	Sprain
		Chronic pain	Adhesion



Figure 12-1 This Labrador retriever is recovering from an open surgical lavage for treatment of a large hematoma and infection after extracapsular stabilization of a torn cranial cruciate ligament. A physical therapist evaluates range and end-feel with stifle extension (*A*) and flexion (*B*), the presence of cranial drawer (*C*), and swelling and adhesions of tissue planes (*D*) in the operated area.

dogs recovering from surgery. For example, range of motion exercises may be used after repair of a fracture of the distal femur in a puppy to prevent quadriceps tie-down. Also, a reconditioning program may be instituted after a major medical condition or abdominal surgery to help return the patient to its previous status. Some of the common conditions appropriate for rehabilitation include surgery for cranial cruciate ligament rupture, patellar luxation stabilization, femoral head and neck ostectomy (FHO), various fractures with and without surgical repair, spinal disk decompression, neurologic conditions such as fibrocartilaginous embolic myelopathy, ligamentous instabilities, overuse injuries, muscular imbalances, chronic pain, and weight loss and general conditioning. Weight management is quickly becoming a common reason for referral to rehabilitation. Obesity in dogs can lead to many of the preceding orthopedic injuries, pain, and general health concerns. The rehabilitation practitioner commonly uses a

Table 12-2 Anomalies in End Feel Assessed during the Physical Rehabilitation Evaluation

Type of End Feel	Definition	Potential Causes	Example
Capsular	Slight elasticity	Normal joint	Normal shoulder extension
Firm capsular	Decreased elasticity	Periarticular fibrosis, adhesions	Loss of stifle extension following cranial cruciate ligament rupture
Springy	Increased bounce	Joint effusion, joint mouse	Always abnormal
Soft tissue approximation	Motion limited by soft tissues	May be normal or caused by swelling	Normal hip flexion
Empty	End of motion cannot be reached	Pain	Intraarticular fracture
Hard	Abrupt stop	Bone on bone contact, mature contractures	Chronic quadriceps contracture following distal femoral physeal fracture

variety of therapeutic exercises, including an underwater treadmill to exercise obese or overweight dogs because the buoyancy of the water decreases joint stress while increasing the metabolic rate for successful weight loss (Figure 12-3). Rehabilitation for weight management works best when done in conjunction with the referring veterinarian's recommendations regarding diet and medication if necessary.

Developing a Physical Rehabilitation Plan of Care

Based on the results of the medical history and diagnosis, as well as the physical rehabilitation evaluation, a clear, evidenced-based plan of care is developed and implemented. In some instances, the plan of care is designed using evidence-based conclusions from human studies and having knowledge of the anticipated tissue responses to therapy in dogs. The therapist chooses the treatment plan that has the fastest, most predictable positive outcome on the patient. Acute postoperative edema, for example, may be treated using ice, bandaging, and pain-free passive range of motion. As the acute inflammation subsides, gradual resumption of activity may be combined with hot packs or ultrasound therapy. The plan of care is subsequently modified based on frequent reassessments. Patients may be treated as inpatients in the hospital or as outpatients in rehabilitation centers. The plan of care must be unique to the patient and must take into account all abnormal findings and other factors, including the severity of the anomalies, the age and disposition of the dog, the expectations for future performance, the urgency of the recovery, the available equipment and technical skills of the clinicians, and the cost of treatment. A typical plan of care may include a choice of thermal and electric modalities and specific exercises aimed at strengthening with gradual reintroduction of specific physical activities. The therapist prescribes the specific type, intensity, duration, frequency, and progression of these exercises. Exercises are generally initiated with a low intensity and duration that increases progressively as healing progresses and tissue strength increases. It is also important to have established treatment goals or end points to guide progress.

The therapist may also develop preventive strategies for patients. These include monitoring the dog's weight, monitoring floor surfaces at home, having a preseason training period in athletic dogs, developing proper warm-up and cool down periods, recommendations for adequate rest between strenuous exercise sessions, and monitoring transportation between locations. For example, a dog with osteoarthritis of the stifle may benefit from weight loss, a judicious exercise plan with frequent exercises of short duration on soft surfaces, a padded surface to sleep, and a careful strategy of pharmacologic management, including analgesic medications and disease-modifying osteoarthritic agents.

Re-Evaluation and Assessment

The rehabilitation practitioner should regularly assess the dog's response to treatment, both during and after each session. During each session, the practitioner looks for adverse responses such as increased lameness or unwillingness to weight bear on a particular limb, fatigue, and increased anxiety. If any of these are apparent during a treatment session, the session may be altered or stopped. The overall treatment plan also may be modified if necessary. Many times, the dog will be given several days to rest and recover before continuing rehab. Following each session, the owners are instructed to watch for increased fatigue or abnormal pain behavior at home and to notify the practitioner and/or referring veterinarian if it does not resolve within 24 hours. In addition to observing response to rehabilitation, objective measurements and functional

University of Tennessee College of Veterinary Medicine

Department of Small Animal Clinical Science
Veterinary Physical Rehabilitation
INITIAL EVALUATION Date: _____ Chief Complaint: _____
Weight: _____
Diagnosis: _____ Surgery Date: _____ Attending /
RDVM: _____
Affected Limb: RF LF RR LR N/A

SUBJECTIVE AND HISTORY

General appearance and disposition:

_____
_____
_____

Onset of problem/injury:

_____
_____

Past pertinent medical history:

_____
_____
_____

Home environment/Baseline activity level:

_____
_____

Diet/medications/supplements:

_____
_____

Additional Information and Owner goals:

_____
_____

OBJECTIVE FINDINGS

PT ORTHOPEDIC LAMENESS (POSTURE/STANCE)

0=normal stance/no lameness
1=slightly abnormal stance (PWB)
2=moderately abnormal stance (PWB-TTWB)
3=severely abnormal stance (TTWB-NWB)
4=unable/unwilling to bear weight (NWB)

PT ORTHOPEDIC LAMENESS (AT WALK & TROT)

0=Normal walk/trot, no lameness
1=Slight or intermittent lameness
2=Obvious weight bearing lameness
3=Severe weight bearing lameness
4=Intermittent non-weight bearing lameness
5=Continuous non-weight bearing lameness

PT NEUROLOGIC GRADING SCHEME

5=normal strength/coordination
no obvious dysfunction
4=can stand to support
minimal paraparesis and ataxia
3=can stand to support but freq. stumbles and falls;
mild paraparesis and ataxia
2=unable to stand or support; with assistance moves
limbs readily but freq. stumbles/mod ataxia and
paresis
1=unable to stand or support; slight movement when
supported by the tail; severe paresis
0=absence of purposeful movement; tetra- or
paraplegic

Rehab Orthopedic Measures

Lameness stance (0-4; 0=normal): Pre-op_____ Post-op _____ Current _____
Lameness walk (0-5; 0=normal): Pre-op_____ Post-op _____ Current _____
Lameness trot (0-5; 0=normal):Pre-op_____ Post-op _____ Current _____

Figure 12-2 Rehabilitation evaluation form. Courtesy University of Tennessee College of Veterinary Medicine.

Continued

Rehab Neurologic Measures

Neurologic Grading Scheme (0-5; 0=normal): Pre-op_____ Post-op _____ Current _____

Gait

_____

_____

Standing limb position:

_____

Sitting limb position:

_____

Willingness to raise contralateral limb: YES NO RELUCTANT UNABLE

Force Plate: YES NO Analysis Results: _____

Skin/Incision: _____

Discoloration: _____ Temperature to touch: _____Other: _____

Swelling/Edema/Muscle Mass

Circumference of limb: Affected ____cm Unaffected ___cm Site & Position: _____

Comments:_____

Additional measurements (if taken):

_____

_____

Range of Motion

Joint (normal range)	R Flex/Ext	L Flex/Ext	End Feel/Comments	Test Position
Carpus (32°-196°)	____/____	____/____	_____	_____
Elbow (36°-166°)	____/____	____/____	_____	_____
Shoulder (57°-165°)	____/____	____/____	_____	_____
Tarsus (38°-165°)	____/____	____/____	_____	_____
Stifle (41°-162°)	____/____	____/____	_____	_____
Hip (50°-162°)	____/____	____/____	_____	_____

Pain/Comments:_____

Functional Assessment

_____

_____

Palpation

_____

_____

Special Tests and Results

_____

_____

Figure 12-2, cont'd

ASSESSMENT

_____

_____

_____

PLAN/ RECOMMENDATIONS

_____

_____

_____

_____

_____

Short-term goals (time frame)

_____

_____

_____

Long-term goals (time frame)

_____

_____

_____

_____

Evaluator Signature/Date

_____

Figure 12-2, cont'd



Figure 12-3 A 6-month-old Labrador retriever is recovering from extracapsular stabilization of his left stifle joint after avulsion of his cranial cruciate ligament. The dog is undergoing static weight shifting exercises (*A*), walking in an underwater treadmill (*B*), trotting on a treadmill (*C*), and walking with an elastic band eliminating the external rotation present in his right pelvic limb during the recovery period (*D*).

assessments are regularly performed and documented. Measurements, such as joint range of motion, muscle girth, and lameness assessments at a walk, trot, or gallop, are frequently assessed and related to changes in function. A functional questionnaire also may be used at the beginning, during, and end of a course of rehabilitation. In all cases, close communication is maintained between the rehabilitation practitioners and referring veterinarian regarding the dog's treatment plan, changes to treatment plan, and overall progress.

Delivery of Care

The owner, trainer, therapist, therapist assistant, and others may deliver aspects of the physical rehabilitation plan of care. Some tasks may be delegated to the owner. Other more technical tasks should be performed by the therapist. Specifically owners may administer some exercises to their dogs and may apply cold packs. Some owners may have the ability to perform tasks such as neuromuscular electrical stimulation or other treatments. The care must be supervised and coordinated by the clinicians who assess the patient, decide who will perform the treatments, and determine how the treatment plan should be modified over time, including increases or decreases in exercise duration and frequency, and initiating or discontinuing modalities. Feedback (i.e., the severity of current clinical signs and the presence or absence of new clinical signs) must be collected from all involved in the dog's care, and adjustments in the patient care plan must be communicated to all involved.

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13 Assessing and Measuring Outcomes

Darryl L. Millis and David Levine

Assessing the outcome of treatments, including physical rehabilitation, is essential to determine how an animal is progressing and to assess the effectiveness of treatment protocols. Review of data and evaluation of protocols are necessary so changes can be initiated to improve outcomes. Assessments should consist of objective data whenever possible because owners and veterinarians often believe a patient is doing better than the data suggest. In addition, documentation of progress is important to provide incentive for owners to continue rehabilitation and justify continued treatment.

Several measurements are useful for assessing outcomes, including the ability to perform functional activities of daily living, gait analysis, pedometers, joint function, muscle mass and strength, body composition, pain assessment, impressions of owners and veterinarians, return to function, and quality of life.

Activities of Daily Living

Activities of daily living (ADLs) can be defined as the activities the animal must perform independently on a daily basis. Despite the best treatments, rehabilitation facilities, and personnel, some conditions are so serious that complete recovery cannot be expected. In these situations, the owner must be informed of a reasonable expected outcome. The rehabilitation goals should be realistic and should concentrate on performing basic life functions, such as eating and drinking with no or minimal assistance, changing body positions without assistance, rising from a sitting position, and walking outside to urinate and defecate with no or minimal assistance. In some cases even these goals may not be met. Progress may be slow but it should be recorded and rehabilitation plans should concentrate on activities to help achieve these rehabilitation goals.

The expected ADLs of working animals undergoing rehabilitation, such as guard dogs, police dogs, and assistance dogs for the disabled, should be discussed and clearly understood at the initial evaluation. A dog that is expected to work for 8 consecutive hours may need intensive rehabilitation often progressing up to several hours/day to ready it to return to work.

Measurements of ADLs are sometimes difficult. The timed get up and go (TUG) test is a measure of mobility that has been studied and used extensively in human rehabilitation.[1] The person is asked to stand up from a standard chair and walk a distance of 3 meters, turn around, and walk back to the chair and sit down again. This test involves a number of tasks, such as standing from a seating position, walking, turning, stopping, and sitting down, which are all important tasks needed for a person to be independently mobile. Normal time required to finish the test is between 7 and 10 seconds. Similar tests could be developed for dogs, but because the TUG depends on a person's mental ability and understanding of what is expected, it must be modified for dogs. (Dogs competing in some sporting events such as fly-ball may be an exception because of their training and understanding of what is expected.) Nevertheless a dog can be timed during a test of rising from a sitting position, walking on leash for a distance, turning, and then sitting. For the average house pet other objective activities that could be measured include how far a dog can walk or trot on a leash before needing to sit or lay down. These can all be compared over time to assess function and mobility.

Gait Analysis

Weight Bearing at a Stance

Evaluation of a dog's stance gives information regarding willingness to place complete weight on an affected limb. Many dogs may have no visible or only very mild lameness at a walk or trot, but will not bear equal weight on the limbs when standing. Weight bearing at a stance may be assessed by observing the placement of a foot in relation to the contralateral front or rear foot. In severe cases the dog may hold the foot completely or partially off the ground. More commonly the dog places a moderate amount of weight on the foot but not complete weight. The toes may point out and when the limb is gently pushed forward it may be moved more easily than the contralateral limb when it is gently pushed. Alternatively the evaluator may place both hands under the fore or rear feet with the palm facing the pads, and the relative amount of weight bearing may be assessed. Weight bearing at a stance may be incorporated into global lameness scores.

An inexpensive method of acquiring more quantitative information regarding weight bearing at a stance is to have

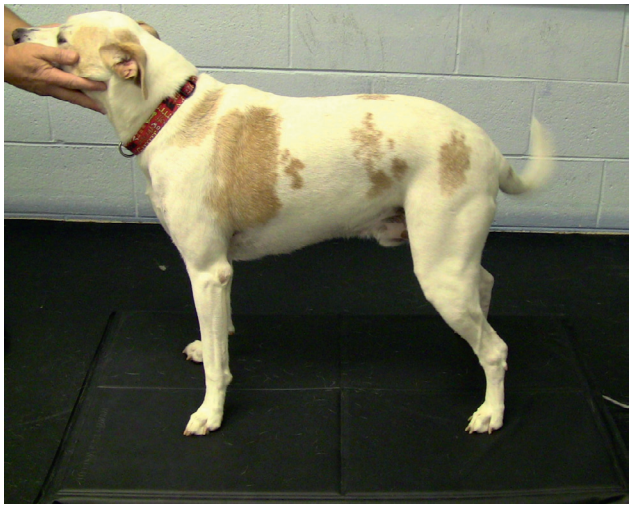


Figure 13-1 Measure of static weight-bearing forces using a computerized stance analyzer (Petsafe, Knoxville, TN).

Box 13-1 Lameness Evaluation at a Walk and a Trot

Lameness Evaluation at a Walk

0 Walks normally
1 Slight lameness
2 Obvious weight-bearing lameness
3 Severe weight-bearing lameness
4 Intermittent non–weight-bearing lameness
5 Continuous non–weight-bearing lameness

Lameness Evaluation at a Trot

0 Trots normally
1 Slight lameness
2 Obvious weight-bearing lameness
3 Severe weight-bearing lameness
4 Intermittent non–weight-bearing lameness
5 Continuous non–weight-bearing lameness

a dog stand with each limb on common household scales. It is important to be certain that the dog is standing squarely in a standard position, with each limb on a scale. Dogs should also undergo a period of acclimation to the scales so that data collection is valid. Scales should be calibrated periodically to standard weights to be certain that accurate weights are recorded. One study found that bathroom scales were a reliable, simple, and cost-effective objective method for measuring static weight bearing in dogs with osteoarthritis in the hindlimbs.[2]

Computerized devices are available that simultaneously measure static weight bearing of all four limbs (Figure 13-1). As with any test, proper data collection is crucial to obtaining reliable information. One study of various conditions during data collection concluded that to achieve consistent results, one standard method of conditions during data collection should be used.[3] Factors to consider include the position of the handler, proximity to walls, and local environment. We recommend having the dog in a quiet environment with minimal noise or distractions, with the dog facing either the center of the room or directly toward, but some distance away from, a wall. Any other walls should be at least 3 to 6 feet from the dog. It is critical that the dog stand squarely and symmetrically, with each foot in the middle of pressure pad. In most cases, lifting the dog's back or front limbs off the ground temporarily and then placing them back on the ground will result in square limb placement. The head and neck should be relaxed in a neutral position and facing straight ahead. We have found that dogs respond well to having the handler facing forward and straddling the dog over its back. Another option is for the handler to squat or kneel directly in front of the dog. The handler's hands can then help to guide the dog's head in a straight position, being careful to avoid touching the dog; the hands merely act as blinders to help keep the head in a straight position. Following these guidelines results in very consistent data collection and has very good correlation with ground reaction forces measured with a force plate.[4,5]

Lameness Scores

Lameness scores are a vital clinical outcome assessment tool. The walk and trot are the gaits most commonly evaluated because the speed of the limbs during movement is easier to assess without specialized equipment, and these gaits are symmetric, making identification of a lame limb easier. The reader is referred to the chapter on orthopedic and neurologic evaluation for information regarding identification of lameness. It is important to separate the walk from the trot when scoring lameness. Dogs are generally less lame at a walk because less force is placed on the limb at this less strenuous gait. Trotting may accentuate a lameness that is mild to moderate at a walk because of the greater forces placed on the limb with increased velocity. Having separate lameness scores for the walk and trot allows finer discrimination of gait analysis and may be more sensitive for detecting subtle improvements during rehabilitation (Box 13-1).

Although lameness scales are simple, commonly used, and inexpensive, they may not be accurate in evaluating subtle or mild lameness. One study compared a numerical scale and visual analog scale (VAS) with ground reaction forces and concluded that subjective scoring scales do not replace force plate gait analysis in dogs with surgically induced lameness.[6] Agreement of experienced evaluators with ground reaction forces was low unless lameness was severe. In addition each observer had his or her own individually unique scale. Therefore observers must stay the same during the evaluation of a patient for accurate analyses. Another study of induced hind limb lameness of dogs

found that subjective evaluation of lameness varied greatly between different observers, and there was poor agreement with objective ground reaction force measurements.[7] Similar results regarding estimating the severity of lameness using a VAS have been found in dogs with fragmented coronoid processes.[8] However, owners may not be able to assign a valid VAS score.[9] Therefore, this form of evaluation is better performed by people experienced in evaluating lameness and pain.

An alternative approach may be to use a questionnaire to assess pain and lameness in dogs. One study evaluated a variety of questions using a VAS and found significant correlation with peak vertical force and vertical impulse.[10] Responses to questions recorded on a standard 10-cm VAS that had significant correlation with ground reaction forces included:

- Overall owner assessment for preceding month
- Mood of dog during preceding month
- Attitude of dog during preceding month
- Frequency of postures of a happy dog
- Change in amount of daily activity
- Willingness to play voluntarily
- How often dog gets exercise
- Stiffness when arising for the day
- Stiffness at end of the day (after activities)
- Indicates lameness when walking
- Pain when turning suddenly while walking

The Helsinki Chronic Pain Index also found mood, play, locomotion while walking and trotting, getting up, difficulty moving after rest, and difficulty moving after major activity to be valuable in helping to identify dysfunction with osteoarthritis.[11] Vocalization, galloping, jumping, and laying down are other activities that help identify dogs with osteoarthritis.

A Canine Brief Pain Index (CBPI) has also been developed to evaluate chronic pain in dogs with osteoarthritis, and consists of 11 questions related to pain, function, and overall impression of quality of life; a 10-point scale is used to indicate the severity of the condition for each of the 11 questions.[12] While the CBPI is able to detect improvement in pain scores of dogs with osteoarthritis after treatment with an NSAID,[13] there is not significant correlation of CBPI scores with ground reaction forces as measured with a force platform, suggesting that owners evaluate things other than lameness in assessing improvement with treatment.[14]

In summary, although lameness scores are commonly used and clinically useful in some circumstances, care must be used in interpreting the scores. In general, agreement among individuals making assessments is greatest in dogs with severe lameness and no lameness. There is generally poor agreement in mild cases of lameness, and there is poor correlation of lameness scores with objective measurement of ground reaction forces using a force platform. VAS scores may offer some advantages, but experience in lameness evaluation is important in assigning values. Finally, various pain indexes may be useful in assessing treatment effects in dogs with osteoarthritis. However, there is relatively poor correlation with ground reaction forces, likely because a variety of functional items other than just lameness are assessed by owners when using indexes.



Figure 13-2 Measurement of ground reaction forces using a force plate.

Kinetic (Force Plate) Analysis of Gait

Kinetic evaluation of gait involves the measurement of ground reaction forces with a force plate or platform (Figure 13-2). It is an objective, repeatable measure of weight bearing on limbs when proper technique is used for data collection. Lameness can be compared over a period of time without relying on memory of previous assessments because data may be stored on a computer. Although measurement of ground reaction forces is a reliable and well-accepted method of determining the degree of weight bearing on the limbs, it is an artificial situation, and some dogs may display different clinical signs in a home environment. Force platform systems are available at many veterinary colleges and some private practices. It is important that appropriate software be used for quadruped animals.

Force plates use either strain gauges or piezoelectric crystals. The force plate is either mounted on a platform or embedded in the floor so that it is even with the surface of the floor. A runway of adequate length is essential. Most systems have timer lights that are triggered as the handler and dog approach and cross the force plate to allow the calculation of mean velocity and acceleration. Control of

velocity and acceleration within appropriate parameters is essential for repeatable data collection, because these greatly affect the force placed on each limb. The force plate is connected to a computer that calculates ground reaction forces. Force plates may also be embedded in treadmills so that consecutive footfalls can be recorded, with the gait being very rhythmic. However, some systems may not be able to accurately evaluate braking and propulsion forces.

Pressure pads or mats are also used for evaluation of canine gait. Many commercially available systems have an adequate number of sensors to detect ground reaction forces with reasonable accuracy. One advantage is that they can measure data from several consecutive footfalls. Another advantage is the ability to determine stride length, cadence, and average velocity.

The most useful forces measured are the peak vertical force (Z_{Peak}) and vertical impulse ($Z_{Impulse}$) (Figure 13-3). Other forces that may be useful are the peak braking ($Y_{A\ Peak}$) and propulsion ($Y_{B\ Peak}$) forces and braking ($Y_{A\ Impulse}$) and propulsion ($Y_{B\ Impulse}$) impulses (Figure 13-4). Medial-lateral forces (X_{Peak}) and impulse ($X_{Impulse}$) are likely too small and variable to be clinically useful. Forces may be measured during stance, walking, or trotting.

It is essential that appropriate technique be used during data collection. In addition to consistent velocity and acceleration targets, an experienced handler is necessary to minimize intertrial variability, although there is little or no difference in measured forces between experienced handlers. The handler should be between the dog's head and shoulder, and the dog should be gaited without undue tension or pulling on the leash. The dog should not alter the gait, throw its head, turn the head, lunge, or make other sudden movements as it approaches and crosses the force plate or pressure mat. The head carriage should be in a neutral position to avoid shifting weight to the front or rear limbs.

In general, dogs bear approximately 30% of their body weight on each front limb and 20% on each rear limb while in a standing position with the limbs placed squarely under the body. Walking at a velocity of 0.7 to 1.0 m/sec results in forces equivalent to approximately 60% of body weight on each forelimb and 40% on each rear limb in a medium to large dog. Increasing the velocity to a trot of 1.7 to 2.0 m/sec results in weight bearing of 100% to 120% of body weight on each forelimb and 65% to 70% of body weight on each hindlimb in a similar size dog. Reduced weight bearing on an individual limb may result in mild weight shifts to the other limbs.

Kinematic (Motion) Analysis of Gait

Kinematic or motion analysis of gait is a powerful tool that can be used to measure flexion and extension angles of

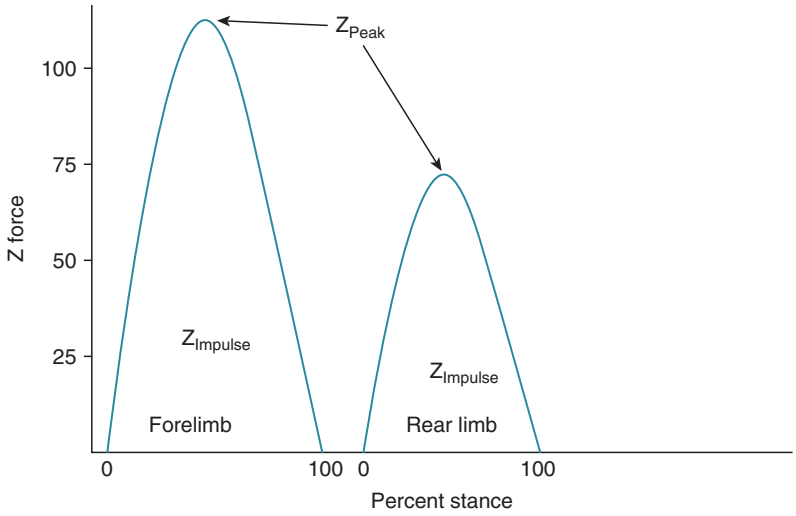


Figure 13-3 Graphic depiction of peak vertical force (Z_{Peak}) and vertical impulse ($Z_{Impulse}$).

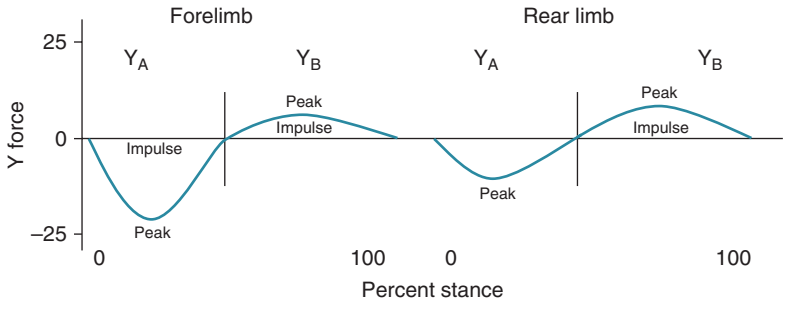


Figure 13-4 Graphic depiction of peak braking ($Y_{A\ Peak}$) and propulsion ($Y_{B\ Peak}$) forces and braking ($Y_{A\ Impulse}$) and propulsion ($Y_{B\ Impulse}$) impulses.



Figure 13-5 The dog is instrumented for kinematic assessment of joint motion and stride characteristics.

joints during gait, stride length, and other parameters of stride. It is usually combined with kinetic gait analysis. There is limited availability of three-dimensional kinematic gait analysis because the necessary equipment is sophisticated and expensive. Two-dimensional systems are less expensive, but they do not capture rotation and circumduction of limbs during gait, although simple flexion and extension of joints may be captured.

Attention to detail is critical for kinematic gait analysis. Most systems use a number of reflective devices that are attached to the dogs at very specific anatomic points to allow repeatable data collection at different times (Figure 13-5). Motion of the markers in relation to the joints is determined with a series of cameras interfaced with a computer. Software for quadruped animals allows reconstruction of a walking "stick figure" on the computer screen. The software is then used to calculate a number of measurements, including flexion and extension angles of joints during gait, angular velocity and acceleration of joints, and stride length and frequency.

Noninvasive, computer-assisted, three-dimensional kinematic gait analysis has been used to study lameness associated with cranial cruciate ligament rupture in dogs.[15] Dynamic flexion and extension angles and angular velocities were calculated for the hip, stifle, and hock joints. Distance and temporal variables were also determined. Mean flexion and extension curves were developed for all joints, and the changes in movement that occurred over time after cruciate rupture were compared. Each joint had a characteristic pattern of flexion and extension movement that changed with cruciate rupture. The stifle joint angle was more flexed throughout stance and early swing phase of stride and failed to extend in late stance. Angular velocity of the stifle joint was damped throughout stance phase, with extension velocity almost negligible. The hip and hock joint angles, in contrast with the stifle joint angle, were extended more during stance phase. Stride length and frequency also varied significantly after cruciate rupture. A change in the pattern of joint movement appeared to occur in which the hip and hock joints compensated for the dysfunction of the stifle joint.

Knowledge of kinematic parameters may allow more effective rehabilitation of patients following surgery or those with chronic conditions. In particular, changes that are noted during the course of treatment may allow for stepping up the level of activity, or more important, may signal changes that occur in other joints as a result of fatigue or overuse. Knowledge of these data may help prevent injuries to other joints or limbs. Recently, studies have been conducted to evaluate joint motion during therapeutic exercises.[16-21] The kinematics of various therapeutic exercises are discussed in the biomechanics of rehabilitation chapter.

Kinetic and kinematic gait analysis techniques have been combined using an inverse dynamics technique to estimate net joint moments and power. This technique has been used to evaluate tibial plateau leveling osteotomy and a lateral fabella-tibial technique to stabilize stifle joints in a cranial cruciate ligament transection model,[22] Labrador retrievers with and without cranial cruciate ligament disease,[23] and forelimb joints.[24] This form of gait evaluation is time-consuming and requires specialized software, but it can provide valuable information.

Pedometers

Most outcome assessment measurements are performed in a clinic in which equipment and personnel are located. However, in many cases, the animal temporarily becomes more active in the clinical environment as a result of traveling, exposure to other animals, and being in a strange environment. In these situations the dog often appears to be better than has been reported in the home environment. A pedometer may give some indication of a dog's activity level in its home environment. Most pedometers have a pendulum that swings back and forth with the normal gait cycle and indicates the number of steps taken. In our experience pedometers may be up to 85% to 90% accurate with regard to counting the number of steps taken by a dog. One study confirmed that pedometers are reasonably accurate, with the exceptions that at a walk, pedometers overestimated the number of steps by approximately 17% in large and medium dogs and underestimated actual number of steps by approximately 7% in small dogs.[25] Although these researchers found that no significant differences between pedometer-recorded and actual number of steps were detected when dogs trotted or ran, our experience has been that the step count is artificially increased when dogs run or climb stairs.

Proper placement of the pedometer is crucial. We have found that placing the pedometer just proximal to the elbow most accurately indicates the number of steps taken



Figure 13-6 Proper placement of a pedometer is crucial. Placing the pedometer just proximal to the elbow is one method that has been used to count the number of steps taken.



Figure 13-7 Joint angles of flexion and extension are measured using a goniometer.

(Figure 13-6). In the study by Chan et al., however, pedometers were worn around the dog's neck by means of an adjustable lightweight chain.[25] However, we have found that when dogs shake their head or scratch their neck, the step count on the pedometer is artificially increased. Because pedometers may not be not accurate in all dogs, it is recommended that the dog be walked 100 steps; the number of steps indicated by the pedometer is compared to see if the pedometer is reasonably accurate to record a dog's activity level. An additional test to determine accuracy is to place the pedometer and then allow the dog to have free activity while 100 steps are counted. Because the pedometer may double-count when the dog is trotting, running, or going up and down stairs, it is used only as a semiquantitative indicator of home activity. Environmental conditions are another major factor in the activity of dogs. Dogs that spend some time outdoors may not be as active if it is raining or cold, or the weather is otherwise inclement. Therefore comparative assessments should be made on days when the weather is as similar as possible.

Recently, accelerometers have been used to estimate activity in dogs. Because of the differences in technology between pedometers and accelerometers, placement at various sites on dogs can give acceptable results. One study found that placement on a collar in the ventral neck region gives acceptable results for home activity monitoring.[26] In addition, accelerometers were able to distinguish the intensity of activity in dogs.[27] Another group found that the use of accelerometers was able to detect an increase of 20% in home activity after treatment for osteoarthritis, while placebo-treated animals had no increase in activity.[28] Activity and location may also be monitored relatively inexpensively using GPS technology for pets.[29]

Joint Function

Joint motion may be evaluated using both objective and subjective assessments. The primary motion of a joint is the movement of bones as a whole, such as occurs with stifle flexion, and is termed physiologic or osteokinematic motion. The quantity of joint flexion and extension motion is measured using a goniometer (Figure 13-7). Unlike human range-of-motion measurements, the actual geometric angles are measured in dogs because the many different body types and conformation in individual dogs make estimation of the neutral or "0-degree" position difficult. Measuring the actual angle eliminates the need to estimate the "normal" standing angle; therefore there are no negative angles.

The maximum angles of extension and flexion are those angles of greatest joint excursion. Normal angles of maximum flexion and extension have been reported for the Labrador retriever and cat (see Appendix 1).[30,31] Excellent inter-tester and intra-tester reliability was reported in those studies. In addition, measurements made in dogs and cats had very good correlation with measurements made from radiographs.[30,31]

Measuring maximum angles may involve some discomfort. An animal experiencing discomfort is unlikely to use the limb at those angles while ambulating. Therefore measuring the comfortable range of motion may be more clinically applicable. To measure the comfortable range of motion, the joint is slowly flexed until the first indication of discomfort, such as tensing the muscles, pulling the limb

away, or turning the head slightly, is noted. The joint is then slowly extended until the first indication of discomfort is noted. These angles are recorded. The mean of three independent measurements is used to be certain that the measured angles are reproducible. Physical rehabilitation may influence the return of joint motion following surgery, such as cranial cruciate ligament stabilization surgery, and joint motion is associated with function; therefore, measuring joint motion is important.

The quality of joint motion is more subjective and involves the assessment of joint biomechanics, crepitus, and pain during motion. The more subtle motions occurring at the surface of the joints are termed accessory or arthrokinematic motions. Examples of these motions are glide (slide), roll, spin, distraction or traction, and compression or approximation. Glides are shear or sliding motions of opposing articular surfaces. A normal amount of glide occurs in normal functioning joints. Glides at joint surfaces often are imposed interventions using joint mobilization techniques to regain normal motion in a joint with pathology. Joint surface geometry, soft tissue resistance, and external forces all affect glide. Rolls involve one bone rolling on another, such as the femoral condyles rolling on the tibial plateau. Gliding motion in combination with rolling is needed for normal joint motion. Spins are joint surface motions that result in continual contact of a single area of articular cartilage on adjacent articular cartilage within a joint. Distraction or traction accessory motions are tensile (pulling apart) movements between bones. Compressive or approximation accessory motions are compressive (pushing together) movements between bones. The quality of the end feel during joint motion may indicate abnormalities such as restriction by fibrous tissue, excess joint capsule, bone, or cartilage. Crepitus is often associated with surface irregularities in articular cartilage or periarticular changes, such as occur in osteoarthritis. The sensations of crepitus palpated during joint motion have been described as being similar to crinkling a piece of cellophane. Other sensations such as cracking, snapping, or popping may indicate abnormalities such as a torn meniscus.

Joint Laxity

Assessment of joint laxity in dogs is generally limited to qualitative evaluation, although some quantitative evaluations have been performed. In general joint laxity may occur as a result of developmental conditions (e.g., hip dysplasia), trauma, or pathologic degeneration of ligaments such as the cranial cruciate ligament.

Damage to collateral supporting structures is most easily assessed by placing varus and valgus stresses on the affected joint. The joints most commonly affected with collateral ligament damage include the hock, stifle, carpus, elbow, and shoulder. The digits are occasionally involved. Assessment is most accurate when the joint is placed in full extension to prevent inadvertent internal or external rotation of the joint that may be mistaken for varus or valgus movement. Breed and age differences exist regarding normal varus and valgus motion, especially in young animals, so comparison with the contralateral normal limb is helpful.

Hip joint laxity is most commonly assessed in young, growing dogs as an indication of early hip dysplasia. Subjective assessment is commonly performed using the Ortolani maneuver to create subluxation of the hip joint. A more quantitative method of assessing hip joint laxity is the use of PennHIP, in which controlled pressure is applied to the hip joint to create subluxation. A radiograph is made with the coxofemoral joint in this position, and measurements are made to quantify the degree of laxity, known as the hip distraction index. The hip distraction index has correlation with the development of hip dysplasia in some breeds of dogs.

Stifle joint laxity is clinically assessed by palpating for cranial drawer motion. There should be no drawer motion in normal dogs, with the exception of puppies, which have some drawer motion that comes to an abrupt stop at the end of drawer. Methods to quantify drawer motion, which are not commonly used in dogs, include measurement of direct cranial drawer, quantitative cranial tibial thrust, and use of instrumented devices.[32] Direct cranial drawer is determined by simply marking the position of the tibial tuberosity with the stifle in reduction on a piece of graph paper, and then remarking the position of the tibial tuberosity with full cranial drawer. Caution must be used to prevent motion of the femur and to keep the marking device perpendicular to the graph paper. Quantitative cranial tibial thrust is measured in a similar fashion, except that the stifle is kept in a fixed position (usually standing angle) while the hock is flexed. This results in cranial displacement of the tibial tuberosity if the cranial cruciate ligament is ruptured. Marks are made on graph paper with the tibia in a reduced position and with the tibia in full cranial tibial thrust. Care is taken to keep the caudal aspect of the femur in contact with a solid surface so the only motion that occurs is the cranial displacement of the tibia.

Instrumented devices for knee-drawer tests have been used in humans to measure shifts of the tibial tuberosity relative to the patella.[33-35] The total anterior and posterior displacement produced by anterior and posterior loads of 20 lb is measured from a reference position. Studies have evaluated the effects of variables on the accuracy and reproducibility of anterior-posterior drawer measurements. Reproducibility is principally affected by deviations in subject positioning. In addition to drawer motion, inadvertent knee flexion and tibial rotation may occur and result in incorrect measurements, despite attempts to prevent these. The effects of different observers, time sequences, different days, and muscle relaxation have been studied and contribute to error rates of 5% to 15%. Stress

radiography, a relatively sensitive method of measuring drawer motion, has been used to compare instrumented systems in humans with tears of the anterior cruciate ligament (ACL). In one study stress radiography was superior to instrumented arthrometer testing for determining cruciate ligament status.[36] Similar studies have not been performed in dogs, but instrumented knee-drawer devices are difficult to use in dogs and require practice to obtain reproducible results. Pediatric devices may be easier to use because the smaller size may allow a better fit of the instrument to the dog's limb.

Muscle Mass and Strength

Assessment of muscle is important in physical rehabilitation. Muscle mass, muscle strength, and muscle injury may be assessed to help evaluate the patient's progress. Regaining muscle mass and strength following injury is important to help improve function and prevent further injury to joints and other soft tissues. The degree of muscle atrophy is also a reasonable indication of limb use. Therefore careful attention to muscle assessment provides a great deal of information regarding the progress of the rehabilitation patient.

Muscle Mass

Muscle mass measurements are a useful outcome measure in veterinary rehabilitation. Muscle mass indicates limb use and is associated with muscle strength. Muscle mass may be estimated with limb circumference measurements, ultrasound, computed tomography (CT), magnetic resonance imaging (MRI), and dual-energy x-ray absorptiometry (DEXA).

Measurement of limb circumference is an indirect method of assessing changes in muscle mass and is inexpensive, quick, and easily performed on clinical patients. Acceptable results depend on using standard, repeatable methods of measuring limb circumference. A measuring tape with a spring tension device is useful in measuring limb circumference to improve consistent tension on the tape when making measurements (Figure 13-8).

One study evaluated the effect of limb position, clipping hair, sedation, and different evaluators on thigh circumference measurements at two different locations before and after stifle stabilization surgery.[37] Thigh length was determined by measuring from the tip of the greater trochanter to the distal aspect of the lateral fabella. Circumference was determined at points equal to 50% and 70% of thigh length, measuring from the greater trochanter distally. Measurements were technically easier when made at the 70% location because the skin of the flank did not impede measurements (Figure 13-9). Clipping had little effect on thigh circumference measurements, but dogs with short hair coats were used (Figure 13-10). Measurements may be affected to a greater extent in dogs with long hair.



Figure 13-8 A measuring tape with a spring tension device is used to measure limb circumference. Use of this instrument allows the consistent placement of tension on the tape when making measurements.

Limb position is important. Full flexion of the stifle results in greater thigh circumference measurements as compared with measurements made with the limb placed at a functional standing angle or with the stifle fully extended (Figure 13-11). Extension of the joint causes the muscle bellies to elongate, whereas flexion results in shortening and bunching of muscle bellies, creating greater thigh circumference. Measurements made in awake dogs with the stifle extended are not significantly different from those made following heavy sedation, as long as the dogs are not overly tense, probably because the muscles are in an elongated position when the stifle is extended (Figure 13-12). Thigh circumference measurements are sensitive enough to detect muscle atrophy 2 weeks after stifle surgery (Figure 13-13). Similar measurements may be obtained by independent evaluators as long as standard technique is used and the evaluators practice before obtaining actual measurements.

The association of limb circumference with actual muscle mass is important if it is to be useful in the evaluation of rehabilitation patients. Human male cadavers were subjected to comprehensive anthropometry and weighing of all skeletal muscle.[38] Limb circumference had good correlation with total skeletal muscle mass for the forearm ($r = .96$), mid-thigh ($r = .94$), calf ($r = .84$), and midarm ($r = .82$).

Thigh circumference also has significant correlation with actual muscle mass in dogs (Figure 13-14). Even greater correlation may be obtained when thigh length is

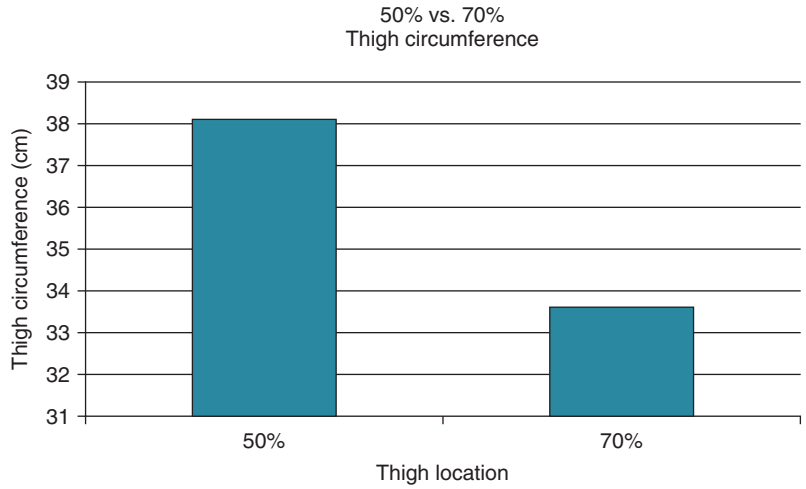


Figure 13-9 Thigh circumference measurements at the 50% and 70% thigh length. Although the 50% leg has greater thigh circumference because of greater muscle mass in that location, the measurements are technically more difficult at that location in some dogs because of the presence of the skin of the flank.

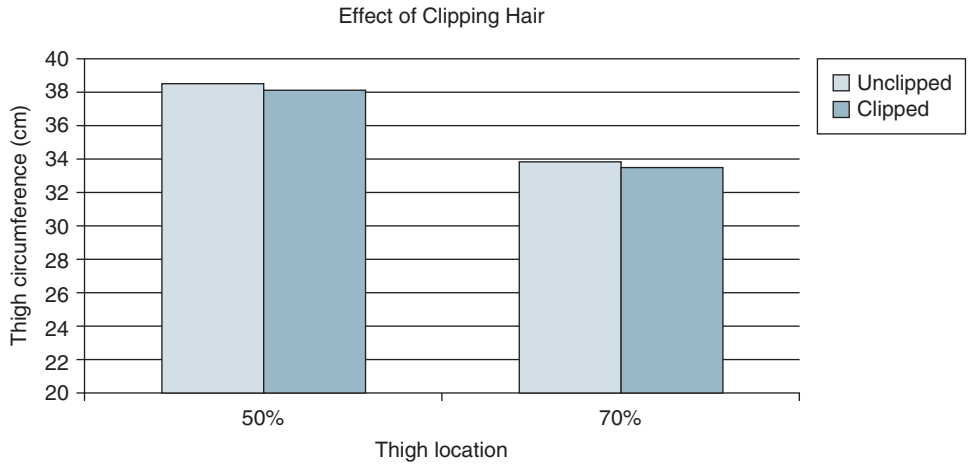


Figure 13-10 Clipping the hair has little effect on thigh circumference measurements in dogs with short hair coats. The effect of clipping would likely be greater in dogs with long hair.

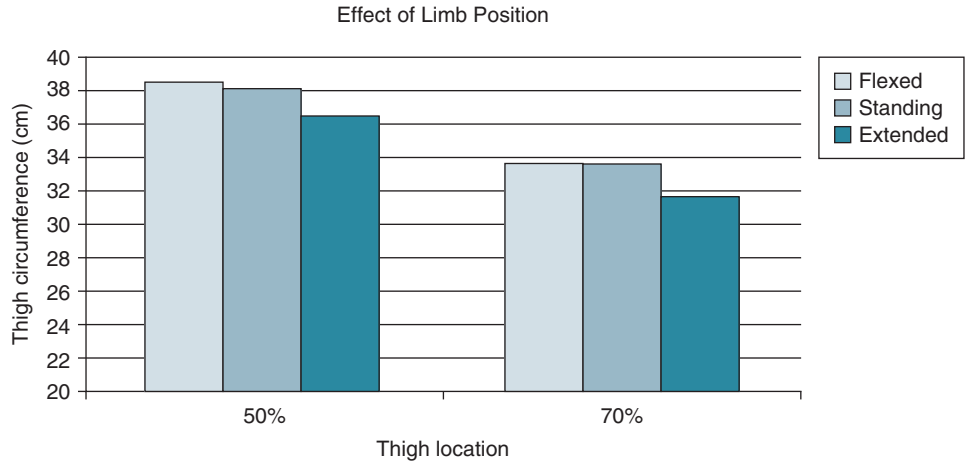


Figure 13-11 Thigh circumference is less with extension of the stifle because the muscles are elongated. Flexion of the limb results in greater thigh circumference, but the measurements are more difficult because of shortening of the hamstring muscles and difficulty in applying the tape measure.

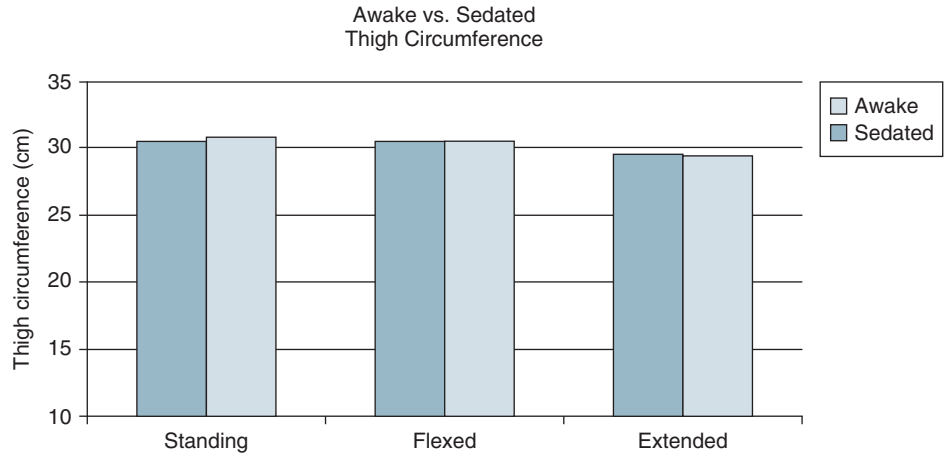


Figure 13-12 Measurements made in awake dogs with the stifle extended are not significantly different from those made following heavy sedation if the patient is not very tense, probably because the muscles are in an elongated position when the stifle is extended.

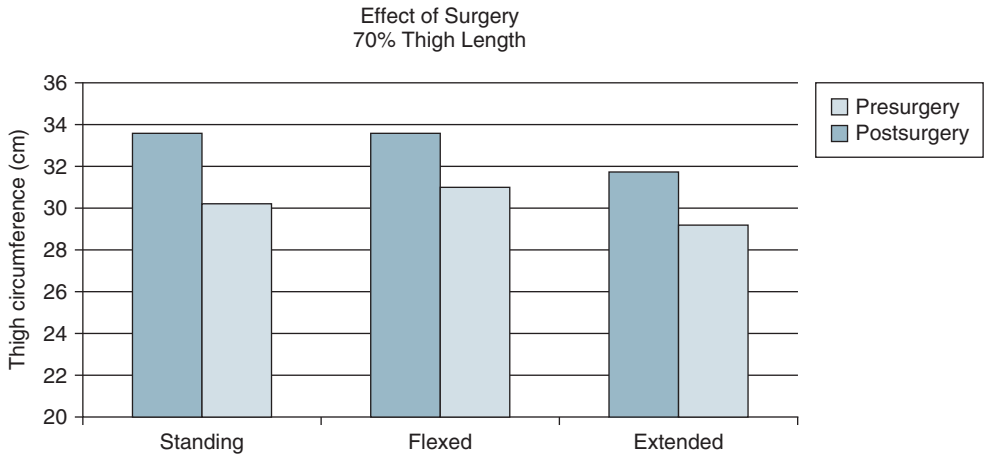


Figure 13-13 Thigh circumference measurements are sensitive enough to detect muscle atrophy 2 weeks after stifle surgery, indicating that this technique may be useful as a simple, cost-effective method of assessing limb mass.

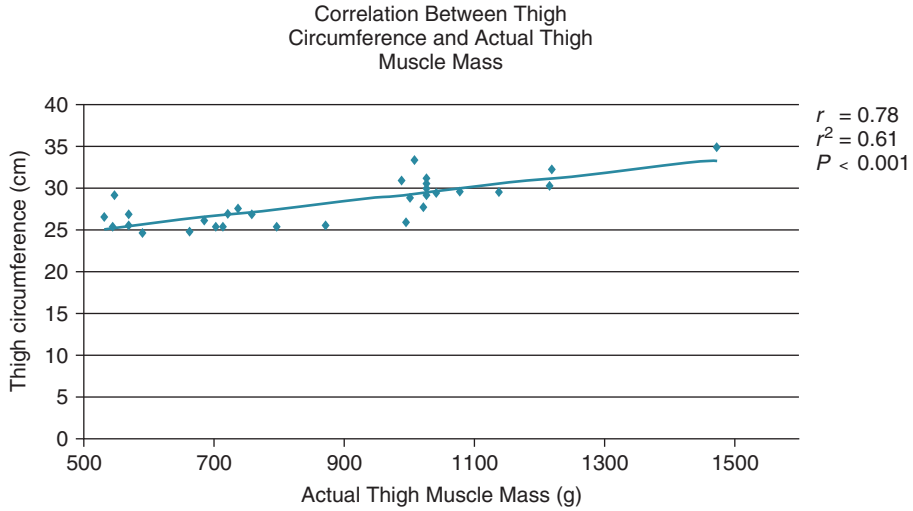


Figure 13-14 Thigh circumference has significant correlation with actual thigh muscle mass in dogs.

also considered and the volume of a cylinder is calculated. Although this method is simplistic because the thigh is not a true cylinder, the volume of a cylinder is easy to calculate and this estimate of muscle mass may be more clinically useful. Similar principles likely exist for circumference measurements of other limbs. Currently we prefer performing thigh circumference measurements with the hair short or clipped, the limb held in an extended position, at 70% of the length of the thigh, and with the animal relaxed, but not necessarily sedated. In the forelimb the length is measured from the proximal aspect of the olecranon to the distal aspect of the lateral styloid process; the limb circumference is measured at a point that is 20% of the limb length as measured from the proximal aspect of the lateral epicondyle of the humerus.

Limb circumference measures are commonly used in people to assess muscle atrophy, but relatively little is known regarding circumference measurement changes following knee injury and subsequent surgery. One study compared thigh and calf circumference measurements of affected and unaffected extremities before and after knee surgery for patients with acute and chronic knee injuries.[39] There were significant differences between affected and unaffected extremities at both the presurgery and postsurgery time periods of the acute and chronic groups. The bulk of the circumference measurement differences existed before surgery for both groups. In another study, the intrarater and inter-rater reliability of lower extremity circumference measurements in humans recovering from ACL reconstructive surgery demonstrated high correlation (0.82 to 1.0 and 0.72 to 0.97, respectively) for both the involved and uninvolved sides in several locations.[40]

Measurement of thigh circumference may be biased by the expectations of observers.[41] Use of anatomic landmarks and a tape measure with a spring tension device may help to minimize bias of the evaluator because the location of the measurement and the end tension of the tape can be standardized. A relatively small change in limb circumference may indicate much greater loss of muscle mass. A study of people with chronic unilateral patellofemoral pain indicated that although symptomatic limbs had significant reductions in limb circumference, very small reductions in thigh circumference (1%) were associated with significant reductions in muscle size (13%).[42]

The thickness of the skin and subcutaneous tissues may be another factor affecting thigh circumference measurements.[43] One investigation developed an equation using circumference and skinfold measurements for estimating anatomic cross-sectional area (CSA) of the quadriceps, hamstrings, and total thigh muscles.[44] Derived equations using midthigh circumference and anterior thigh skinfold assessment may be of value for estimating muscle CSA values when more sophisticated procedures are not available. Other studies have made similar assumptions that a cross-section of the thigh could be represented as a circle with concentric layers of skin, fat, muscle, and bone.[45] However, approximations of thigh muscle CSA may underestimate the fat-plus-skin compartment when calipers are used.

Although thigh circumference has been used as an effective method of measuring muscle size to evaluate the effect of an injury or effectiveness of an intervention, some studies in humans have shown this technique to be unreliable under certain conditions. Therefore other methods of measuring muscle size may be useful. Such modalities, although costly, include ultrasound scans, DEXA, CT, or MRI of muscles.

One study investigating the validity and reliability of measuring human quadriceps CSA with ultrasound scanning at the level of the mid-thigh found it to be a reliable method.[46] In a similar study, thigh circumference and segment volumes of the thigh were more strongly correlated to anterior versus posterior thigh musculature (when corrected for skinfold thickness) when B-mode ultrasound scanning was used.[43]

The question of whether various evaluation techniques are sensitive enough to detect changes in muscle mass was addressed in a training study of men.[47] Men were exposed to conditions designed to elicit differential hypertrophic adaptations following 21 sessions of squat training. A control group did no formal physical training. Tests used to evaluate muscle size included thigh circumference and quadriceps femoris and hamstring thicknesses via B-mode ultrasound. Thigh circumference and quadriceps femoris thickness were greater in groups that trained compared to than in controls.

Magnetic resonance imaging and computed tomography are promising reference methods for quantifying whole body and regional skeletal muscle mass. In people, arm and leg skeletal muscle CSA estimates obtained by standard MRI and CT methods had good correlation with corresponding cadaver values.[48] Magnetic resonance imaging used to estimate muscle CSA also had good correlation with actual cross-sections of anatomic specimens in other studies.[45,49] These findings strongly support the use of MRI and CT as methods to determine appendicular skeletal muscle mass in vivo.

Neurogenic muscle atrophy induced by crushing the sciatic nerve was examined using CT in dogs.[50] The CT number and CSA in denervated muscles decreased 1 to 2 weeks after denervation and were significantly less after 3 weeks. Examination with CT may be useful to evaluate neurogenic muscular atrophy.

Some work has compared various methods of muscle mass estimation in people. For example, the CSA and volume of the quadriceps femoris muscle using MRI was compared with B-mode ultrasound.[51] There was no significant difference in the CSA estimates or volume estimates when ultrasound and MRI were compared at several sites of the thigh.

Dual-energy x-ray absorptiometry is another noninvasive method of measuring lean tissue mass, but the estimations are generally limited to the whole body or an entire limb because it is difficult to determine lean tissue mass of relatively small regions. Although CT and MRI have been used to measure limb circumference and have the advantage that the area of large muscles may be measured, the equipment is expensive and interpretation must be performed by a person trained in these diagnostic modalities. Dual-energy x-ray absorptiometry is somewhat less expensive, and less training is required for interpretation of data, although patient positioning is critical to obtain accurate results.

The accuracy of DEXA for measuring total body fat–free mass and leg muscle mass at four leg regions was assessed in people.[52] Computed tomography of the legs was also performed. Fat-free mass and muscle mass by DEXA was positively associated with CT at all four leg regions (r^2 = .86 to .96), but the correlation of muscle mass with DEXA was higher than with CT in three regions. A comparison of DEXA with MRI to evaluate estimates of muscle and adipose tissue in human lower limb sections indicated high agreement between DEXA and MRI for muscle, but less so for adipose tissue.[53]

Muscle Strength

Little investigation has been performed in dogs regarding muscle strength estimates, likely because of the difficulty in making such measurements in animals. One study of Beagle dogs measured muscle strength as indicated by measuring maximum isometric extension torque of the hindlimb.[54] This required general anesthesia, a surgical approach to the thigh of the dog, placement of a stimulating electrode near the femoral nerve, and instrumentation of the limb to measure muscle torque while the muscle was electrically stimulated. Such invasive procedures are not clinically applicable. However, maximum isometric extension torque had strong correlation with muscle fiber diameter obtained from biopsies of the vastus lateralis muscle. Obtaining muscle biopsies is an invasive procedure, although less so than the technique for determining muscle torque, and less instrumentation is required.

Other studies of muscle strength of humans may provide information that may be applicable to dogs. One study determined the relationship between selected anthropometric dimensions and strength in resistance-trained athletes.[55] Athletes were measured following the completion of a 10-week resistance training program for one-repetition maximum lifts. The highest relationships existed between estimates of regional muscle mass (arm circumference, arm muscle CSA, and thigh circumference) and lifting performance. The fewer joints and muscle groups involved in a lift, the greater was the predictive accuracy from structural dimensions.

Although muscle mass is clearly associated with muscle strength, the type and strength of this relationship are less clear. The use of limb circumference measurements as an indication of muscle strength in humans is somewhat controversial. A predictive model was developed to evaluate the relationship of peak knee flexion and extension torque production to thigh circumference.[56] The authors concluded that peak knee torque production can be predicted with significant accuracy (r^2 = .78 to .87). A similar study assessed the correlation of maximal isometric force production of leg extensor muscles of elite human weight lifters with thigh circumference.[57] Maximal isometric force had significant correlation with thigh circumference, but surprisingly not with the mean muscle fiber area of the vastus lateralis muscle. The CSAs of fat, muscle, and bone tissues of limbs as well as maximal voluntary isokinetic strength were measured in men and women in another study.[58] Anatomic CSAs were determined by ultrasound of the upper arm and thigh. The isokinetic strength of the elbow and knee extensor and flexor muscles was measured using an isokinetic dynamometer. There was significant correlation between CSA and strength in all muscle groups except for the elbow extensors of the men and the elbow flexors of the women.

Another study correlated thigh circumference, muscle CSA by MRI, and isokinetic strength in patients 48 months after surgery for ACL injury.[59] There was a significant 1.8% decrease in thigh circumference, an 8.6% decrease in quadriceps CSA area by MRI, and a 10% decrease in average quadriceps torque in the involved extremities as compared with the uninvolved extremities. A positive correlation was found between MRI CSA and quadriceps and hamstring peak torque in involved and uninvolved extremities. A positive correlation among thigh circumference, quadriceps, and hamstring peak torque was found in uninvolved extremities but not in operated extremities. Another study assessed whether measurements of thigh circumference of patients undergoing unilateral meniscectomies and rehabilitation exercises would be an indicator of muscle power.[60] In this study muscle power was not predicted from thigh circumference measurements. Another study also found that the changes in muscle strength were not directly correlated to limb circumference. In this study isometric quadriceps strength and thigh circumference were determined in humans before and after a unilateral strength-training protocol of the quadriceps muscles for 5 weeks.[61] There were no significant changes in the untrained thighs. The trained quadriceps increased their isometric strength by 15%, whereas they changed their CSA by only 6%. Quadriceps hypertrophy was underestimated by measurements of thigh circumference. Nevertheless CSA measurements may indicate improvement in strength produced by training.

Although limb circumference may be associated with muscle power and strength in some situations, other factors

may affect muscle strength besides muscle size. A single measurement of circumference in both thighs of subjects with unilateral injury may not be adequate to assess muscle function, but serial measurements over time may be of value as an index of muscle power. One study assessed the relationship between thigh circumference and muscle strength and power in noninjured and injured human subjects.[62] The correlation between the torque produced at the knee by the knee extensors and flexors and a single thigh circumference measurement was not significant. In contrast, repeated measurements over a 6- to 8-month period showed a significant relationship between change in thigh circumference and change in quadriceps power.

Muscle Injury

Evaluation of muscle injury during rehabilitation may provide important information, especially as it pertains to prevention of overuse injuries. Magnetic resonance spectroscopy (MRS) and MRI are powerful tools to study tissue biochemistry and provide precise anatomic visualization of soft tissue structures.[63] These techniques may be used to study exercise-induced muscle injury. Magnetic resonance spectroscopy measurements show an increase in the ratio of inorganic phosphate to phosphocreatine (Pi/PCr) after eccentric exercise. This increase could be due to either increases in extracellular Pi or resting muscle metabolism. Increased Pi/PCr is also seen during training programs and may indicate persistent muscle injury. Increased resting Pi/PCr with injury is not associated with altered metabolism during exercise. Elevations in resting Pi/PCr have been used to show increased susceptibility of dystrophic muscle to exercise-induced injury. Progressive clinical deterioration in dystrophic dogs is marked by impaired muscle metabolism, and the presence of low oxidative muscle fibers not seen in normal dogs. Unfortunately MRS is not commonly available for use in animals.

Magnetic resonance imaging shows changes following eccentric exercise that last up to 80 days after injury and can reflect muscle edema as well as longer lasting changes in the characteristics of cell water. Magnetic resonance imaging shows the precise localization of the injured area. Thus MRS can provide information on the metabolic response to injury, whereas MRI provides information regarding the site and extent of the injury. These tools are promising as aids in understanding exercise-induced muscle injury.

Body Composition

Assessment of body condition is important in rehabilitation patients because obesity has been associated with the exacerbation of some conditions, such as osteoarthritis. In addition many companion animals are obese or overweight, and this may affect performance and health. Conversely poor muscle mass may indicate inadequate nutrition, pathologic conditions, injuries, or poor conditioning. Morphometric techniques such as body condition scoring systems, commonly used in clinical practice, are simple, inexpensive, and generally reasonably accurate. Body conformation is assessed from the side and the dorsal aspect; the animal is also palpated over certain regions. These profiles are then compared with a standard and a body condition score is assigned (Figure 13-15). Other morphometric measures, including tape measurements of body areas, such as the cranial thoracic region and abdomen, are somewhat useful for assessing body composition, but because of the variation in body type in dogs, they may not be better than body condition scores.[64]

Although body condition scores are clinically useful, they are not as precise as other methods of noninvasive body composition assessments, such as DEXA analysis.[64] These units (Figure 13-16) measure lean body mass, fat, and bone mineral and are accurate within 2% of actual measurements. In addition many software programs allow the generation of body composition of various portions of the body, such as the left and right forelimbs and rear limbs. Although generally considered the gold standard for the noninvasive assessment of body composition, animals must be sedated or anesthetized to obtain information. Dual-energy x-ray absorptiometry units are relatively expensive and are limited to institutions at this time.

Skin caliper measurements, although having relatively good correlation to body composition in humans, are generally not very useful in dogs, perhaps because of differences in subcutaneous tissues and fat distribution.[64] Other methods of assessing body composition, such as diagnostic ultrasound, electrical impedance, and isotope dilution using the deuterium oxide method, have insufficient investigation, require specialized equipment, or are limited to research institutions.

Pain Assessment

Assessment of pain and discomfort is important in the physical rehabilitation of animals. Excessive discomfort may prevent or slow progress during treatment, but objective measurement of pain is difficult in animals because they do not verbalize the level of pain that they may be experiencing. Therefore pain assessment scores or questionnaires are generally used to evaluate behaviors that are believed to be associated with pain and discomfort (Box 13-2). In addition, ordinal or VASs are sometimes used in which owners are asked how painful they believe their pet is. Some physiologic parameters, such as heart rate, respiratory rate, and blood pressure, have been used to evaluate pain in the acute postoperative period, but these are generally not as useful for assessing pain in chronic conditions.

Multiple studies have examined the validity and clinical usefulness of pain scales and pain questionnaires. The

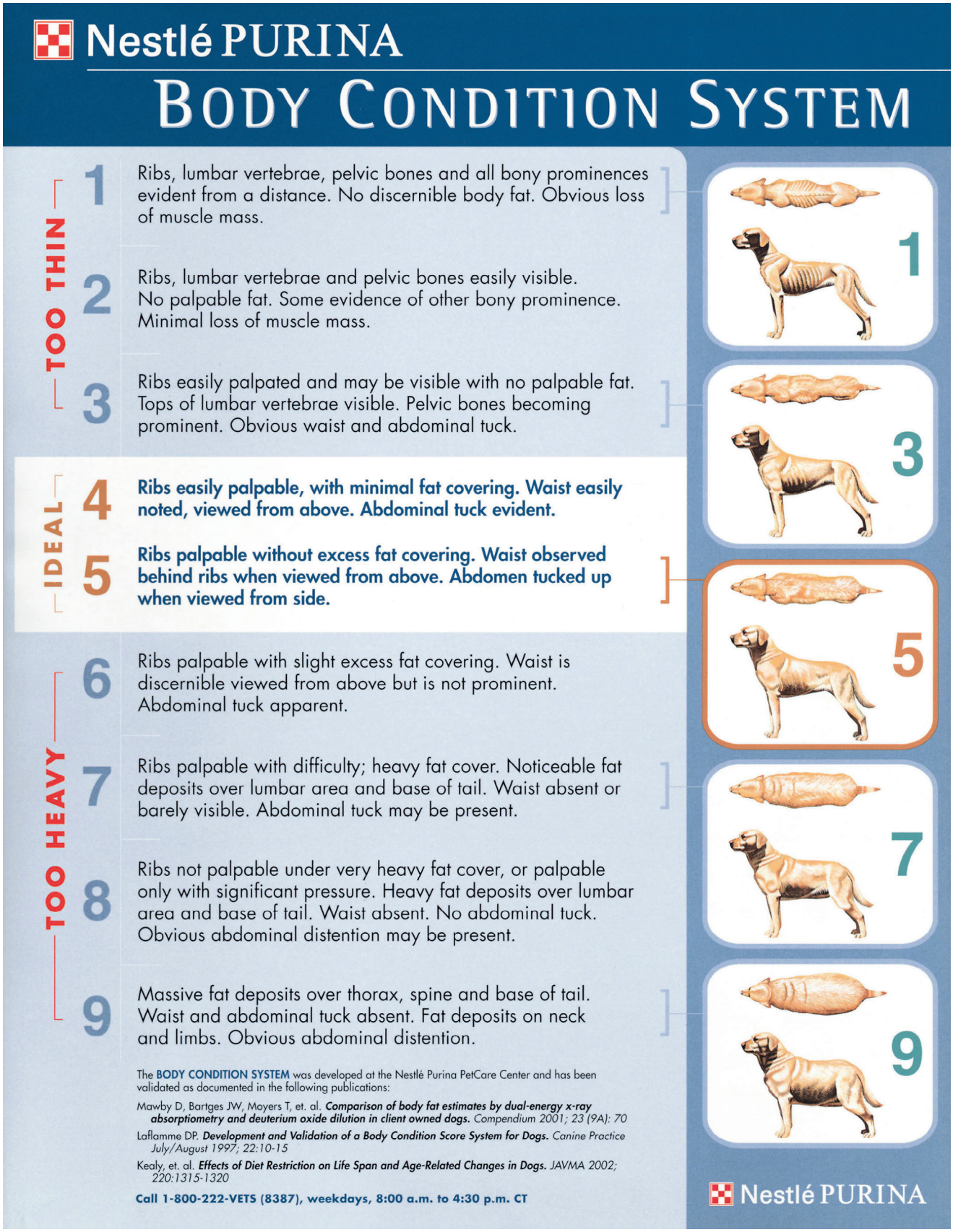


Figure 13-15 Body condition scores may be assigned to patients. The patient is assessed from the side and the dorsal aspect and the conformation is compared to a standard. *(Body condition score courtesy Iams Co., Dayton, OH.)*



Figure 13-16 A dual-energy x-ray absorptiometer may be used to measure lean body mass, fat mass, and bone mineral content.

Box 13-2 Simple Pain Assessment Score

0 No signs of pain during palpation of affected joint
1 Signs of mild pain during palpation of joint
2 Signs of moderate pain during palpation
3 Signs of severe pain during palpation
4 Dog will not allow examiner to palpate joint

canine brief pain inventory (CBPI) is based on the human Brief Pain Inventory (BPI) and has been shown to be reliable and valid in dogs with osteoarthritis,[12] and in a separate study on osteoarthritic dogs was shown to be sensitive enough to detect changes in pain with nonsteroidal antiinflammatory (NSAID) use.[13] The CBPI has also been validated for use in dogs with bone cancer.[65]

The Helsinki chronic pain index (HCPI) was created to assess chronic pain in dogs[11] (Table 13-1) and was found to be a valid and reliable tool for assessment of chronic pain by owners of dogs with osteoarthritis.[66] However, caution should be used with any subjective evaluation of pain in dogs with osteoarthritis. One study compared subjective evaluation of dogs receiving a placebo for the treatment of osteoarthritis and found that caregivers indicated that 40% of patients improved when evaluated by subjective measures, while measurement of ground reaction forces indicated no improvement.[67]

Other studies have evaluated the usefulness of questionnaires to evaluate acute pain in dogs. A short form of the Glasgow Composite Measure Pain Scale was able to identify those patients that required analgesic treatment from those that did not.[68] This pain scale evaluates six behavioral categories including vocalization, attention to wound, mobility, response to touch, demeanor, and posture/activity. Descriptions of the severity of each category are provided, and the items are placed in increasing order of pain intensity and numbered. The observer chooses the item within each category that best describes the dog's behavior and the scores are summed. The University of Melbourne Pain Scale incorporates heart rate, respiratory rate, and behavioral responses, such as response to palpation, activity, mental status, posture and vocalization. This scale is most useful in acute postoperative patients.[69]

Painful behavior in dogs may be indicated by whining, crying, or other forms of vocalizing when the animal moves or the affected body part is manipulated. Holding an affected limb in a tightly flexed or guarded position is frequently noticed after surgery. Animals may be particularly resentful of palpation and manipulation of the area. Painful behavior may also be exemplified by lying quietly in the back of a run or in the corner of a room with little desire to rise and move about. This may be a protective mechanism to avoid further injury and pain from moving about. The degree of pain in these patients is frequently underestimated because of their quiet nature.

Impressions of Owners and Veterinarians

Dogs and cats have different personalities and may respond very differently to various therapies. It is important for individuals who best know patients to evaluate their progress. For example, owners are often very aware of subtle changes in their pet's behavior. Although it is somewhat difficult to quantify these changes, these subjective findings often provide the therapist with important information regarding patient progress. If possible, the changes should be as objective as possible, such as recording the amount of time a pet spends playing rather than resting, measuring the distance that an animal is able to walk before needing to rest, or evaluating the length of time a neurologic patient is able to stand unassisted.

Owners should keep a log of daily activities to further define the progress of the patient at home. This may also provide incentive for owners to continue a home rehabilitation program if they can see the progress that is made from week to week and month to month. Poor compliance is a common reason for failure of a patient to progress. Regularly checking the log gives the veterinarian and owner an opportunity to evaluate the patient's progress, as well as make alterations to the rehabilitation program that account for an owner's ability to provide continued home care. In addition, videotaping the patient's activity on a regular schedule allows comparisons to be made over relatively long periods of time.

Return to Function

Return to function is perhaps the best indication of a successful outcome following treatment of an acute or chronic condition. The desired level of function depends on the intended use of the patient, which varies from a highly

Table 13-1 Pain Assessment in Dogs with Chronic Osteoarthritis

Question Topic	SCORES FOR DOGS WITH CHD Median	SCORES FOR DOGS WITH CHD Range	SCORES FOR CONTROL DOGS Median	SCORES FOR CONTROL DOGS Range	*P* Value
POSITIVE BEHAVIOR					
Appetite	0	0-3	0	0-2	0.59
Mood*	1	0-3	0	0-1	<0.001
Frequency of contact with human family members	1	0-2	0	0-2	0.086
Frequency of tail wagging	1	0-3	0	0-2	0.013
Activity	1.5	0-4	1	0-3	0.052
Play and games*	1	0-4	0	0-1	<0.001
NEGATIVE BEHAVIOR					
Excessive panting	1	0-4	0	0-2	<0.001
Licking of lips	0	0-4	0	0-3	0.97
Vocalization (audible complaining)*	1	0-3	0	0-1	<0.001
Vocalization when stretching hind legs caudally	2	0-4	0	0-1	<0.001
Aggressiveness toward humans	0	0-3	0	0-2	0.88
Aggressiveness toward other dogs	2	0-3	1	0-3	0.47
Aggressiveness toward dogs in its own pack	1	0-4	1	0-2	0.09
Submissiveness in the pack	1.5	0-4	2	0-4	0.27
LOCOMOTION					
Walking*	1	0-3	0	0-1	<0.001
Trotting*	1.5	0-4	0	0-1	<0.001
Pacing	1	0-4	2	0-4	<0.001
Galloping*	1	0-4	0	0-1	<0.001
Jumping*	2	0-4	0	0-1	<0.001
Climbing stairs	2	1-4	0	0-1	<0.001
Descending stairs	2	0-4	0	0-2	<0.001
Laying down*	2	0-4	0	0-1	<0.001
Getting up*	2.5	0-4	0	0-1	<0.001
Difficulty moving after rest*	2	0-4	0	0	<0.001
Difficulty moving after major activity*	3	1-4	0	0-1	<0.001
Chronic pain index (sum of answers to 11 questions)	19	7-35	2	0-5	<0.001

*Question selected for inclusion in chronic pain index. Values of $P < 0.005$ were considered significant.
Comparison of 25 questionnaire answers (scores, 0-4) provided by owners of 41 dogs with pain associated with canine hip dysplasia (CHD) with those provided by owners of 24 dogs with no pain.
From Hielm-Björkman AK, Kuusela E, Liman A, et al: Evaluation of methods for assessment of pain associated with chronic osteoarthritis in dogs. *J Am Vet Med Assoc* 222:1552-1558, 2003.

trained working dog or racing greyhound to a house pet. Some outcomes are relatively easy to quantify, such as winning a race in the same class that the animal raced in before an injury or being able to perform a particular task in the case of a working dog. Others are more nebulous, such as a return to acceptable house pet function. Regardless of the desired outcome, reasonable expectations must be set based on the severity of the condition being treated, and this must be conveyed to the owner to avoid future disappointment when a higher level of function is expected than may be achieved.

For example, it is unreasonable to expect an elderly arthritic dog to return to competitive coursing, but it may be reasonable to expect the dog to be able to play ball for short periods of time. In addition, expectations may need to be altered if the response to treatment is better or worse than expected. In severely affected neurologic patients, reasonable expectations should center on functional activities of daily living, such as being able to climb stairs to get in and out of the house, go outside to eliminate with minimal or no assistance, and take short walks with the owner.

Functional scales are used extensively in human medicine and rehabilitation in orthopedics, neurology, cardiopulmonary, pain, child development, and other areas. Functional scoring systems for specific injuries may be useful, such as a functional stifle score. Such scoring systems have been used for outcome assessment in people recovering from ACL surgery because an ACL may result in functional disability. Two of the most commonly used measures are the Lysholm Knee Scale and the Cincinnati Knee Rating Scale.[70,71] Although these scales are well validated in humans, the optimal method to measure the degree of functional disability is unknown in dogs. A useful system in dogs likely should incorporate limb use, lameness, stance, pain, muscle atrophy, range of motion, joint effusion, drawer motion, and functional activities to evaluate limb function following cranial cruciate ligament surgery (Table 13-2). A subjective and functional rating system in people related six activity levels to pain, swelling, giving-way, and overall activity.[72] In this study, activity level, symptoms, clinical laxity, meniscal damage, lower limb alignment, tibiofemoral crepitus, patellofemoral factors, rehabilitation, and patient compliance were identified as risk factors for future joint arthrosis.

Other functional tests that have been used in humans may be applied to dogs, with modification. For example, one study of people assessed the sensitivity of four different types of one-legged hop tests to determine alterations in lower limb function in ACL-deficient knees.[73] Comparisons were made between limb symmetry as measured by the hop tests and muscle strength, symptoms, and self-assessed function. Statistical trends were noted between abnormal limb symmetry on the hop tests and low-velocity quadriceps isokinetic test results. The time and distance that a dog will walk on the rear limbs with the forelimbs elevated (dancing) may provide similar information. Several easy to administer functional scoring scales have been developed for dogs with spinal cord injuries including one for acute injuries[74] (Box 13-3), and another that is based on the normal neurologic examination[75] (Box 13-4). An example is Figure 13-17 where dogs with intact deep pan recovered faster and to a greater degree than dogs without deep pain.

Box 13-3 Functional Scoring System in Dogs with Acute Spinal Cord Injuries

The five stages of recovery of use of pelvic limbs in dogs with spinal cord injuries. Each stage is subdivided on the basis of recovery patterns (i.e., yielding a scale of 0 to 14). Dogs were considered as weight-bearing when the full weight was born with joints extended for at least two steps (i.e., standing alone was not considered weight-bearing).

Stage 1

0 No pelvic limb movement and no deep pain sensation.
1 No pelvic limb movement with deep pain sensation.
2 No pelvic limb movement but voluntary tail movement.

Stage 2

3 Minimal non-weight-bearing protraction of the pelvic limb (movement of 1 joint).
4 Non–weight-bearing protraction of the pelvic limb with >1 joint involved <50% of the time.
5 Non–weight-bearing protraction of the pelvic limb with >1 joint involved >50% of the time.

Stage 3

6 Weight-bearing protraction of pelvic limb <10% of the time.
7 Weight-bearing protraction of pelvic limb 10% to 50% of the time.
8 Weight-bearing protraction of pelvic limb >50% of the time.

Stage 4

9 Weight-bearing protraction 100% of the time with reduced strength of pelvic limb. Mistakes >90% of the time (e.g., crossing of pelvic limbs, scuffing foot on protraction, standing on dorsum of foot, falling).
10 Weight-bearing protraction of pelvic limb 100% of the time with reduced strength. Mistakes 50% to 90% of the time.
11 Weight-bearing protraction of pelvic limb 100% of the time with reduced strength. Mistakes <50% of the time.

Stage 5

12 Ataxic pelvic limb gait with normal strength, but mistakes >50% of the time (e.g., lack of coordination with thoracic limb, crossing of pelvic limbs, skipping steps, bunny-hopping, scuffing foot on protraction).
13 Ataxic pelvic limb gait with normal strength, but mistakes made <50% of the time.
14 Normal pelvic limb gait.

From Olby NJ, De Risio L, Muñana KR, et al.: Functional scoring system in dogs with acute spinal cord injuries. *Am J Vet Res* 62:1628, 2001.

Quality of Life

Questionnaires to assess the quality of life of dogs have been an area of recent study. Dogs with spinal cord injuries have been studied and a questionnaire was developed and evaluated for owner-perceived quality of life. Results of this study[76] suggest that this questionnaire can be used to obtain owner-perceived quality of life assessments. A follow-up study[77] evaluated owner-perceived quality of life in 100 dogs with spinal cord injuries. An interesting finding was that ambulation was the factor that owners perceived as having the biggest impact on quality of life.

Health-related quality of life in dogs with cardiac disease has also been investigated.[78] Three hundred and sixty dogs were evaluated by veterinarians and scored in terms of clinical signs of cardiac disease. Owner questionnaires were significantly correlated with the FETCH (Functional Evaluation of Cardiac Health) questionnaire and with the ISACHC (International Small Animal Cardiac Health Council) classification. Results of this study suggest that the questionnaire is a valid and reliable method for assessing quality of life in dogs with cardiac disease. Owner questionnaires have also been studied to help

Table 13-2 Proposed Stifle Function Scale

Category	Section	Findings	Maximal Score	Score
Limb use at a Trot	Normal	No lameness and weight bearing on all strides	10	10
	Mild disuse	Lame but weight bearing on >95% of strides		6
	Moderate disuse	Lame but weight bearing on >50% and <95% of strides		4
	Severe disuse	Lame but weight bearing on <50% and >5% of strides		2
	Very severe disuse	Continuous non–weight-bearing lameness or weight bearing on <5% of strides		0
Limb use at a Walk	Normal	No lameness and weight bearing on all strides	10	10
	Mild disuse	Lame but weight bearing on >95% of strides		6
	Moderate disuse	Lame but weight bearing on >50% and <95% of strides		4
	Severe disuse	Lame but weight bearing on <50% and >5% of strides		2
	Very severe disuse	Continuous non–weight-bearing lameness or weight bearing on <5% of strides		0
Lameness at a trot	None	Normal locomotion	10	10
	Slight	Trots with a slight (barely perceptible) lameness, but strides appear to have normal length		8
	Mild	Trots with a mild lameness, but strides appear to have normal length		6
	Moderate	Trot with a moderate (obvious) lameness or a shortened stride length on affected side when trotting, but is bearing weight on that limb		4
	Severe	Is intermittently non–weight-bearing on that limb when trotting		2
	Very severe	Is completely non–weight-bearing on that limb when trotting		0
Lameness at a walk	None	Normal locomotion	10	10
	Slight	Walks with a slight (barely perceptible) lameness, but strides appear to have normal length		8
	Mild	Walks with a mild lameness, but strides appear to have normal length		6
	Moderate	Walks with a moderate (obvious) lameness or shortened stride length on affected side, but is bearing weight on that limb		4
	Severe	Is intermittently non–weight-bearing on that limb when walking		2
	Very severe	Is completely non–weight-bearing on that limb when walking		0
Stance	Normal	Stands with equal weight on both pelvic limbs	10	10
	Mild asymmetry	Bears less weight on the affected pelvic limb or limb trembles when standing		6
	Moderate asymmetry	Puts limb down for balance but bears weight <10% of normal weight		3
	Severe asymmetry	Does not bear weight on affected limb while standing		0

Continued

Table 13-2 Proposed Stifle Function Scale—cont'd

Category	Section	Findings	Maximal Score	Score
Stair climbing	Normal	No difficulty	5	5
	Mild	Has slight difficulty climbing steps		3
	Moderate	Skips steps or bunny hops		1
	Severe	Cannot climb stairs		0
Sitting and standing	Normal	Easily goes from a sitting to standing or a standing to sitting position. Sits and rises squarely.	5	5
	Mild	Sits or stands with some difficulty (slight hesitation or delay)		4
	Moderate	Sits or stands with difficulty (hesitation or delay)		2
	Severe	Cannot sit or stand without assistance		0
Dancing	Normal	Moves freely forward and backward	5	5
	Mild anomaly	Resists moving forward and backward		3
	Severe anomaly	Unable to bear weight on pelvic limbs during forward and backward dancing motion		0
Pain response (palpation)	None	No pain response is elicited during palpation of the joint	5	5
	Mild	Mild pain response (i.e., head turning) is elicited during palpation of the joint		3
	Moderate	Moderate pain response (i.e., slight vocalization, increased reaction) is elicited during palpation of the joint		2
	Severe	Severe pain response (i.e., immediate reaction, loud vocalization, attempt to bite) is elicited during palpation of the joint		0
Stifle Effusion	None	No effusion of stifle	5	5
	Mild	Slight loss of patella ligament distinctness		3
	Moderate	Patella ligament not distinct		2
	Severe	Cannot distinguish patella ligament due to effusion		1
Muscle atrophy	None	Normal muscle mass	10	10
	Mild	If disease is unilateral, thigh girth is 0% to 5% smaller than the opposite limb. Otherwise, a slight atrophy of the biceps femoris or other thigh muscles is noted/palpated.		6
	Moderate	If disease is unilateral, thigh girth is 6% to 10% smaller than the opposite limb. Otherwise, a moderate atrophy of the biceps femoris or other thigh muscles is noted/palpated.		4
	Severe	If disease is unilateral, thigh girth is >11% smaller than the opposite limb. Otherwise, a severe atrophy of the biceps femoris or other thigh muscles is noted/palpated.		0
Joint motion* (extension)	Normal or increased	Extension 160° or more	5	5
	Mild loss	Extension 150° to 159°		3
	Moderate loss	Extension 140° to 149°		1
	Severe loss	Extension <139°		0

Table 13-2 Proposed Stifle Function Scale—cont'd

Category	Section	Findings	Maximal Score	Score
Joint motion* (flexion)	Normal	Flexion 45° or less	5	5
	Mild loss	Flexion 46° to 50°		3
	Moderate loss	Flexion 51° to 60°		1
	Severe loss of flexion	Flexion >60°		0
Drawer Motion or Cranial Tibial Thrust (for TPLO or TTA)	None	Less than 2 mm	5	5
	Mild	2-4 mm		3
	Moderate	5-7 mm		1
	Severe	.7 mm		0
Total score				100

*Jaegger G, Marcellin-Little DJ, Levine D: Reliability of goniometry in Labrador retrievers. *Am J Vet Res* 63:979-986, 2002.

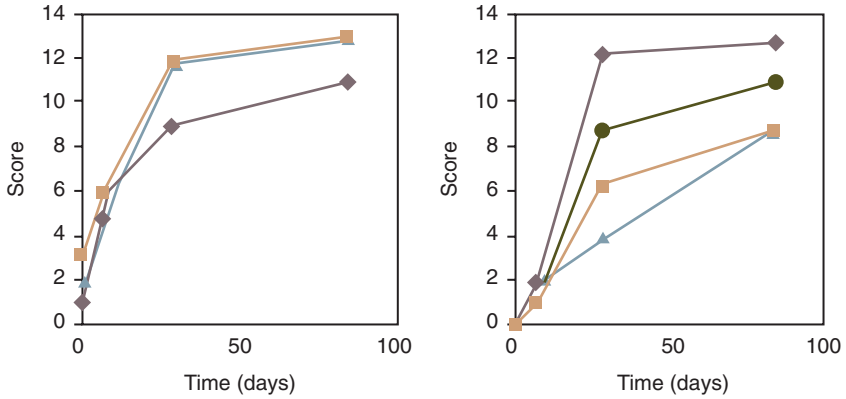


Figure 13-17 Typical course of recovery of pelvic limb function during a 3-month period in five dogs with spinal cord injuries that were nonambulatory paraparetics or paraplegics with intact deep pain sensation (left) and without deep pain sensation (right). Recovery scores are based on the system described in Box 13-3. (A score of 0 is poor, with no limb movement or deep pain; a score of 14 is best, with recovery of normal pelvic gait.) (From Olby NJ, De Risio L, Muñana KR, et al.: Functional scoring system in dogs with acute spinal cord injuries. *Am J Vet Res* 62:1628, 2001.)

Box 13-4 Texas Spinal Cord Injury Score for Dogs

Gait[a]

0 No voluntary movement seen when supported
1 Intact limb protraction with no ground clearance
2 Intact limb protraction with inconsistent ground clearance
3 Intact limb protraction with consistent ground clearance (>75%)
4 Ambulatory, consistent ground clearance with moderate paresis-ataxia (will fall occasionally)
5 Ambulatory, consistent ground clearance with mild paresis-ataxia (does not fall, even on slick surfaces)
6 Normal gait

Proprioceptive Positioning[b]

0 Absent response
1 Delayed response
2 Normal response

Nociception[c]

0 No deep nociception
1 Intact deep nociception, no superficial nociception
2 Nociception present

[a]Ground clearance refers to the ability to lift the limb off of the ground when it is being protracted.
[b]Proprioceptive positioning is performed by supporting the dog's weight and gently placing the dorsum of the paw on the ground. A delayed response is indicated by a >2-second lag between paw placement and correction.
[c]Deep nociception is measured by cross-clamping the distal limb or nail bed with hemostats. Superficial nociception is tested by pinching the interdigital webbing with hemostats.
From Levine GJ, Levine JM, Budke CM, et al: Description and repeatability of a newly developed spinal cord injury scale for dogs. *Prev Vet Med* 89:121-127, 2009.

Table 13-3 Descriptive Terms Used in the Development of a Quality of Life Scale in Dogs with Chronic Pain

Domains	Negative Descriptors	Positive Descriptors
Activity	Apathetic, apprehensive, lackluster, lethargic, listless, reluctant, sleepy, slowed, sluggish, tired weary	Active, boisterous, bouncy, energetic, lively, playful, tireless
Comfort	Complaining, groaning, moaning, pained, sore, stoic, uncomfortable	Comfortable, stretching
Appetite	Off food, picky (with regard to food)	Enthusiastic about food, greedy, interested in food, thirsty
Extroversion-introversion	Detached, quiet, subdued, unresponsive, unsociable, withdrawn	Affectionate, bold, curious, eager, excitable, friendly, fun loving, nosy, outgoing, sociable
Aggression	Aggressive, grumpy, irritable, territorial or protective	Good-natured, even tempered, placid
Anxiety	Anxious, cautious, distressed, frightened, nervous, panicky, strained, uneasy, upset	Accepting, easygoing, laid-back
Alertness	Depressed, dull, confused, uninterested	Alert, bright, inquisitive, interested, keen, obedient
Dependence	Attention seeking, clingy, comfort seeking, pathetic or pitiful	Confident, independent
Contentment	Miserable, sad, sorrowful, resigned, unhappy	Contented, happy
Consistency	Inconsistent	Consistent
Agitation	Agitated, crying, disturbed, panting, restless, unsettled, whining	Calm, at ease
Posture-mobility	Awkward, limping, stiff	Athletic, fit, relaxed
Compulsion	Compulsive	No terms

From Wiseman-Orr ML, Nolan AM, Reid J, Scott EM: Development of a questionnaire to measure the effects of chronic pain on health-related quality of life in dogs. *Am J Vet Res* 65;1077-1084, 2004.

determine the quality of life for dogs with chronic pain.[79] These types of measures may guide decision making and treatment interventions (Table 13-3).

Summary

Assessment of patients is important to document the benefits of a rehabilitation program and help to improve protocols. Assessment techniques may be relatively simple and inexpensive, or more elaborate with expensive equipment. New assessment techniques must be developed to allow adequate comparison of a patient's progress over time and make specific changes in protocols to achieve the best possible outcome.

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14 Rehabilitating the Painful Patient: Pain Management in Physical Rehabilitation

Steven M. Fox and Robin Downing

Divinum est opus sedare dolorum.
(Divine is the work to subdue pain.)

Galen

Although pain in canine patients is inevitable in the face of surgery, trauma, injury, osteoarthritis, and so on, suffering is optional. It is well established that uncontrolled pain is not only ethically problematic, but physiologically damaging as well.[1] In recent years pain management modalities have been developed in human medicine to address the evolving understanding of the complexities of the pain experience. Many of these pain treatment strategies have been imported into veterinary medicine. Pain and suffering should be sought out, the cause identified to the greatest degree possible, and alleviated when feasible.

Canine physiotherapy/rehabilitation is a discipline that encompasses the application of physical therapy techniques (developed in humans) to dogs whose comfort and function have been compromised in some way. A wide variety of modalities and methods can be applied, from the most highly technical and advanced techniques used for complex orthopedic surgery recoveries, to simple therapeutic techniques that can be taught to pet owners for use at home. The overall goal is to restore, maintain, and promote optimal function, optimal fitness, wellness, and quality of life as they relate to movement disorders and health.

Often the patient who presents for rehabilitation is reluctant to move and exercise. This reluctance may be caused by the patient's unwillingness, inability, or a combination of these factors. It is important to understand that most of the dogs who are candidates for physiotherapy and rehabilitation are painful. It is equally important to remember that as veterinary health care providers, we have an obligation to provide compassionate care. If the pain of a rehabilitation patient is not appropriately or adequately addressed, then physiotherapy will be problematic. Consequences of unresolved pain may include:

- Unnecessary suffering for the painful patient
- Tissues that are unable to heal normally
- Restriction of range of motion and other tissue dysfunction from the influence of pain signals
- Undermining the trust inherent in the family–pet relationship
- Tissue contraction/atrophy from disuse driven by pain
- Resentment on the part of the patient of all caregivers
- The transition/transformation from adaptive pain that serves a purpose to a maladaptive pain state in which the pain itself is pathologic

An underlying tenet of veterinary medicine is "Do No Harm," and this applies equally to physical rehabilitation. Fundamental to the success of any physical rehabilitation regimen is the control of pain. And fundamental to the success of controlling pain is the understanding that a multifaceted (also called multimodal) approach appears to provide optimal outcome. The goal for successful integration of pain management and physical rehabilitation is to build a pain management plan that allows for maximum comfort to open the door for maximum movement.

Pain Processes

There are excellent sources for in-depth discussions of pain physiology and pathophysiology.[2-4] Within the context of physical rehabilitation it is important to have an understanding of basic pain processes, as this information will influence treatment choices for individual patients. For instance, patients experiencing acute pain following orthopedic surgery have different needs than the elderly dog experiencing the chronic maladaptive pain associated with long-standing osteoarthritis. Practitioners must appreciate the differences among patients and their pain experiences so that they may create individualized pain management strategies. Likewise, practitioners must understand properties unique to different drugs and drug classes as well as these drugs' mode of action to create a targeted approach to pain.[5]

Adaptive and Maladaptive Pain

Pain can be divided into two general categories recognized as adaptive and maladaptive. Adaptive pain protects the body from injury and promotes healing by inhibiting activity when injury has occurred. In contrast, maladaptive pain reflects pathologic activity of the nervous system—the

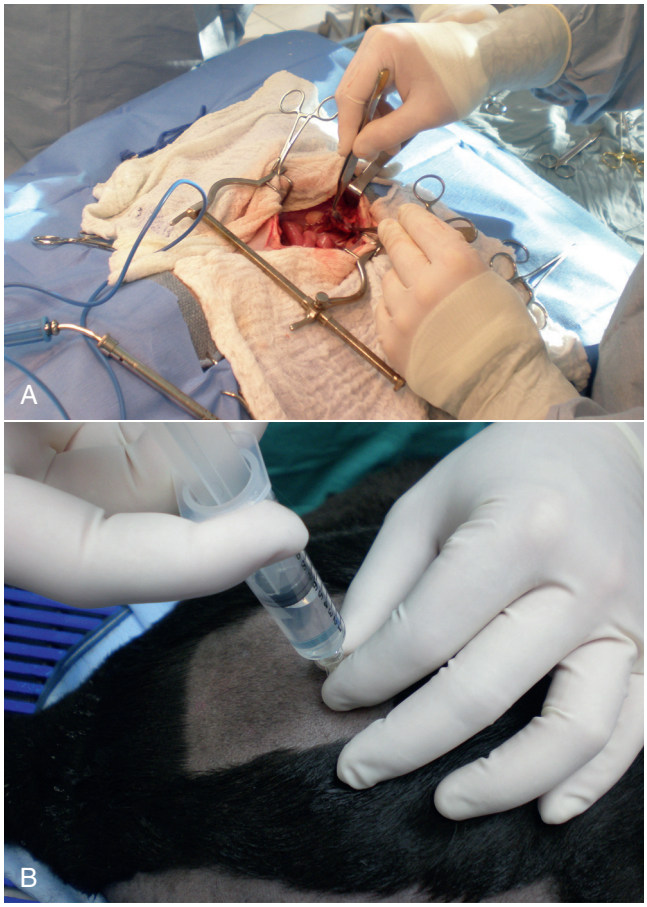


Figure 14-1 Examples of acute pain: surgery (*A*) or a needle insertion (*B*).

result of complex processes of neuronal plasticity—pain as a disease state.[5,6]

Adaptive, everyday acute pain (Figure 14-1) is also known as nociceptive or physiologic pain and occurs when noxious stimuli (mechanical, thermal, or chemical) are transduced into electrical signals that are then transmitted to the spinal cord. These signals undergo modulation in the spinal cord before being relayed to the brain, where the conscious brain interprets these transmissions, resulting in pain perception. Adaptive pain is purposeful. Dr. Frank Vertosick states, "Pain is a teacher, the headmaster of nature's survival school."[7] Acute pain tends to be of relatively short duration and, if addressed properly, is relatively easy to treat.

Chronic maladaptive pain may result from sustained noxious stimuli such as ongoing inflammation or profound tissue damage, or it may be present independent of the inciting cause. Regardless of its etiology, maladaptive pain offers no or little useful biologic function or survival advantage. The nervous system itself becomes the source of the pathology and contributes to patient morbidity. Effective treatment for maladaptive pain can be an enigma. A number of studies have shown that the longer a pain lingers, the harder it is to eradicate because of resultant changes to both the peripheral and central nervous system.

Chronic pain was traditionally defined as pain lasting more than 3 or 6 months, depending on the source of the definition.[8,9] More recently, chronic pain has been defined as "pain that extends beyond the period of tissue healing and/or with low levels of identified pathology that are insufficient to explain the presence and/or extent of pain."[10] There is no general consensus on the definition of chronic pain. In clinical practice it is often difficult to determine when acute pain has become chronic—when adaptive pain crosses the threshold to become maladaptive.

Adaptive to Maladaptive Pain

A progression from adaptive to maladaptive pain can be described as a spectrum composed of three major stages or phases of pain, suggesting that different neurophysiologic mechanisms are involved, depending on the nature and time course of the originating stimulus.[11] These three phases are (1) the processing of a brief noxious stimulus, (2) the consequences of prolonged noxious stimulation, leading to tissue damage and peripheral inflammation, and (3) the consequences of neurologic damage or changes, including peripheral neuropathies and central pain states. This spectrum of architectural change within the nervous system and the resultant pain pathology may be represented graphically as in Figure 14-2.

The journey of the nervous system toward maladaptive pain is often described as windup. Windup describes activity-dependent plasticity characterized by a progressive increase in action potential output from dorsal horn neurons elicited during the course of prolonged, repeated low-frequency C-fiber or nociceptor stimuli.[12,13] One receptor critical to this process is the *N*-methyl-D-aspartate (NMDA) receptor. NMDA receptors are only activated in the face of prolonged depolarization such as occurs with chronic pain.[14] Their activation can cause neural cells to sprout new connective endings,[15] thus expanding the receptive fields of the painful patient. Accompanying windup, less glutamate is required to transmit the pain signal and more antinociceptive input is required for analgesia. The complexity of maladaptive pain and the phenomenon of windup continues to provoke ongoing study. Studies have implicated activated microglia, norepinephrine (NE) levels, serotonin (5-HT) activity, as well as transient receptor potential (TRP) receptors as playing a role in maladaptive pain.

The rehabilitation practitioner must grasp the concept of this transition from adaptive to maladaptive pain in order to understand how best to address the pain management needs of the patient that is presented for physiotherapy. There are multiple potential targets of analgesic treatment in the patient experiencing both adaptive and maladaptive pain. It is appropriate, when putting together the pain management strategy for a specific patient, to address whatever targets

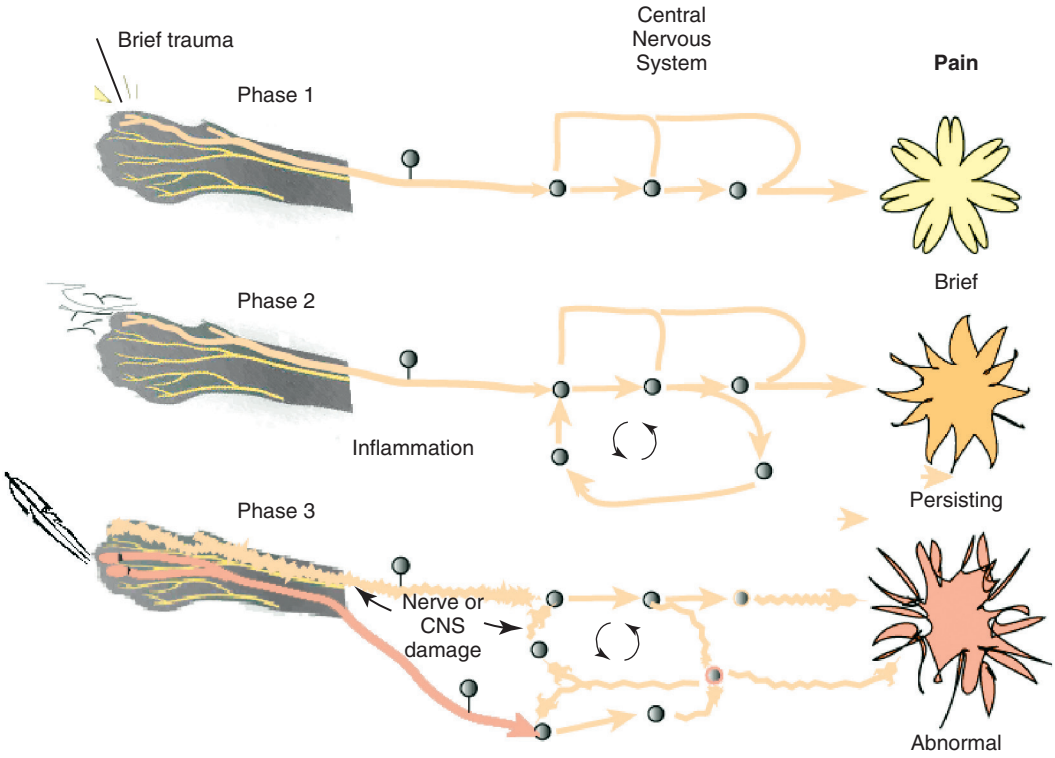


Figure 14-2 Proposed pain states as pain transitions from adaptive to maladaptive.

of therapy may be relevant. The rehabilitation practitioner *must* work in tandem with the primary care practitioner, the surgeon (when appropriate), and the dog's human companions to facilitate open and reciprocal communication about the patient's pain status at every stage of treatment. Some patients require a very complex and aggressive pain management protocol to accomplish the goals of physical rehabilitation. As physiotherapy progresses, and the patient improves, the pain management strategy must be adjusted to meet those changing needs. Likewise, if the patient worsens during rehabilitation, reassessment of the patient and revision of its pain protocol are essential. Without some understanding of the complexities of long-standing pain, and of treating pain as a disease in and of itself, the rehabilitation patient may suffer needlessly.

Putting Together a Pain Management Plan for Surgery Patients

Postsurgical Pain and Rehabilitation

Whenever possible, the rehabilitation practitioner should be in contact with the primary care practitioner and surgeon before a surgical patient is referred for physiotherapy. This allows for dialog about the patient's diagnosis, the surgical plan, and the pre-, intra-, and postoperative pain management strategy. It also allows the rehabilitation practitioner to design the postoperative physiotherapy program and implement any appropriate techniques immediately after surgery—either at the surgical practice or in the home during the initial postoperative healing period. It also allows the rehabilitation practitioner the potential to play a role in preventing the patient from evolving into a maladaptive pain state.

Preemptive Analgesia

Pain memories imprinted within the CNS, mediated by NMDA receptors, produce hyperalgesia and contribute to allodynia—pain that is produced by a stimulus that does not normally provoke pain. A preoperative blockade of nociception may prevent postsurgical wound pain or pain hypersensitivity following surgery. This is the concept of preemptive analgesia articulated by the eminent pain physiologist Patrick Wall.[16] In 1988, Professor Patrick Wall introduced the concept of preemptive analgesia to clinicians with his editorial in the journal *Pain*.[17] Successful preemptive analgesia must meet three criteria. It must be (1) intense enough to block all nociception, (2) wide enough to cover the entire surgical area, and (3) prolonged enough to last throughout surgery and even into the postoperative period.

General anesthesia with an inhalant agent (e.g., isoflurane, sevoflurane) does *not* prevent central sensitization. The potential for central sensitization exists even in unconscious patients who are unresponsive to surgical stimuli.[18,19] Noxious stimuli still enter the spinal cord during apparently adequate anesthesia.[20] The purpose of preemptive analgesia is to prevent sensitization of the nervous system

throughout the perioperative period. Pain is to be expected from an initial surgery and the hypersensitivity that subsequently develops. Analgesia administered after sensitization may decrease pain somewhat, but has little long-term benefit in addressing the pain resulting from postsurgical inflammation. Analgesia administered before surgery limits inflammatory pain and decreases subsequent hypersensitivity. The most effective preemptive analgesic regimen occurs when it is initiated before surgery and continued throughout the postoperative period. Equally important is to use a complement of medications that work synergistically; for example, an opioid, alpha-2 agonist, NMDA antagonist, local anesthesia, and an NSAID.[21-23] It bears reminding that good nursing care in the immediate postoperative period can contribute to the comfort of the surgical patient.[24]

Postoperative Analgesia

For the majority of rehabilitation patients that have experienced surgery, NSAIDs form the foundation of pain management in the postoperative period. Antiinflammatory drugs play such a substantial role in perioperative pain management[25] because surgery cannot be performed without subsequent inflammation. Reducing the inflammatory response in the periphery, and thereby decreasing sensitization of the peripheral nociceptors, should attenuate central sensitization.[26] In human medicine, the use of perioperative NSAIDs has reduced the use of patient-controlled analgesic morphine by 40% to 60%.[27,28]

Depending upon the patient's needs, an NSAID may be adequate as a stand-alone pain management agent. However, the rehabilitation practitioner must be especially vigilant about pain issues in the immediate postoperative period. Inadequately managed postoperative (adaptive) pain can transition to maladaptive pain, creating additional problems for the physiotherapy patient. A postoperative rehabilitation patient should be assessed for pain regularly, both at the time of therapy and through conversation and questioning with the pet owner to evaluate activities of daily living. One source of methods for scoring and evaluating pain may be found in the AAHA/AAFP Pain Management Guidelines for Dogs and Cats.[29] It may be that a postsurgical patient may need one or more of the adjunctive pain management strategies outlined in the following chronic pain section.

The Elements of a Comprehensive Pain Management Plan for Chronic Pain Patients

Pharmaceutical Pain Management for the Maladaptive Pain Patient

A majority of nonsurgical patients presented for physical rehabilitation are dogs suffering from chronic, maladaptive pain—often as a result of the degenerative changes that characterize osteoarthritis. Regardless of the underlying etiology, maladaptive pain patients present unique challenges to the rehabilitation practitioner, the primary care provider, and the pet owner who has witnessed the erosion of the dog's activities of daily living. The goal is to break the cycle of maladaptive pain to allow the patient to engage in physical rehabilitation activities designed to restore function, build strength, and ultimately minimize the use of pharmacologic agents to sustain comfort.

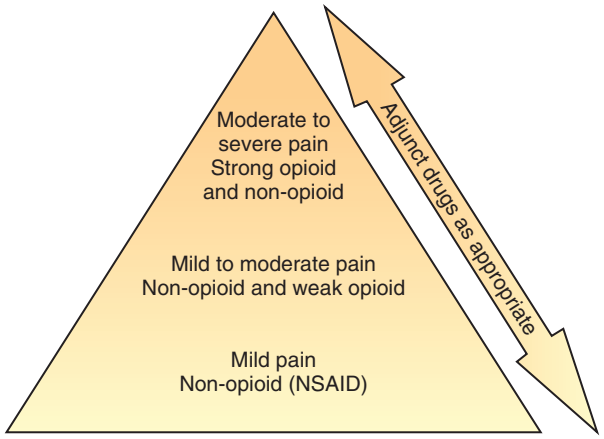


Figure 14-3 The World Health Organization Cancer Pain Ladder. (Adapted from WHO 31.)

When building a comprehensive pharmacologic pain management plan for these patients, it is instrumental to consider the World Health Organization (WHO) Cancer Pain Ladder as a model (Figure 14-3). The principle articulated in this model is to begin pain management using medications that are effective against mild to moderate pain—generally NSAIDs—and building upon that foundation by adding adjunctive medications that attack maladaptive pain via synergistic pathways/modes of action as well as nonpharmacologic strategies. The result is a pyramid in which each addition builds upon the last layer of pain management, providing a foundation for the next layers to be added. The WHO ladder for treating cancer pain in humans[30] has been adopted for a variety of different types of pain in both humans and veterinary patients.

As veterinary pain management becomes more sophisticated, pharmacologic combination therapies from human medicine are being considered and implemented. It is much easier to identify maladaptive pain states in humans than in nonhuman species. Figure 14-4 suggests a logical approach to managing the progressive pain state, wherein pain management uses a multimodal scheme in which drug classes are added rather than substituted. The doses are empiric, and one could debate the order of implementation from bottom to top. The merit of this approach is the addition of agents from different drug classes, attempting to block as many of the pain pathways (transduction, transmission, modulation, and perception) as possible. The

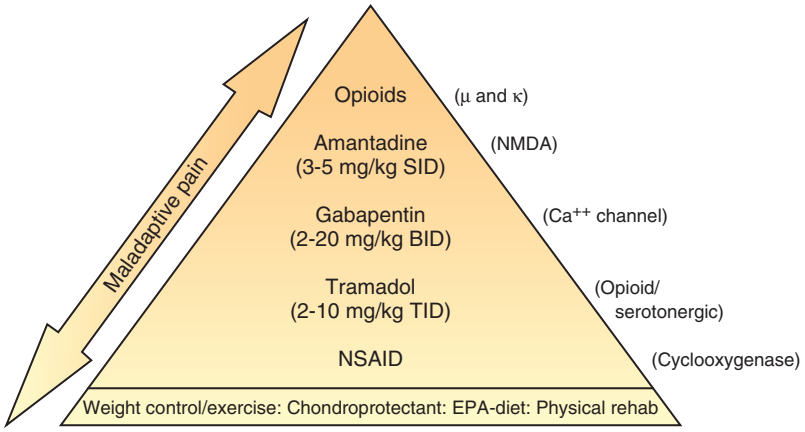


Figure 14-4 Proposed analgesic ladder for progressing pain states using drugs with different (complementary) modes of action. (Doses are empiric, and combination efficacy and safety data are unexplored.)

illustration also acknowledges the important role of nonpharmacologic contributions to pain management. Care must be taken as clinicians await clinical trials that clearly and specifically provide information about (1) each drug's mode of action, (2) the pharmacokinetics in the target species, and (3) the physiology as well as pathophysiology in the veterinary target species. At the moment, only a handful of studies may be cited in support of the use of several of the adjunctive agents that are enjoying increasing use in veterinary patients.[31-34]

Before beginning any multimodal pharmacologic pain management strategy for the rehabilitation patient with maladaptive pain, it is important to ensure due diligence to uncover any existing disease or co-morbidity that could influence decisions about medications. It is also imperative to understand the potential interactions among medications that may be chosen to manage maladaptive pain, as these drugs will presumably be given over long periods—potentially months to years.

Pretreatment screening should include a thorough physical examination, including a review of the patient's medical history and medical record for any previous or concurrently administered medications. A metabolic profile to evaluate renal and hepatic function and a hemogram are fundamental. A more sensitive way to assess for potential renal compromise is to include a test for microalbuminuria[35]—a marker for increased permeability of the basement membrane of the glomerulus. Although not pathognomonic for chronic renal failure, microalbuminuria alerts the practitioner to the need for additional vigilance of renal function. There is no consensus regarding the frequency of monitoring metabolic and organ function in the face of chronic-use medications for pain; however, a 2- to 4-month interval for monitoring is reasonable in most older dogs.

Nonsteroidal Antiinflammatory Drugs

Nonsteroidal antiinflammatory drugs remain the cornerstone for treating pain associated with degenerative joint disease (osteoarthritis; OA) and other sources of chronic pain. Nonsteroidal antiinflammatory drugs have effects at numerous locations along nociceptive pathways, both peripherally and centrally. This class of drugs manifests antiprostaglandin effects peripherally, but they are also considered central-acting because of their inhibition of cyclooxygenase within the dorsal horn of the spinal cord. Cyclooxygenase-2 (COX-2)–mediated PGE_2 production contributes to a late-onset, prolonged, and diffuse phase of central sensitization.[36] Cyclooxygenase-2-specific NSAIDs dampen central sensitization, and therefore play an important role in a multimodal pain management strategy. Cyclooxygenase-1–sparing NSAIDs (Deracoxib,[37] meloxicam, and firocoxib[38]) have their optimal theoretical advantage where there is concern for platelet aggregation, or homeostasis.

The integration of an NSAID in a pain management protocol is particularly appealing when considering the theory by some[39] that the origin of all pain is inflammation and the inflammatory response. However, it must be appreciated that the ceiling effect of NSAIDs can limit their analgesic potential, thereby suggesting they are best, when used alone, for mild and moderate pain. Among a population of canine pain patients, all NSAIDs are effective, but a practitioner may witness variable responses to NSAIDs among individuals.

Tramadol

Tramadol has become a popular analgesic adjunct over the past decade, having gained a worldwide reputation as an effective, safe, and well-tolerated drug for inhibition of moderate pain in humans. It has come to fill the gap on the WHO analgesic ladder between the weak analgesics (NSAIDs) and strong opioids (e.g., morphine). It is not classified as a true opioid, but does have a weak affinity for μ-receptors. In addition, tramadol inhibits both serotonin and norepinephrine reuptake.

Adverse effects include sedation, dysphoria, vomiting, diarrhea, and inappetence. Tramadol tablets are extremely bitter, so care must be taken to hide the tablet to prevent the dog from tasting it and becoming food averse. Because

of its effects on serotonin reuptake, concomitant use of a selective serotonin reuptake inhibitor (SSRIs such as tricyclic antidepressants) or monoamine oxidase inhibitor (MAOI) can lead to serotonin syndrome. The signs of serotonin syndrome include: agitation, confusion, rapid heart rate, dilated pupils, shivering, high fever, seizures, and unconsciousness. Serotonin syndrome is potentially life-threatening.

Doses range from 2 to 10 mg/kg po tid. This is empiric dosing, as tramadol is not approved for dogs or cats. Only oral preparations are available in the United States. The pharmacokinetics of tramadol have been studied in dogs, and it appears that the half-life is quite short, approximately 1.7 hours, necessitating frequent dosing.[40] Evaluation of an oral sustained-release formulation of tramadol did not yield positive results in the dog.[34] Within the veterinary profession, there are not yet dose titration studies in our target species. To date there are no published dose titration studies or studies of safety or efficacy in dogs or cats.

Anticonvulsants

The anticonvulsant gabapentin is structurally analogous to γ-aminobutyric acid (GABA) and was initially used as an antiepileptic, particularly in children. Gabapentin is widely used in human medicine for its activity in certain maladaptive pain syndromes including postherpetic neuralgia, painful diabetic peripheral neuropathy, and fibromyalgia. Gabapentin is theorized to work by binding to the α_2-δ_1 subunit of presynaptic voltage-gated calcium channels that are upregulated during central sensitization. Its analgesic effect may well be proportional to the magnitude of sensitization within the dorsal horn.[41,42]

Gabapentin may be most useful as an adjunct to other analgesics, particularly in those patients suspected of experiencing maladaptive pain. One study in rats suggested that gabapentin may be analgesic in the pain associated with osteoarthritis.[43] Gabapentin use in human pain medicine has recently extended into the management of more acute conditions, including an adjunctive role in the perioperative period for the prevention of persistent postoperative pain.[44] The liquid form of gabapentin, labeled for human use, should not be used in pets, as it contains xylitol, which is hepatotoxic in dogs. Gabapentin can be compounded into liquid form for veterinary use, however.

Simultaneous administration of oral antacids may decrease the bioavailability of gabapentin. They should be given at least 2 hours apart. Also, coadministration of morphine may increase gabapentin's effects and increase the likelihood of side effects such as sedation. The use of gabapentin in three dogs suspected of experiencing neuropathic pain, a maladaptive pain state, has been documented.[33] Additional studies need to be conducted in veterinary species.

In dogs clinicians are using gabapentin as an adjunct to other therapies for those patients suspected of experiencing maladaptive pain.[33] Reported starting doses are 5 to 10 mg/kg po bid-tid. The most common adverse effect of gabapentin in humans is dose-dependent sedation, and in clinical practice this appears to be the case for dogs. For most patients a downward dose adjustment resolves the issue. In dogs with maladaptive pain, positive results to the addition of gabapentin may manifest in as little as 48 to 72 hours. As stated the analgesic effect of gabapentin may be proportional to the level of maladaptive pain the patient is experiencing. Thus the dose of gabapentin may be increased to meet the need of the patient, with sedation as the primary dose-limiting effect. Gabapentin should not be discontinued abruptly because withdrawal may precipitate rebound pain. Once the dog's pain is well managed the dosage can be slowly reduced over a period of weeks to months.

N-Methyl-D-Aspartate Antagonists

The NMDA receptor plays a major role in central sensitization[45]; therefore inhibition of this receptor validates the implementation of an NMDA-antagonist in an analgesic protocol. Amantadine is an NMDA receptor antagonist and acts through stabilization of NMDA channels in the closed state.[46] Amantadine has been used to treat neuropathic pain in humans. A clinical trial was reported in which dogs with refractory osteoarthritis received amantadine as an adjunctive therapy in addition to an NSAID.[32] The conclusion was that amantadine performed well in improving physical activity in the treated dogs. Toxicity studies have been performed,[31] and a dose of 8 mg/kg once daily was not implicated in adverse signs.

As an adjunct to an NSAID in the dog with chronic OA pain, a dose of 3 to 5 mg/kg per day po may be given. In the author's (Downing) experience, this drug and dose is extremely well tolerated. Amantadine is available commercially as 100 mg capsules and as a 10 mg/mL oral syrup.

Tricyclic Antidepressants

Tricyclic antidepressants (TCAs) are characterized by their multiple modes of action, with a particular ability to inhibit reuptake of monoamines (serotonin and norepinephrine) from presynaptic terminals. Additionally TCAs block several receptors (cholinergic, adrenergic, histaminergic) and ion channels, including Na^+ channels. The mechanism of TCA action might best be identified as five drugs in one: serotonin reuptake inhibitors, norepinephrine reuptake inhibitors, anticholinergic-antimuscarinic drugs, α-1 adrenergic antagonists, and antihistamines. These drugs have been used in humans to treat maladaptive pain at much lower doses than those used to treat depression.

Longer-term administration alters the receptor binding of NMDA. Tricyclic NMDA activity is likely analgesic via its inhibition of neuronal hyperexcitability.[47] Alpha-adrenergic-receptor blockade in peripheral neuropathy may be relieving pain generated or maintained by

noradrenergic stimulation of highly sensitive receptors such as those identified on sprouts from diseased peripheral nerves.[48] Tricyclic antidepressants may stabilize both diseased peripheral nerves and hyperexcitable neurons of the central nervous system (CNS) by blocking sodium channels.[49]

Although TCAs may be effective as an adjunctive therapy for maladaptive pain in animals, they have not yet been evaluated. The TCAs should not be used concomitantly with drugs that affect serotonin levels such as tramadol.

Orally Administered Opioids

Although controversial, studies suggest that maladaptive pain secondary to peripheral nerve damage, more often than not, shows reduced sensitivity to opioids.[50] This is attributable to a reduction in spinal opioid receptors, non-opioid receptor-expressing Aβ fiber-mediated allodynia, increased cholecystokinin antagonism of opioid actions, and NMDA-mediated dorsal horn neuronal hyperexcitability, likely requiring a greater opioid inhibitory countereffect. Another problem with oral opioids in dogs is the very low (methadone <30%; morphine 5%) bioavailability[51,52] because opioids are metabolized in the liver. Codeine has the highest bioavailability, approaching 60%,[53] and is often administered in combination with acetaminophen (***acetaminophen is lethal in cats***). Although morphine and hydromorphone are available as suppositories, there appears to be little difference in efficacy or bioavailability (≈20%) from oral administration.[54] However, this delivery form is an option if oral administration is unavailable. Although injectable opioids remain the gold standard for managing severe acute pain, oral opioids seem lacking in their application (orally) in most animals with chronic maladaptive pain.

Nonpharmacologic Approaches to the Chronic Pain Patient

Acupuncture

Acupuncture falls under the category of complementary and alternative medicine (CAM) as part of traditional Chinese medicine (TCM) and is used in humans in at least 78 countries worldwide.[55] There are sound physiologic mechanisms for the analgesic effects of this treatment.[56] A common feature shared by all styles of acupuncture is the use of needles to initiate changes in the soft tissue. Needles and needle-induced changes are believed to activate the built-in survival mechanisms that normalize homeostasis and promote self-healing. In this context acupuncture can be defined as a physiologic therapy coordinated by the brain that responds to the stimulation of manual or electrical needling of peripheral sensory nerves, in which acupuncture does not treat any particular pathologic system, but normalizes physiologic homeostasis and promotes self-healing.[57] Acupuncture can be effective for both peripheral soft tissue pain and internal disorders, but in the case of peripheral soft tissue pain the result appears more predictable because of the local needle reaction.

Hypothesis of Acupuncture Analgesia Mechanisms

The leading hypotheses of acupuncture analgesia mechanisms include local effects of needling, neuronal gating, the release of endogenous opiates, and the placebo effect. It is further proposed that the CNS is essential for the processing of these effects via its modulation of the autonomic nervous system, the neuroimmune system, and hormonal regulation. Clinical observation suggests that acupuncture needling may achieve at least four therapeutic goals:

1. Release of physical and emotional stress
2. Activation and control of immune and antiinflammatory mechanisms
3. Acceleration of tissue healing
4. Pain relief secondary to endorphin and serotonin release.

Acupuncture therapy is considered to activate built-in survival mechanisms (e.g., self-healing potential). Therefore it is effective for those symptoms that can be completely or partially healed by the body. Additionally each individual has a different self-healing capacity influenced by genetic makeup, medical history, lifestyle, and age, all of which may be dynamically changing.[58] Acupuncture can be used to release trigger or tender points in addition to its application for musculoskeletal pain.

Acupuncture can be initiated immediately for the benefit of the rehabilitation patient experiencing maladaptive pain. In this context, acupuncture may be used to augment the effects of the pharmacologic intervention that is chosen. It is the author's experience (Downing) that acupuncture often results in reduced doses of medications needed to break the cycle of a rehabilitation patient's maladaptive pain.

Other Therapies

Other techniques exist for augmenting the pharmacologic intervention on behalf of a painful dog presented for rehabilitation. These include the application of low-level laser therapy (LLLT), also referred to as cold laser, therapeutic laser, extracorporeal shock wave therapy, therapeutic ultrasound, cryotherapy, heat therapy, and electrical stimulation. These modalities are described in detail elsewhere in this book and by others.[59-61]

Environmental Modification

It is difficult to overstate the positive difference that simple environmental changes can make for the maladaptive pain patient. Yet, this is one area that is easy to overlook because it does not involve as much hard science as choosing a pharmacologic regimen or calculating an appropriate nutritional profile. With a few simple questions it is fairly

straightforward to determine if the rehabilitation patient will benefit from environmental modification. It does mean taking the initiative to gather information about the home, such as floor surfaces, stairs, placement of food and water dishes, type of bedding, location of bedding, and so on.

One of the simplest environmental modifications that can have a positive effect on the chronically painful rehabilitation patient is to protect the dog from slippery floor surfaces. Aging patients, particularly those with OA, experience a loss of proprioceptive function in joint receptors versus younger patients.[62,63] Nonskid area rugs, flooring used in children's play areas, and rubber-backed mats are some examples of ways a dog owner can make the home more comfortable for the pain patient.

Raising food and water dishes to between elbow and shoulder height makes for more comfortable meal times. Be sure the dog is able to stand on a nonskid surface while eating and drinking. Have the client explore the home for potential problem spots. Steps in and out of the house, patio stones, and garage floors can create unintentional challenges for the painful dog. Ramps for getting into and out of vehicles should be recommended. Likewise, have the client consider ramps or steps if the dog is used to getting onto and off furniture. It may be best to use child restraint gates at the top and bottom of staircases to prevent unsupervised access. Memory foam or eggshell foam may be a more comfortable sleeping surface for the painful dog. Assistive devices like slings and wheelchairs can sustain mobility during the initiation of appropriate multimodal pain management strategies. Finally it is best for the dog to receive moderate exercise every day than to do excessive exercise on the weekend, requiring the rest of the week to recover.

Putting It All Together for the Painful Patient

In human pain medicine, pain practitioners understand that pain is what the patient says it is, and that their obligation is to believe the patient. In veterinary medicine, the practitioner, whether the primary care provider, the surgeon on an orthopedic case, or the rehabilitation practitioner, has an obligation to advocate on behalf of a patient that cannot advocate for itself, and that means actively looking for pain in the patients we see. Assessing nonhuman patients presents its own challenges; however, these challenges must not prevent caretakers from taking an aggressive approach to pain, particularly to the long-standing suffering of the maladaptive pain patient. For assistance in choosing a pain scoring system, the reader is referred to the AAHA/AAFP Pain Management Guidelines for Dogs and Cats,[29] and the International Veterinary Academy of Pain Management (www.ivapm.org).

There are many things to consider when confronted with a painful rehabilitation patient. The most important first step is recognizing that rehabilitation will be counterproductive if the patient's pain is not identified and managed appropriately. Pain is a subjective, individual phenomenon, and there is tremendous variability among individual patients. For these reasons, when in doubt, it is best to treat. Often after pain management is initiated the client will report a normalization of the dog's behaviors and activities of daily living. It is common to hear a client say, "I didn't know he was painful."

Considering that each patient is unique, an individual pain management protocol should be designed to accompany each patient's rehabilitation plan. The pharmacologic component of that protocol is based on assessment of the individual animal and the particular rehabilitation treatment/session. For immediate postoperative rehabilitation the importance of a comprehensive multimodal, perioperative analgesic protocol cannot be overstated because of its impact on the postoperative recovery period. For the dog with chronic maladaptive pain, monotherapy with a single-agent like an NSAID is likely to fail.

How does a rehabilitation practitioner begin putting together a multimodal pain management strategy? Earlier in this chapter the analogy was drawn between building a pain management plan and building a pyramid. This provides us with a visual expression of our task on behalf of the painful rehabilitation patient. Because each patient is an individual, each pain management protocol must be created with that patient's individual needs in mind. However, there are some general principles to consider based on the author's (Downing) experience in a pain management referral practice:

1. Start with a complete and thorough physical examination, including a neurologic evaluation, orthopedic evaluation, and careful palpation for pain.
2. Spend time talking with the client to understand the dog's activities of daily living, the daily household routine, and the home environment (stairs, floor surfaces, placement of food and water dishes, etc.).
3. Conduct a complete metabolic profile to assist in the choice of medications as well as to identify any co-morbidities. Treat any co-morbidities (e.g., hypothyroidism) concurrently.
4. Focus on BCS (body condition score). If the dog is overweight this *will* interfere with pain management and subsequent rehabilitation.[64,65] Create a nutritional plan to assist the dog in achieving a normal BCS.
5. For those dogs with OA provide an EPA-supplemented nutritional product.[66-68]
6. Create a pharmacologic intervention plan. If NSAIDs are appropriate for the patient, based on the metabolic profile, prescribe one. For a maladaptive pain patient, gabapentin is a reasonable addition in the first round of therapy.
7. If possible consider acupuncture, laser therapy, cryotherapy, or another modality to provide relief.

8. Create expectations for the client so he or she can appropriately gauge the success or failure of each step in the treatment progression.
9. Create a written plan for the client, the medical record, and the primary care provider to minimize misunderstanding as well as the risk for medical error. Painful rehabilitation patients require complex care and monitoring, so make it as easy for the client as possible.
10. Schedule each pain reassessment for chronic pain patients *before* the client leaves the facility. The first pain reassessment should be performed 7 to 10 days after initiating treatment. Subsequent reassessments should be scheduled at 10- to 14-day intervals until the dog's pain is well managed. Although we should not increase the dose of an NSAID above the recommended dosing, it is common to increase the dose of gabapentin to meet the patient's need. For patients with long-standing maladaptive pain, it is not uncommon to need gabapentin doses approaching 30 to 40 mg/kg bid-tid. Patients being treated for acute postoperative pain should be assessed several times per day for adequate comfort.
11. If the patient remains painful at the third visit following an increase in gabapentin dosing, consider adding amantadine, and schedule the next recheck in 7 to 14 days, depending on the patient's level of discomfort. The greater the discomfort, the shorter should be the recheck interval. If needed, tramadol may be used for episodes of breakthrough pain. Likewise, a TCA may be used for refractory pain. Do not use tramadol and a TCA together.
12. After the dog's pain is reasonably controlled rehabilitation can begin in earnest.
13. Schedule appropriate metabolic monitoring based on the patient and the medications chosen.
14. After pain is well managed and the dog is actively engaged in rehabilitation activities, coordinate the titration of medication doses to achieve minimum effective doses, recognizing that some individuals may need multiple medications long term.

The goal as practitioners is to achieve and maintain a delicate balance with painful patients to keep them as comfortable, active, mobile, and functional as possible to sustain the precious family–pet relationship. After a pain management strategy is under way remember the "three Rs"—Recheck, Reassess, and Revise. Painful rehabilitation patients provide a moving target for our care. Remain in dialog with the clients to notice patterns of behavior that might precipitate modification of the treatment plan. Daily journals of the dog's activities and interactions with the family can provide important insights.

Our obligation to our painful rehabilitation patients is to advocate on behalf of creatures who cannot advocate for themselves. We need to practice compassionate care medicine. Animals must have their pain relieved for their physiotherapy to be maximally effective.

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15 The Role of Chondroprotectants, Nutraceuticals, and Nutrition in Rehabilitation

D.M. Raditic and J.W. Bartges

Veterinary rehabilitation is a rapidly developing field and is an integral therapy for many medically and surgically managed musculoskeletal, neurologic, and other conditions in dogs and cats. Nutrition plays an integral role in prevention and management of many conditions commonly seen in this field. This chapter discusses how nutrition can be used to help facilitate and promote healing and influence the rehabilitation process. Nutritional concepts such as overall caloric, protein, carbohydrate, vitamin, and mineral requirements are discussed, and a variety of supplements and their evidence are reviewed.

Nutritional Plan

To integrate appropriate nutritional recommendations for patient management, a two-step process is undertaken: a nutritional assessment phase (based on the Circle of Nutrition by the American College of Veterinary Nutrition; http://www.acvn.org) and an analysis, interpretation, and action phase.[1] In the assessment phase three sets of factors are assessed: animal-specific factors, diet-specific factors, and feeding management and environmental factors.[2] Animal-specific factors include age, physiologic status, disease processes, and activity of the patient. Diet-specific factors include safety and appropriateness of diet fed and nutrients of interest. Feeding factors include frequency, timing, location, and method of feeding, and environmental factors include living space and quality of the patient's surroundings.[1] An important part of the assessment phase is obtaining body weight, body condition score, and muscle condition score.[1] Body condition score evaluates body fat (Figure 15-1).[3-5] The goal for most patients is a body condition of 2.5 to 3.0 out of 5 or a 4 to 5 out of 9, which is associated with decreased health problems, including musculoskeletal conditions.[6-10] Muscle condition score evaluates muscle mass, which can be independent from body fat content assessed by body condition score (Figure 15-2).[11] Evaluation of muscle mass includes visual examination and palpation over temporal bones, scapulae, lumbar vertebrae, and pelvic bones. Muscle loss adversely affects strength, immune function, and wound healing. After collecting information from the assessment phase information is analyzed and interpreted and an action plan is implemented. After implementation of the action plan the patient undergoes repeated assessment and adjustment of the plan.

Role of Nutrition in the Prevention of Musculoskeletal Disease

Nutrition interacts with musculoskeletal disease. The prevalence of musculoskeletal disorders for all dogs at multicenter referral practices has been reported to be approximately one in four.[12] In dogs less than 1 year of age the prevalence is 22%, with 20% of these possibly having a nutrition-related cause.[13] We focus on two specific common conditions.

Developmental Orthopedic Disease

The role of nutrition in the development of musculoskeletal disease in growing dogs has been recognized for decades. Developmental orthopedic disease refers to a group of skeletal abnormalities (e.g., osteochondritis dissecans, hip dysplasia, wobblers' syndrome, ligamentous laxity, and hypertrophic osteodystrophy) that affect primarily rapidly growing, large- and giant-breed dogs. Nutrient excess (calcium and energy) and rapid growth (overfeeding and excess energy) are known risk factors for developmental orthopedic disease in dogs that have genetic risk.[14-18] Restricting food intake during growth slows growth rate without significantly reducing adult body size and is associated with decreased incidence of developmental orthopedic disease.[8,19] Dietary calcium greater than 3% on a dry matter basis is associated with increased risk despite an appropriate calcium-to-phosphorus ratio.[15] Even if the diet contains less calcium than this, excess calcium intake can occur if owners provide supplemental calcium. For example, two level teaspoons of calcium carbonate added to a growth diet fed to a 15-week-old Rottweiler puppy doubles the calcium intake. For large- and giant-breed dogs during growth, recommended dietary composition includes the following[20]:

- Energy: 3.2 to 4.1 kcal/kg of diet (lower end of range if at risk for developmental orthopedic disease or if clients use free-choice feeding)

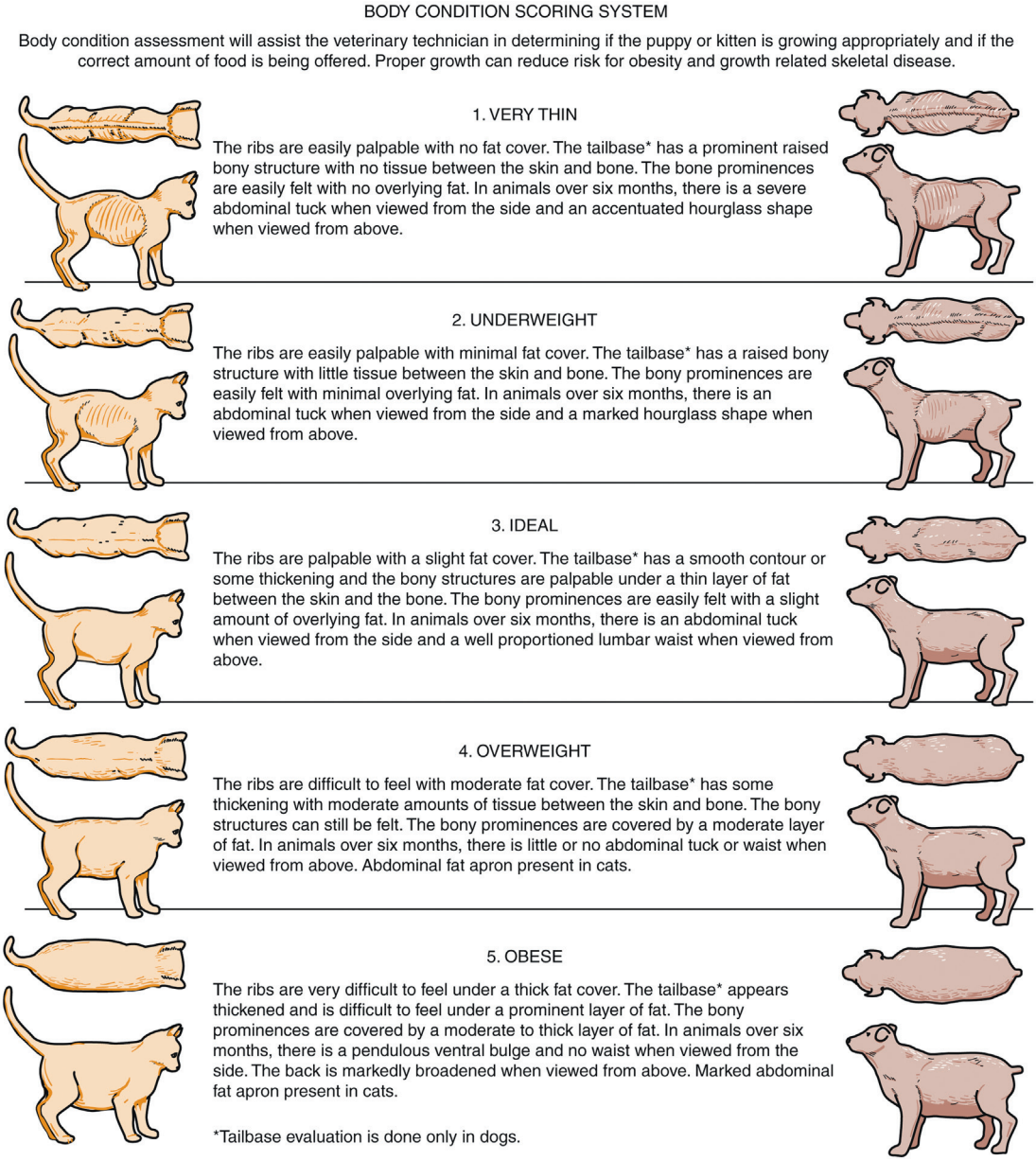


Figure 15-1 Body condition scoring (BCS) systems for the dog and the cat. (From Bassert JM, McCurnin DM: *McCurnin's clinical textbook for veterinary technicians,* ed 7. St. Louis, Saunders, 2010.)

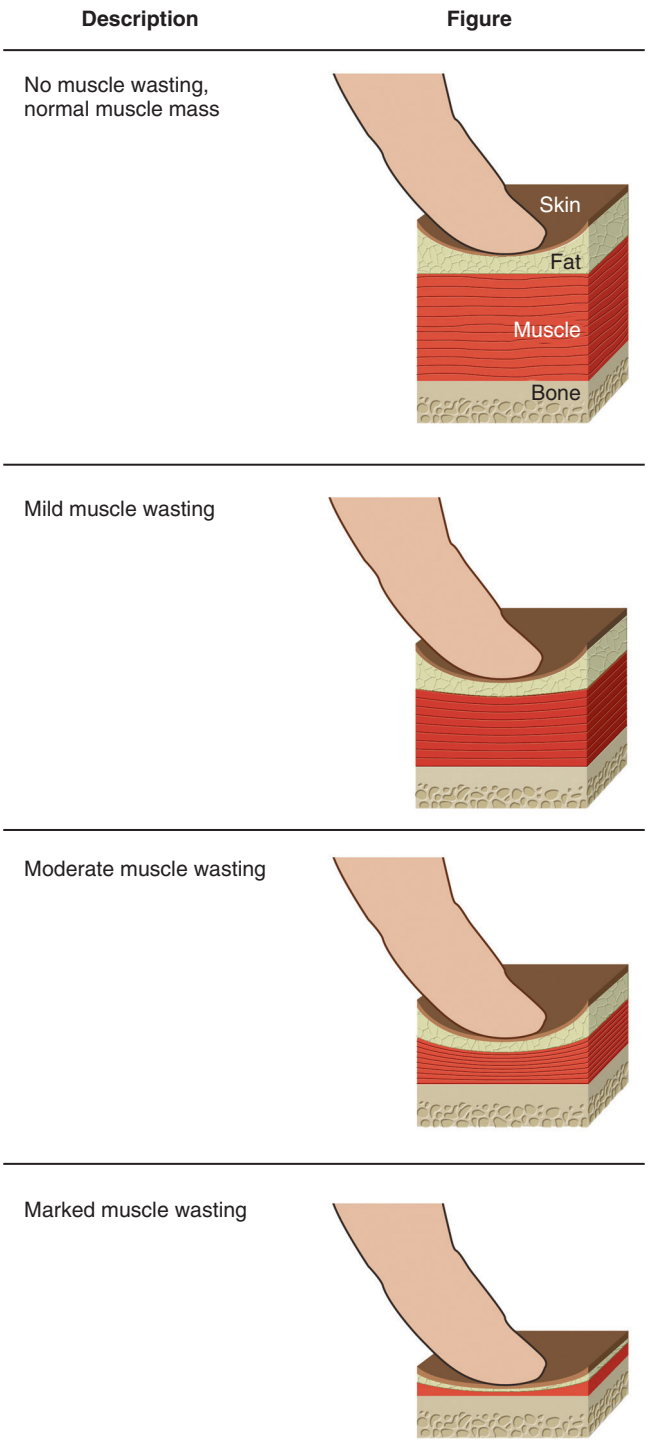


Figure 15-2 A muscle condition scoring (MCS) system. Evaluation of muscle mass includes visual examination and palpation over the temporal bones, scapulae, ribs, lumbar vertebrae, and pelvic bones. (Adapted from Baldwin K, Bartges J, Buffington T et al.: AAHA Nutritional assessment guidelines for dogs and cats, J Am AnimHospAssoc 46:285, 2010. IN Little S: The Cat: Clinical Medicine and Management, ed. 1, St. Louis, 2012, Saunders.)

- Crude fat: 8.5% to 11% on a dry matter basis
- Docosahexaenoic acid: ≥0.02% on a dry matter basis
- Protein: ≥27% on a dry matter basis
- Calcium: 0.8% to 1.2% (≤3% on a dry matter basis) with a calcium-to-phosphorus ratio of 1.1 to 2.0 : 1.0

The risk of developmental orthopedic disease is reduced if clients use limited meal-feeding rather than free-choice feeding and if growth rate is limited during the postweaning period.[21]

Obesity

Obesity can be defined as accumulation of body fat in excess of what is necessary to maintain optimum condition and health. This may be obvious in a grossly obese individual particularly when obesity-related disease is present; however, it is less obvious in a patient who is overweight and otherwise clinically healthy, but who is at risk for obesity-related disease such as osteoarthritis. Quantitatively, obesity is generally defined as exceeding ideal body weight by 15% to 20% or more. One technique that is useful in the management of clinical patients is a body condition score and muscle condition score as previously discussed. More sophisticated methods of body composition assessment include body composition determination by dual-energy x-ray absorptiometry (DEXA), quantitative computed tomography (quantitative CT) scans, or bioelectrical impendence.

Obesity may contribute to the development and progression of osteoarthritis because of excess forces placed on joints and articular cartilage, which may lead to inactivity and further development of obesity. Thus a vicious cycle ensues. Additionally adipose tissue is recognized as being metabolically active and proinflammatory; therefore obesity may contribute to inflammation.[22-26]

Several studies have demonstrated a relationship between obesity and osteoarthritis[9]; however, a cause and effect has not been determined.[27,28] A long-term study was performed of 48 Labrador retrievers from seven litters divided into two dietary groups. One group was fed an adult maintenance dog food at 0.27 kJ of metabolizable energy per kilogram of body weight per day, and the second group was fed the same diet at 75% of the amount (0.20 kJ of metabolizable energy per kilogram of body weight per day).[8,29] Restricted fed dogs lived on average 1.8 years longer, weighed less, had better body condition scores, and had longer delay to treatment of chronic disease including osteoarthritis.[21] In addition the restricted fed group had a lower incidence of osteoarthritis in most major joints, and if it occurred, it was less severe. Maintaining optimal or slightly lean body condition may be associated with lower risk of developing osteoarthritis, development of less severe osteoarthritis if osteoarthritis occurs, and delay of onset of clinical signs of osteoarthritis in dogs.

Role of Nutrition in the Treatment of Musculoskeletal Disease

Nutrition and Surgery

The role of nutrition in surgery in the context of physical rehabilitation and rehabilitation falls under two major categories: healthy surgical patients undergoing procedures for developmental (e.g., osteochondritis dissecans) or acquired (e.g., repair of cranial cruciate ligament damage) conditions, and posttrauma surgical patients undergoing procedures (e.g., fracture repair from motor vehicle injury).

Nontraumatized Surgical Patients

Patients undergoing surgery that are healthy and in optimal body and muscle condition (see Figures 15-1 and 15-2) require no further intervention nutritionally other than to continue feeding the current diet at the same amount and frequency. Patients are not fed 8 to 12 hours before anesthesia and surgery, but can resume normal feeding patterns once recovered from the procedure. It may be necessary to add flavoring agents or feed a different diet to stimulate appetite in the postoperative period. Patients should be assessed daily following surgery to insure they are eating adequately and that no adverse events occur either from the procedure or from postoperative medications such as analgesics and sedatives. Energy requirements during hospitalization and in the immediate postoperative period when activity is restricted is estimated in most patients to be the resting energy requirement (RER).[30,31]

$$\text{RER (kcal/day)} = 30\text{BW}_{\text{kg}} + 70$$

or

$$\text{RER (kcal/day)} = 70\text{BW}_{\text{kg}}^{0.75}$$

Although both equations are used to estimate RER, the authors prefer the second equation because body weight and RER are not linearly related as estimated with the first equation. As the patient becomes more active or as a rehabilitation program is initiated, energy requirements increase (see Nutrition and Rehabilitation).

In many instances surgery in these patients is elective; therefore, depending on the urgency of the surgical procedure, consideration should be given to weight reduction before surgery in patients that are obese (see Obesity). Reducing body weight to the optimal weight decreases anesthetic risk, improves immune function, and allows patients to more easily undergo postsurgical physical rehabilitation.[32]

Traumatized Surgical Patients

Patients with surgical musculoskeletal disease associated with trauma may or may not have increased nutritional requirements depending on the degree of trauma. If the trauma is less severe, then energy requirements are similar to nontraumatized surgical patients, and energy requirements approximate resting energy requirements.[30,33] Energy requirements are determined by estimating RER as stated.[30,31]

Severely injured patients or those with closed head injuries typically have energy requirements exceeding resting energy requirements because of the extent of the injuries or activation of the sympathetic nervous system, in part.[34] In these patients energy requirements may exceed two times the estimated resting energy requirements.[30,31]

Likewise protein requirements may exceed that required for maintenance to facilitate recovery, immune function, and wound healing.[35,36] Recommended protein intake for adult dogs at maintenance is 2.62 to 3.28 g/$\text{BW}_{\text{kg}}^{0.75}$ and for adult cats is 3.97 to 5.90 g/$\text{BW}_{\text{kg}}^{0.75}$.[37] With trauma or strenuous exercise, protein requirements may exceed these by 50% to 100%. Some patients may require facilitated nutritional support enterally or parenterally.[30,38-44] Nutritional support in these patients should be individualized and intensive monitoring is required to maximize recovery and response to supportive care. Convalescent diets are available that are formulated to contain higher levels of protein and fat are more calorically dense, highly palatable, and have a homogeneous texture suitable for feeding tubes.

Nutrition and Rehabilitation

Research into nutritional needs of patients undergoing various rehabilitation programs is nonexistent. Individuals developing rehabilitation programs should have knowledge of nutrition. Rehabilitation can include simple strengthening to extensive long-term physical rehabilitation; therefore, the nutritional plan for a patient must adjunctively meet the goals set during therapy and after goals are met. Nutritional plans should be included as part of the rehabilitation therapy. As described previously, a nutritional plan involves two phases: an assessment phase and an analysis, interpretation, and action phase. Reassessment is required until nutritional and rehabilitative goals are met.

Essential Nutritional Factors

Water

Water is the most essential nutrient. It is the transport medium for nutrients, heat, and waste products. Total body water is divided into four compartments with 64% being intracellular, 22% in interstitial spaces, 7% in plasma, and the remaining 7% transcellular.[45] Total body water is determined by total water intake and metabolic water production minus evaporation, urine, and fecal losses. An increase in muscle activity results in an increased demand for nutrients and removal of waste products. Only 20% to 30% of energy consumed by muscle tissue produces actual work, with the remaining 70% to 80% resulting in heat.[45]

Increased blood flow during exercise dissipates this heat. Increases in body heat during exercise must be removed because increases not only limit work, but also damage tissues.

During rehabilitation, excitement, fear, pain, and other stress factors affect the simplest of nutritional needs, water and electrolytes. These stress factors can increase body temperature and respiration alone or during exercise. It is important to monitor hydration and electrolyte balance during the program. A study of sprinting and endurance racing dogs showed water intake can directly affect biochemical and red blood cell profiles.[46] The same study showed increased blood cortisol and decreased blood glucose in sprint-racing sled dogs. These changes were attributed to metabolic stress and the physiologic response to high-intensity exercise.[46] High-intensity exercise is not commonly used in a rehabilitation setting, but the summation of patient stress, new exercise level, and perhaps changes in water and food intake should be noted.

Patients boarded for continuous rehabilitation may have poor diet and water intake in an unfamiliar environment.[45] Water and food intake should be recorded for hospitalized patients. Owners of outpatient dogs should be questioned about food intake, water intake, and other nutritional changes they may have noted during the program. Water intake for healthy dogs approximates 50 to 60 mL/kg per day, but may be more with rehabilitation.

Energy

Energy is a primary nutrient for maintenance, stage of life, and the therapy program. Diet supplies energy from carbohydrate, fat, and protein. Fats and carbohydrates are preferred substrates for exercise, whereas protein provides structural and functional tissue. Energy requirements correlate to work intensity, duration, and frequency, with intensity being the primary determinant. Studies show that sprinters depend on carbohydrates, whereas endurance activities use fat stores. Sprinters such as greyhounds that are running with maximum intensity have low energy requirements because of the short race duration and low frequency of training.[47] These dogs mainly use carbohydrates to meet energy requirements. Studies of endurance sled dogs with long durations of work are at the other extreme, requiring high energy intake. High-fat diets are fed to meet these high-energy requirements.[45,47,48]

Exercise intensity has not been quantified in rehabilitation, unlike performance and working dogs. Therapy is likely an intermediate level of exercise, which has not been well defined. Intensity, duration, and frequency of exercise are highly variable; therefore it is difficult to predict the energy needs of these patients. Consideration of historical exercise level combined with current patient and diet assessment may provide some insight into energy needs. A house pet with a body condition score (BCS) of 4 to 6/9 (see Figure 15-1), which usually has low energy requirements, that begins a rigorous short duration, increasing intensity, daily program, may need energy intake adjustment during rehabilitation.

The dog's daily energy requirement (DER) is based on the resting energy requirement (RER) multiplied by a factor for life stage or disease. Resting energy requirements are estimated as described in the preceding equations and defined as the energy requirement (kcal/day; 1 kcal = 4.184 kJ) for a healthy but fed animal at rest in a thermoneutral environment.[31] Maintenance, exercise, and life stage (growth, gestation, and lactation) are combined to develop factors of RER to estimate a patient's DER. Life stage factors commonly used in clinical nutrition are as follows[49]:

- Intact adult: 1.6
- Neutered adult: 1.4
- Obese prone: 1.2
- Weight loss: 1.0
- Gestation: 1.8 to 3.0
- Lactation: 4.0 to 8.0
- Growth: 1.6 to 2.5
- Adult, light work: 2.0
- Adult, moderate work: 3.0
- Adult, heavy work: 4.0 to 8.0
- Critical care: 1.0

These life stage factors are guidelines. Variables such as breed, disease, environment, and age can greatly affect energy requirements.

In rehabilitation, energy required to participate in a therapy program affects DER. If the therapist defines the treatment plan as work, then intensity, duration, and frequency of exercises are factored into the DER. Studies show sprinting greyhounds can have a DER of 1.6 to 2 × RER, whereas endurance sled dogs may require greater than 5 × RER.[47] This comparison is useful because there are nutritional studies of racing greyhounds that may apply to a rehabilitation program. Assigning an exercise factor to common physical rehabilitation programs would be useful in calculating the dog's DER. Until such data are provided it is important to recall that most house dogs have less than light exercise and may be obese or obese prone; therefore it is prudent to use low end exercise factors initially.

Reassessment of the patient during rehabilitation will determine if changes in energy intake are needed. If a patient loses weight during therapy, energy intake should increase. If weight gain is noted, energy intake should decrease. When starting a rehabilitation program with a patient with a BCS of 6 or 7, it may be best to make no energy changes despite the need for weight loss. The exercise program itself may induce weight loss and an ideal body condition score. Once the program is complete, client education about the patient's energy needs are discussed to maintain optimal body condition.

Fat

Fat provides the most calories in dog foods at 8.5 kcal/g of dry matter diet. Carbohydrates and protein supply 3.5 kcal/g of dry matter in a diet. To increase the energy density of a commercial diet, the fat content of the diet is increased. Fat also increases palatability of diets. Studies of endurance sled dogs show a positive effect for higher fat diets regarding performance. Sled dogs have been known to consume 80% of their caloric needs from fat.[45] In a study of well-conditioned beagles running on a treadmill, time to exhaustion correlated with fat intake, diet digestibility, and energy concentration of the diet.[50] Increasing fat intake improves performance of dogs with extreme energy requirements. In rehabilitation therapy, the level of exercise is unlikely to equate to the extreme energy requirements of sled dogs. Fat intake still should be considered when a patient is in therapy, however. If we use the example of a sprinting greyhound, dogs in therapy may have similar fat requirements with fat providing 20% to 30% of the total energy requirement. This equates to a diet with a fat content of 8% to 10% on a dry matter basis.[45] Stress factors of a new environment, exercise, fatigue, surgery, and illness may decrease diet intake. If there is appetite suppression, a higher-fat diet, which has a higher energy concentration and increased palatability, can be used to increase voluntary intake. Some dogs losing weight during a program are on diets too low in fat and energy concentration to meet the energy requirements of the rehabilitation program. The diet can be changed to a higher-fat diet with increased energy concentration to meet energy requirements. The type of fat in the diet should be evaluated. Diets must contain essential fatty acids in the form of unsaturated fatty acids. Linoleic acid, a 20-carbon n-6 fatty acid, is required in a dog's diet. N-3 fatty acid utilization is an area of extensive research in both humans and dogs and is discussed in a later section.

Carbohydrates

Carbohydrates are required only during gestation because dogs readily maintain normal blood glucose and tissue glycogen on a no-carbohydrate diet.[45] However, dogs can readily use carbohydrates for energy and most pet foods contain 40% to 60% carbohydrates on a dry matter basis. Racing greyhounds depend on carbohydrates that are metabolized into muscle glycogen. Muscle glycogen is a readily available source of energy for these sprinters with utilization of 70% of muscle stores in a single race. In humans, rats, and horses, increasing muscle glycogen improves exercise performance.[51,52] It is unknown whether increased carbohydrate intake in dogs enhances exercise performance or increases muscle glycogen stores. Studies have mixed results evaluating carbohydrate loading in racing greyhounds.[45,53] Extrapolation from human data suggests that 50% to 70% of total energy intake should be fed to maintain muscle glycogen stores for sprinting.[45] This is because anaerobic metabolism of glucose and glycogen is the dominant energy pathway that produces energy for sprinting. Because of the intensity of work and immediate energy needs for the racing greyhound, this extrapolation is logical, and these dogs are generally fed carbohydrates at a level that meets 50% or greater of the energy requirements. In physical rehabilitation, exercises are unlikely to have the intensity of work that is necessary to have an absolute carbohydrate requirement to employ anaerobic metabolism pathways. Carbohydrate intake using commercial dog foods appears reasonable.

Protein

Protein is required for structure and function, and to a lesser extent, energy. Current recommendations with exercise are to increase protein 5% to 15% regardless of the level of exercise, with dietary protein providing approximately 25% of the energy needs. Dietary protein is the most important nutrient with regard to muscle protein, and exercise increases protein requirements. Many of the goals of canine physical rehabilitation require increased muscle tissue. Studies of protein intake during exercise are needed to identify ideal protein intake for dogs during physical rehabilitation.

In a postabsorptive state, protein that has been broken down to amino acids provides the substrate for protein synthesis elsewhere in the body. Exercise increases protein synthesis and breakdown with the net result of decreased muscle protein. Ingestion of amino acids stimulates muscle protein synthesis, exceeding the rate of breakdown, and muscle mass increases. In one study of elderly humans, patients maintained on bed rest for 10 days were given a dietary supplement of amino acids or placebo in a double-blind randomized fashion.[54] Supplementing essential amino acids in these individuals with absolute bed rest prevented decreases in muscle strength and function. Furthermore amino acid supplementation prevented the decreased rate of muscle synthesis that is normally seen with bed rest.[54] Amino acid stimulation of muscle protein synthesis and exercise is synergistic. Exercise combined with amino acid intake produces a greater response regarding muscle synthesis than response to individual treatments. This suggests exercise can prime the muscle response to the anabolic effects of amino acids or exercise activates muscle synthesis, but adequate amino acid precursors are needed to increase muscle mass. Postexercise supplementation with carbohydrate and protein or protein hydrolysate increases muscle glycogen stores and may facilitate recovery.[55-57] The effect of amino acid supplementation via infusion on muscle proteolysis in beagles was found to require concomitant glucose infusion; therefore an energy substrate for protein anabolism is required for the desired beneficial effect.[58] Branched chain amino acids (leucine, isoleucine, and valine) supplementation decreases branched chain amino acid catabolism and

exercise-induced muscle damage promoting muscle protein synthesis in humans[59-61]; however, results are contradictory and branched chain amino acids may only facilitate recovery of muscle following exercise.[62] Alanine supplementation improves high-intensity aerobic performance of muscle.[63] Carnitine is a quaternary ammonium compound biosynthesized from lysine and methionine and is a cofactor for transport of long-chain fatty acids across the mitochondrial membrane to liberate energy via β oxidation. In dogs, it has been shown to improve function and energy metabolism in muscle and may be useful in rehabilitation.[64,65] However glutamine supplementation does not appear to be beneficial in preventing muscle protein catabolism with exercise.[66]

Studies of protein nutrition often evaluate total dietary protein content instead of amino acid composition. Investigation to optimize amino acid profiles for specific benefits has identified "slow" and "fast" proteins. This classification is determined by the rate a protein exits the stomach, is hydrolyzed into peptides and amino acids, and is then absorbed. Plasma amino acid profiles of fast and slow proteins have shown that "blends" are more likely to produce maximal amino acid concentration for optimal protein synthesis. The blends maintain amino acid concentrations for an optimal time for longer duration of anabolism. Identifying and using protein blends for maximal muscle synthesis and strength during physical rehabilitation may be a reasonable goal with these current studies.

Obesity

Weight reduction from an obese state is beneficial in the management of osteoarthritis in humans.[67-69] In addition to improvement in mobility, weight reduction was associated with better quality of life scores in humans with knee osteoarthritis.[70] Three uncontrolled clinical studies of obese dogs with osteoarthritis demonstrated improvement in mobility.[71,72] In one study of nine dogs with body condition score of 5 out of 5 and coxofemoral osteoarthritis, obesity management resulted in loss of 11% to 18% of body weight, a decrease in body condition to optimal, and improvement in severity of subjective hind limb lameness scores.[71] In the second study of 16 dogs with coxofemoral osteoarthritis, weight loss of 13% to 29% of body weight and decrease of body condition to optimal resulted in improvement of ground reactive forces as well as improvement in subjective mobility and clinical signs of osteoarthritis.[72] In the third study, 14 client-owned dogs with clinical and radiographic signs of osteoarthritis participated in an open prospective clinical trial. Results indicated that body weight reduction caused a significant decrease in lameness with a weight loss of 6.10% or more. Kinetic gait analysis supported the results with a body weight reduction of 8.85% or more. These results confirm that weight loss should be presented as an important treatment modality to owners of obese dogs with osteoarthritis, and that noticeable improvement may be seen after modest weight loss of 6.10% to 8.85% body weight. Although these are uncontrolled clinical trials of a small number of dogs, results suggest that weight reduction may be beneficial in dogs with osteoarthritis.

Weight loss is beneficial when used in combination with physical rehabilitation in dogs. In a non-blinded prospective randomized clinical trial, 29 adult dogs that were overweight or obese (body condition score of 4/5 or 5/5) having clinical and radiographic signs of osteoarthritis were evaluated.[73] All dogs underwent weight loss. One group received caloric restriction and a home-based physical rehabilitation program and the other group received an identical dietetic protocol and an intensive physical rehabilitation program, including transcutaneous electrical nerve stimulation. Significant weight loss and improved mobility were achieved in both groups; however, greater weight reduction and better mobility was obtained in the group receiving intensive physical rehabilitation.[73]

Nutritional Compounds Evaluated in Dogs or In Vitro Using Canine Cells

There are other dietary supplements recommended for patients with osteoarthritis.[74-87] Very few have been evaluated in a controlled manner and even fewer have been evaluated in dogs with osteoarthritis.

Antioxidants

Formation of free radicals as a consequence of cellular metabolism occurs constantly, but potential deleterious effects are minimized by antioxidants. Balance between free radicals and antioxidant defenses is an essential factor in preventing development of noxious processes at cellular and tissue levels. Recent evidence supports the theory that excessive production of free radicals or the imbalance between concentrations of free radicals and antioxidant defenses may be related to processes such as aging, cancer, diabetes mellitus, lupus, and arthritis.[88] Progressive hypoxia resulting in production of reactive oxygen species may play a role in rheumatoid arthritis.[89] Thus antioxidant therapy may be of benefit in the treatment of osteoarthritis.[90]

S-Adenosylmethionine. S-adenosylmethionine (SAMe) is a co-substrate involved in transmethylation, transsulfuration, and aminopropylation reactions, which occur primarily in the liver. In controlled trials of humans with osteoarthritis, SAMe was as effective as nonsteroidal anti-inflammatory drugs (NSAIDs) and better than placebo in reducing pain and improving function with a lower likelihood of side effects[91-98]; however, no difference with an NSAID drug was found in one study.[99] A systematic review was inconclusive and evidence of efficacy was hampered by inclusion of small trials of questionable quality.[100] A double-blinded, placebo-controlled clinical trial of 33 dogs with presumed osteoarthritis did not show a benefit of

SAMe as a sole treatment.[101] An in vitro study showed SAMe adversely affected chondrocyte viability.[102] SAMe may reduce inflammatory mediators, increase levels of the antioxidant glutathione, increase cartilage synthesis, be chondroprotective, and maintain DNA methylation.[103]

Other Antioxidants. Most studies of antioxidant therapy for arthritis in humans involve rheumatoid arthritis, although several studies of knee osteoarthritis have been published. An early pilot study of vitamin E (tocopherols) demonstrated improvement in clinical signs and pain scores in 52% of 29 patients with knee osteoarthritis compared with 4% receiving placebo.[104] In another placebo-controlled study of humans with osteoarthritis of coxofemoral or knee joints, high-dose tocopherols was as efficacious as diclofenac in reducing pain and improving mobility.[105] This was also found in studies of humans with rheumatoid arthritis.[106,107] Other studies have not demonstrated a benefit of vitamin E for either rheumatoid arthritis or osteoarthritis.[108-110] Vitamin E has not been evaluated in dogs with osteoarthritis; however, large breed puppies fed a diet proportionately higher in protein, calcium, n-3 fatty acids, and antioxidants had increased lean body mass and improved cartilage turnover as maturity was attained.[111] In greyhounds and sled dogs, supplementation is associated with increased plasma levels; however, no difference in muscle damage with exercise has been noted.[112-114] Efficacy of vitamin C in humans with osteoarthritis has been difficult to determine due to contradictory results.[115-118] In dogs, vitamin C administration was found to increase vitamin C plasma levels, but was associated with decreased racing performance in greyhounds.[119] Selenium was not shown to be beneficial in humans with rheumatoid arthritis.[120] An antioxidant cocktail was administered for 6 weeks to 48 dogs. Dogs were assigned to four groups: untrained/not supplemented, untrained/supplemented, trained/not supplemented, and trained/supplemented. Metabolomic profiling showed that dogs receiving the antioxidant cocktail recovered to baseline values 24 hours after exercise, whereas dogs receiving no supplementation did not. Therefore administration of an antioxidant cocktail apparently facilitates recovery from exercise.[121]

Avocado/Soybean Unsaponifiables

Avocado/soybean unsaponifiables (ASUs) are composed of the unsaponifiable fractions of avocado and soybean oils in a one-third to two-thirds proportion.[122,123] Avocado/soybean unsaponifiables have anti-osteoarthritic properties by inhibiting interlukin-1 and stimulating collagen synthesis in cartilage cultures.[123-125] In vivo there is one report in an ovine meniscectomy model of osteoarthritis that supports disease-modifying capabilities.[125,126] Human clinical trials have shown some beneficial effects of ASUs on clinical symptoms of osteoarthritis and they may have some structure modification capabilities.[127-129] However there are conflicting data as other studies found no long-term benefits.[128,130-132] In one study of dogs, osteoarthritis was induced by anterior cruciate ligament transection that then received placebo or ASUs (10 mg/kg per 24 hours).[133] The size of macroscopic lesions of the tibial plateau, severity of cartilage lesions, synovial cellular infiltration, and inducible nitric oxide synthase were decreased significantly and there was reduced loss of subchondral bone volume and calcified cartilage thickness in the group receiving ASUs.[133]

Blueberries

Blueberries are flowering plants of the genus *Vaccinium*, which includes cranberries and bilberries. They may have antioxidative and antiinflammatory actions because they contain pterostilbene, anthocyanins, proanthocyanidins, resveratrol, flavonols, and tannins.[134] In a study of sled dogs, those receiving a blueberry supplement had higher antioxidant status than those not receiving the supplement; however, there was no difference in creatinine kinase activity and isoprostane levels (a measure of oxidative stress) in dogs receiving blueberries and those that did not.[135]

Boswellia serrata

Boswellia, also known as Boswellin or Indian frankincense, comes from the Indian *Boswellia serrata* tree. Resin from the bark of this tree is purported to have antiinflammatory properties derived primarily from 3-O-acetyl-11-keto-β-boswellic acid (AKBA), which inhibits 5-lipoxygenase and matrix metalloproteinases, and decreases tumor necrosis factor α and interleukin 1β.[136,137] Following oral administration, AKBA has poor bioavailability. However, two AKBA-containing compounds, Alfapin and 5-Loxin, have good bioavailability.[138] Boswellia resin has been shown to improve clinical signs and pain in humans in controlled studies.[139-141] Boswellia resin has been evaluated in 24 dogs in an open multicenter study.[142] Improvement in clinical signs, lameness, and pain was found in 17 of 24 dogs. Diarrhea and flatulence occurred in five dogs.

Chondroprotectants

Chondromodulating agents are purported to slow or alter the progression of osteoarthritis. These agents are considered to be slow-acting drugs in osteoarthritis (SADOA) and can be subdivided into symptomatic slow-acting drugs (SYSADOA) and disease-modifying osteoarthritis drugs (DMOAD). Beneficial effects may include a positive effect on cartilage matrix synthesis and hyaluronan synthesis by synovial membrane, as well as an inhibitory effect on catabolic enzymes in osteoarthritis joints.[143] Compounds fall under two different categories. One group includes agents that are approved by the US Food and Drug Administration, such as polysulfated glycosaminoglycan, which can have label claims of clinical effects. The second group

includes products that are considered to be nutritional supplements, which are not required to undergo efficacy testing, and legally cannot claim any medical benefits. Examples of this group include glucosamine and chondroitin sulfate. Although many of these products are administered as a supplement or alternative treatment, some, such as glucosamine and green-lipped mussels, are incorporated into pet foods.

Glucosamine and Chondroitin. Glucosamine is an amino sugar that is a precursor for biochemical synthesis of glycosylated proteins and lipids. D-glucosamine is made naturally in the form of glucosamine-6-phosphate and is the biochemical precursor of nitrogen-containing sugars.[144] Specifically glucosamine-6-phosphate is synthesized from fructose-6-phosphate and glutamine[145] as the first step of the hexosamine biosynthesis pathway. The end product of this pathway is UDP-N-acetylglucosamine, which is used for making glycosaminoglycans, proteoglycans, and glycolipids. Because glucosamine is a precursor for glycosaminoglycans, and glycosaminoglycans are a major component of joint cartilage, supplemental glucosamine may help enhance cartilage synthesis and there are in vitro and clinical data to support this claim, although in vitro studies often use concentrations not achieved in serum or plasma after oral administration.[146-152] There are conflicting data on evidence of clinical effect of glucosamine-chondroitin in osteoarthritis.[74,75,153-161] In a randomized, double-blind, positive-controlled clinical trial comparing glucosamine hydrochloride and chondroitin sulfate to carprofen in 35 dogs with osteoarthritis, carprofen-treated dogs had improvement in five subjective measures, whereas dogs treated with glucosamine-chondroitin sulfate had improvement in three of five measures, but only at the final assessment point of 70 days.[162] A 60-day prospective, randomized, double-blinded, placebo-controlled trial of 71 dogs with osteoarthritis assessed subjective and objective measures comparing carprofen, meloxicam, glucosamine-chondroitin, and placebo. Results indicated that objectively measured variables improved significantly with carprofen and meloxicam, but not with glucosamine-chondroitin or placebo. Subjective findings of veterinarians agreed with results of objective evaluation, but subjective assessment by owners identified improvement only with meloxicam.[163] Based on these results there is weak clinical evidence of benefit of glucosamine-chondroitin in dogs with osteoarthritis.[153-155]

When evaluating glucosamine and chondroitin sulfate inclusion in a manufactured dog food, two questions should be asked. Is the glucosamine and chondroitin sulfate in the food stable and bioavailable? And, is the amount of glucosamine and chondroitin sulfate in the food enough to provide a beneficial effect? It is difficult to find the amount of glucosamine and chondroitin sulfate in pet foods. Many dog foods formulated and marketed for adult dogs, geriatric dogs, and large breed growth contain glucosamine and chondroitin sulfate, but the amounts are not readily available from the manufacturer or from electronic or print information. These compounds are not recognized by the American Association of Feed Control Officials, and so are not included in dog nutrient profiles. Furthermore, they are not considered as generally regarded as safe (GRAS) ingredients. However, there is no evidence to suggest that they are not safe at the levels used in food.

The New Zealand Green-Lipped Mussel

The New Zealand green-lipped mussel (*Perna canaliculus*) is a rich source of glycosaminoglycans, although its proposed benefit is thought to be from its antiinflammatory effects.[164] In 1986 dried mussel extracts became available that were stabilized with a preservative. The earlier studies that found no beneficial effect of the green-lipped mussel on arthritis used preparations that had not been stabilized, a point that may help explain some of the discrepancies in the research. A stabilized lipid extract is more effective than a nonstabilized extract at inhibiting inflammation.[165] In an uncontrolled clinical trial stabilized lipid extract administration resulted in an 80% improvement in humans with rheumatoid arthritis.[166] In a randomized controlled clinical study of 31 dogs with arthritis, green-lipped mussel powder (0.3%) was added to the diet during processing for one group of dogs.[167] Compared with the control group, which was fed the same diet without added green-lipped mussel powder, there were significant improvements in subjective arthritis scores, joint swelling, and joint pain in the treated group. However, these data must not be overinterpreted. In a randomized, double-controlled and double-blinded clinical trial, 45 dogs with osteoarthritis were evaluated. Dogs receiving green-lipped mussels had improved mobility when compared with placebo; however, it was not as effective as carprofen.[168] In a study of 81 dogs with presumptive osteoarthritis, clinical signs improved in both the green-lipped mussel and placebo-treated groups when compared with baseline. On day 56 of the study, dogs receiving green-lipped muscle had improved clinical signs but not musculoskeletal scores.[169] An uncontrolled study of 85 dogs that were fed a green-lipped mussel supplemented diet for 50 days showed reduction of a composite arthritic score when compared with baseline scores on various diets dogs were consuming.[170] In systematic reviews of agents used to treat canine osteoarthritis, the data regarding the benefits of green-lipped mussel extract in dogs were promising but uncertainties existed relating to the scientific quality of the data and no definitive relationship has been proved between clinical improvements and the therapy.[154,155]

Omega-3 Fatty Acids

Degenerative osteoarthritis involves an inflammatory component. Thus, it may be possible to modify the inflammation by nutritional components, specifically omega-3

(n-3) fatty acids. Arachidonic acid (an n-6 fatty acid) is incorporated into cell membranes, and when metabolized, it yields prostaglandins, leukotrienes, and thromboxanes of the two and four series. Many drugs used to treat degenerative osteoarthritis inhibit conversion of arachidonic acid to these eicosanoids. These n-6-derived eicosanoids have, for the most part, vasoactive and proinflammatory effects. Substituting an n-3 fatty acid in the membrane may decrease these responses. Metabolism of n-3 fatty acids results in eicosanoids of the three and five series, which are less vasoactive and less proinflammatory. In addition to modulating cytokines, n-3 fatty acids have been shown to reduce expression of cyclooxygenase-2, lipoxygenase-5, aggrecanase, matrix metalloproteinase 3 and 13, interleukin-1α and β, and tumor necrosis factor α.[171-175] In addition to these effects, novel oxygenated products generated from n-3 fatty acids, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) have been identified in resolving inflammatory exudates and tissues and are called Resolvins (resolution phase interaction products) and docosatrienes.[176-178] They may have a role in inflammatory diseases such as osteoarthritis.[179-184]

Several studies have demonstrated a beneficial response to n-3 fatty acid incorporation into diets of humans with rheumatoid arthritis,[185-189] although other studies have not demonstrated a benefit.[190] There is a growing body of data showing positive effects of n-3 fatty acids on cartilage and its metabolism in the face of degradative enzymes; n-3 fatty acid supplementation can reduce inflammatory and matrix degradative responses elicited by chondrocytes during osteoarthritis progression.[172-174,191] Eicosapentaenoic acid appears to be more effective than DHA and α-linolenic acid.[171]

An unpublished study was performed in dogs evaluating omega-3 fatty acids and experimentally induced stifle arthritis.[192,193] Eighteen dogs were randomly assigned to one of three isocaloric diet groups containing 21.4% fat (dry matter basis) differing only in their fatty acid composition: a diet with an n-6 to n-3 fatty acid ratio of 28.0:1.0 (high n-6 diet), a diet with an n-6 to n-3 fatty acid ratio of 8.7:1.0 (control diet), and a diet with an n-6 to n-3 fatty acid ratio of 0.7:1.0 (high n-3 diet). Diets were fed for a total of 21 months. Dogs were begun on their assigned diet 3 months before surgical transection of the left cranial cruciate ligament, then continued until surgical repair 6 months later, and were maintained for an additional 12 months following repair. When compared with the high n-6 and control diets, consumption of the high n-3 diet was associated with lower serum concentrations of cholesterol, triglycerides, and phospholipids, lower synovial concentration of prostaglandin E2, better ground reaction forces, and less radiographic changes of osteoarthritis. Synovial membrane fatty acid composition mirrored the fatty acid composition of the diets consumed by the dogs. Another study in dogs reported results of owner observations who perceived improvement in their pet's arthritic signs when treated with fatty acids for various dermatologic problems.[194] In a study of 127 dogs with osteoarthritis, dogs fed a diet with a 31-fold increase in total n-3 fatty acid content and a 34-fold decrease in n-6 to n-3 fatty acid ratio for 6 months had improved ability to rise from a resting position and play when compared with dogs fed a control diet.[195] In a randomized, double-blinded, controlled clinical trial of 38 dogs with osteoarthritis, dogs fed a diet containing 3.5% n-3 fatty acids for 90 days had improved peak vertical force values and subjective improvement in lameness and weight bearing when compared with a control diet.[196] Another randomized, controlled clinical trial was performed with 131 dogs with stable chronic osteoarthritis treated with carprofen. Over a 12-week study carprofen dosage decreased significantly faster in dogs fed a diet supplemented with n-3 fatty acids when compared with a control diet.[197] Based on results of these studies there is a rationale for n-3 fatty acid supplementation or feeding diets containing increased n-3 fatty acid levels to dogs with osteoarthritis.

P54FP (Turmeric)

P54FP is an extract of Indian and Javanese turmeric, *Curcuma domestica* and *Curcuma xanthorrhiza*, which contains a mixture of active ingredients including curcuminoids and essential oils.[198] There is evidence that these active ingredients possess antiinflammatory activity. Specifically curcumin has been shown to inhibit prostaglandin E2, cyclooxygenase-2, nuclear factor κB, matrix metalloproteinase-3 upregulation, and IL-1β–induced proteoglycan degradation.[199-201] One randomized, blinded study in dogs with osteoarthritis found no difference in objective ground reaction forces but did see some improvements in certain subjective outcome measures.[198]

Special Milk Protein Concentrate

Milk contains a number of biologically active compounds including immunoglobulins, cytokines, enzymes, hormones, and growth factors. These compounds impart antiinflammatory properties that have been recognized in human breast milk[202] and milk from hyperimmunized cows.[203,204] A special milk protein concentrate prepared from milk of hyperimmunized cows exerts antiinflammatory properties.[205] The antiinflammatory property does not appear to be caused by inhibition of arachidonic acid metabolism, but by suppression of neutrophil migration from the vascular space.[206] A randomized, controlled clinical trial has been performed evaluating special milk protein concentrate (Microlactin, Stolle Milk Biologics, Cincinnati, OH) in dogs with naturally occurring osteoarthritis.[207] In this study dogs receiving the special milk protein concentrate had improvement in subjective clinical signs of osteoarthritis and owner global assessment compared with

dogs receiving placebo. Further studies need to be completed to validate these findings.

Zeel®

Zeel® is an over-the-counter homeopathic preparation based on highly diluted extracts from plants, animals, and minerals (sulfur) as well as defined biochemical substances including coenzyme A, DL-alpha lipoic acid, sodium diethyl oxalate, and nicotinamide adenine dinucleotide.[208,209] It has been evaluated in two controlled studies in dogs. Active ingredients (common name) include *Arnica montana* (mountain arnica), *Sanguinaria canadensis* (blood root), *Solanum dulcamara* (bittersweet), *Symphytum officinale* (comfrey), *Toxicodendron quercifolium* (poison oak), *Cartilago suis* (porcine cartilage), *Embryo totalis suis* (porcine embryo), *Funiculus umbilicalis suis* (porcine umbilical cord), *Placenta totalis suis* (porcine placenta), coenzyme A, dl-α-lipoic acid, nicotinamide adenine dinucleotide, sulfur, and sodium diethyl oxalate.[208,209] In one study in dogs ($n = 68$) greater than 1 year of age diagnosed with osteoarthritis, it was compared with carprofen in a multicenter, prospective, observational open-label cohort study in 12 German veterinary clinics.[208] In another study in dogs ($n = 44$) greater than 1 year of age diagnosed with osteoarthritis, it was compared with carprofen and a placebo.[209] Clinical signs and several measures of osteoarthritis improved significantly with treatment in both studies; however in one study,[209] it was not as effective as carprofen. The composition of the products and the dosage of Zeel® differed between the two studies, which confounds interpretation of results.

Other Nutritional Compounds Evaluated in Other Species or In Vitro

Articulin F

Articulin F is an herbo-mineral formulation containing roots of *Withania somnifera*, the stem of *Boswellia serratia*, rhizomes of *Curcuma longa*, and a zinc complex. In a double-blind, placebo-controlled study of 42 humans with osteoarthritis, Articulin F was associated with improvement in pain and function, but not in radiologic findings.[139]

Barberry

Extracts obtained from the roots and barks of various *Berberis* species are used as folk remedies worldwide for the treatment of various inflammatory ailments. Effects of the extracts and fractions from the roots of *Berberis crataegina* have been studied using various in vivo models of inflammation in mice and rats and have resulted in potent inhibitory activity against carrageenan- and serotonin-induced hind paw edema, acetic acid-induced increased vascular permeability, castor oil–induced diarrhea, and Freund's complete adjuvant–induced (FCA) arthritis models.[210,211]

Boron

Boron deficiency in food may be part of the cause of some arthritides.[212] Epidemiologic studies suggest that humans in countries with low boron intake (less than 1.0 mg/day) have a higher risk of arthritis development when compared with humans in countries in which boron intake is higher (3-10 mg/day).[213] In a double-blind, placebo versus boron supplementation trial with 20 subjects with osteoarthritis, a significant favorable response to a 6-mg boron/day supplement was found; 50% of subjects receiving supplement improved compared with 10% receiving the placebo.[213] Whether boron deficiency occurs in pet dogs is unknown. Furthermore whether boron supplementation and what dosage of boron would be best is unknown.

Cat's Claw

Cat's claw, an Amazonian medicinal plant (*Uncaria tomentosa* and *U. guyanensis*), has antiinflammatory and antioxidant effects and has been shown to decrease clinical signs of knee arthritis and rheumatoid arthritis in humans.[214,215] Cat's claw has been found to inhibit lipopolysaccharide-induced inducible nitric oxide gene expression, nitrate formation, cell death, PGE_2 production, activation of NF-κB, and TNFα.[216-219] It also acts as a free radical scavenger, suppresses TNFα and apoptosis, and is cytoprotective against oxidative damage.[217,220] It has been used to treat osteoarthritis in humans. In a metaanalysis of available data,[81] three studies demonstrated effectiveness of cat's claw alone or in combination with other treatment for osteoarthritis.

Cetyl Myristoleate

Cetyl myristoleate is the cetyl ester of myristoleic acid and has antiinflammatory and immune-modulating activity.[221] Despite conflicting results in rodent models,[222,223] it was shown to improve function and range of motion when compared with placebo in 64 humans with knee osteoarthritis.[224]

Chinese (Baikal) Skullcap

Chinese or Baikal skullcap (*Scutellaria baicalensis*) is a flowering plant used in traditional Chinese medicine.[81] Sometimes *Scutellaria lateriflora* (North American Skullcap) is mistaken for *Scutellaria baicalensis* (Baikal Skullcap). This can result in intake of the incorrect species that is often contaminated with other plants at high enough levels to be hepatotoxic.[225] Chinese skullcap or its B-ring flavones and flavonoids have been shown to exhibit antispasmogenic, antivasoconstrictor, antihypertensive, antiplatelet aggregation, and antiinflammatory properties including inhibition of cyclooxygenase in vitro.[81,226-229] Flavocoxid (Limbrel®, Primus Pharmaceuticals Inc., Scottsdale, AZ) contains a proprietary blend of free-B ring flavonoids and flavins from the root of *Scutellaria*

baicalensis and the bark of *Acacia catechu*. The primary components of Flavocoxid are baicalin and catechin, concentrated and standardized to greater than 90% purity.[230] It has been shown to inhibit activities of cyclooxygenase 1-, 2-, and 5-lipoxygenase[231-233] and was demonstrated to be safe[230]; however, its safety has not been evaluated when administered with other cyclooxygenase inhibitors such as NSAIDs or glucocorticoids. Caution should be exercised when used with concurrent cyclooxygenase inhibitors. It appears to be beneficial in humans with osteoarthritis.[231,234]

Collagen Hydrolysates

Collagen hydrolysate is obtained by enzymatic hydrolysis of collagenous tissues from mammals, which is similar in amino acid composition to type II collagen, and provides high levels of glycine and proline that are involved in regeneration and stability of cartilage.[82,235] Animal models showed benefits with treatment of induced osteoarthritis.[236] In humans, it has been shown to improve joint pain and mobility and reduce analgesic medications in healthy active individuals with joint pain but without osteoarthritis, and in humans with mild to severe osteoarthritis.[237-240]

Creatine

Creatine is used in muscle for production of adenosine triphosphate (ATP), which provides energy for muscle contraction. Creatine is sometimes used by body builders and people who exercise with the intent to increase muscle mass and muscular energy. However, some recommend its use with arthritis, especially rheumatoid arthritis in humans. In rheumatoid arthritis skeletal muscle weakness often accompanies the arthropathy. In one study creatine supplementation to humans with rheumatoid arthritis increased serum and skeletal muscle creatine content, but failed to increase skeletal muscle creatine phosphate concentration or strength.[241] In another study of humans undergoing total knee replacement, creatine supplementation did not improve body composition or skeletal muscle strength.[242] Creatine supplementation has been shown to improve physical function, lower limb lean mass, and quality of life in postmenopausal women with knee osteoarthritis undergoing strengthening exercises.[243]

Devil's Claw

Harpagophytum procumbens (devil's claw) has been used as an analgesic, a remedy for fever and allergies.[244] The major chemical constituents of *Harpagophytum* are iridoid glycosides, phytosterols, aromatic acids, and flavonoids. Glycosides found in the tubers of the plant appear to be the most therapeutically important constituents. Whole-plant extracts appear to have a better therapeutic effect than those prepared from isolated parts.[244] A review of clinical trials using *H. procumbens* preparations for treatment of joint and lower back pain found that using extracts containing 50 to 60 mg harpagoside daily gave more reliable results and were more effective at alleviating pain and improving mobility than extracts with lower amounts.[245,246] An in vitro study using two rat cell lines found that the glycosides inhibited lipopolysaccharide-induced inducible nitric oxide and cyclooxygenase-2 expression via nuclear factor κB suppression, thereby inhibiting inflammation.[247] Devil's claw is chondroprotective, possibly because of inhibition of inflammatory mediators, including cyclooxygenase-2, leukotrienes, nitric oxide, tumor necrosis factor-α, and interleukin-1β, and by inhibiting matrix metalloproteinases and elastase.[247,248] (Note that devil's claw may increase gastric acidity; therefore caution is recommended when using this compound with NSAIDs or corticosteroids.)

Du Huo Ji sheng Wan

Du Huo Ji sheng Wan is a Chinese herbal product containing *Angelica Pubescentis*, Mulberry mistletoe, *Campanulaceae*, *Hoelen*, *Eucommiae*, *Angelica*, *Rehmannia*, cinnamon, licorice (Glycyrrhiza), and ginger. In a double-blind, controlled study comparing it with diclofenac, pain and function scores improved in both groups to the same extent but occurred more slowly in the Du Huo Ji sheng Wan treatment arm.[249]

Ginger

Ginger (*Zingiber officinale*), rich in gingerols and shogaols, has been advocated for treatment of a variety of diseases, including osteoarthritis because it suppresses prostaglandin and nitric oxide production.[250,251] In one crossover study in humans significant improvement in symptoms for both ginger and ibuprofen groups was found before crossover; however, at the study's end there was no difference between ginger and placebo.[252] In another study it was found to be less effective than turmeric.[253]

Green Tea

Green tea (*Camellia sinensis*) is a rich source of polyphenols known as catechins, including epigallocatechin 3-gallate (EGCG), which has shown promise in treatment of arthritic diseases.[254] Epigallocatechin 3-gallate has been shown in vitro and in vivo in rodent models of arthritis to have chondroprotective activity through modulation of interleukin-1 and -6 and decreased matrix metalloproteinase and nitric oxide activities.[255-259]

Homeopathy

Homeopathy is a form of alternative and complementary medicine based on vitalist philosophy, which interprets diseases and sickness as caused by disturbances in vital force or life force. Homeopathic practitioners treat patients using highly diluted preparations that follow the "law of similars" in which "let like be cured by like." Homeopathic remedies are prepared by serial dilution with shaking by

forceful striking on an elastic body (called *succussion*). Each dilution followed by succussion is assumed to increase effectiveness (called *potentization*). In humans with osteoarthritis, homeopathy has not been shown effective, has been found to be less effective than analgesic administration, or beneficial effects were attributed to the consultation and not to the homeopathic remedy.[132,260-265] One homeopathic remedy, Zeel®, has been evaluated in dogs (see Nutritional Compounds Evaluated in Dogs).

Methylsulfonylmethane

Methylsulfonylmethane (MSM) is a sulfur-containing compound found in plant and animal tissues. Three clinical trials in humans have shown positive results when compared with placebo either alone or in combination with glucosamine[266-268]; however, metaanalyses have failed to demonstrate a clinically effective reduction in pain or improvement in function.[269,270]

Phytodolor N

Phytodolor N is a fixed combination of extracts from aspen leaves and bark (*Populus tremula*), common ash bark (*Fraxinus excelsior*), and golden rod herb (*Solidago vigaurea*). It appears to have antiinflammatory, antiedematous, antioxidative, and analgesic properties.[271,272] Three studies in humans reported results in favor of Phytodolor N when compared with placebo, although results are skeptical as the manufacturer undertook the studies or used a higher dose.[273,274]

Pine Bark Extract (Pycnogenol®)

Pycnogenol® is a special standardized extract containing polyphenols and procyanidins from the bark of the French maritime pine (*Pinus pinaster*).[82] It exhibits antiinflammatory and antioxidative activity as well as inhibits matrix metalloproteinase and inducible nitric oxide production.[275] Studies in humans with osteoarthritis show a positive effect on decreasing pain and increasing function.[276-279]

Quercetin

Quercetin is a plant-derived flavonoid that may exhibit antiinflammatory and antioxidant properties via inhibition of tumor necrosis factor-α–mediated interleukin-8 and monocyte chemoattractant protein-1 expression.[280] In two studies, it was not effective or only minimally effective in vivo or in vitro[281,282]; however, in one study in humans with osteoarthritis or rheumatoid arthritis, it improved synovial fluid quality and pain scores when administered in combination with glucosamine and chondroitin sulfate.[283]

Resveratrol

Resveratrol is a type of natural phenol found in high concentration in grape skin and red wine. It has antiinflammatory, antioxidant, and chondroprotective properties such as increasing proteoglycan synthesis and chondrocyte proliferation, and by suppressing interleukin-1, reactive oxygen species, p53-induced apoptosis, leukotriene B4, prostaglandin E2, tumor necrosis factor α, and matrix metalloproteinases, which may be beneficial for osteoarthritis.[284-288] It acts, in part, by activating Sirtuin 1, a histone deacetylase, and Bcl2.[289,290] Only in vitro studies have been performed.

Rose Hip and Seed Powder

The rose hip is the fruit of the rose plant that typically is red-to-orange, but ranges from dark purple to black in some species. It has antiinflammatory and antioxidative effects and has shown benefit in humans with rheumatoid arthritis or chronic pain.[291-294]

Rosemary

Rosemarinus officinalis is a common spice and household plant that is also used for medicinal purposes. Experimentally in rodents, it reduces nocioceptive pain in various kinds of induced pain.[295] It has been studied as rho iso-alpha acids from hops, rosemary, and oleanolic acid and decreases prostaglandin production through inhibition of cyclooxygenase, thereby decreasing pain.[296,297]

SKI 306X

SKI 306X is a purified extract from a mixture of three oriental herbal medicines (*Clematis mandshurica*, *Trichosanthes kirilowii*, and *Prunella vulgaris*) that has been widely used for the treatment of inflammatory diseases including knee osteoarthritis in humans. In vitro studies show SKI 306X or *Clematis* inhibits cartilage degradation, chondrocyte apoptosis, and production of tumor necrosis factor α and interleukin-1 β.[298-303] Extracts from *Clematis vitalba* exhibit antiinflammatory, antinociceptive, and antipyretic effects in induced osteoarthritis in mice.[304] In a double-blind, controlled study of 96 patients with knee osteoarthritis, patients receiving SKI 306X showed lower levels of pain and better function when compared with placebo.[305] In another study of 249 patients with knee osteoarthritis, SKI 306X was equally as effective as diclofenac.[306]

Willow Bark Extract (*Salix* spp.)

Extracts from the bark of *Salix* species have been used for treatment of pain. The analgesic effect is attributed to salicin and/or its metabolite, salicylic acid.[307] It should be administered cautiously or not at all with NSAIDs and glucocorticoids because of these compounds. The mechanisms of action of willow bark extract include inhibition of cyclooxygase-2,[308] lipoxygenase,[309] cytokine release,[308,309] and hyaluronidase,[310] and it may have antioxidant activity.[309,311,312] Evaluation in humans with osteoarthritis or rheumatoid arthritis has yielded differing results regarding effectiveness. A moderate analgesic effect was shown in one study,[313] whereas in two other studies no effect was

found.[314] (Note that because willow bark extract contains the parent compound of aspirin, concurrent use with other NSAIDs or corticosteroids is not recommended. Also, the half-life of this compound is relatively long.)

Nutrition and Osteoarthritis in Cats

Osteoarthritis occurs commonly in cats[315-319]; however, there is a paucity of information regarding nutrition or nutritional compounds in management. In a prospective, blinded, parallel group study evaluating a diet high in n-3 fatty acids and supplemented with green-lipped mussel extract and glucosamine/chondroitin fed to cats with osteoarthritis over 9 weeks, there was an improvement in mobility of cats fed the supplemented diet and a decline in cats fed the control diet.[320] Although other nutritional compounds are used in cats with osteoarthritis, none has been evaluated in a controlled manner.[86,317,321,322]

Summary

In summary, this chapter provides guidelines on nutritional requirements for dogs undergoing rehabilitation and how nutrition can influence the healing process. Numerous supplements are available and used in small animal rehabilitation, and their efficacy should be evaluated using an evidence-based approach to define their use in rehabilitaiton.

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16 Nursing Care of the Rehabilitation Patient

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Nursing care is essential during treatment of the physical rehabilitation patient. As discussed in depth in Chapter 7, muscles and tendons quickly atrophy with immobilization or reduced activity, and other tissues such as cartilage, ligaments, and bones also undergo rapid changes during the postoperative period. Starting physical rehabilitation as soon as it is safe can minimize these changes. There are several considerations one should consider when working with a postoperative patient, some of which may be severely injured and therefore recumbent or nonambulatory. Taking care of a patient that is nonambulatory or weakly ambulatory can be emotionally and physically challenging. It may be best to start with basic functions, such as learning to stand and walk again. Nursing care during the critical postoperative period in the hospital can be achieved by veterinarians, technicians, assistants, and ultimately the owner. It is important for every person involved to understand the recovery process to provide the best supportive care possible. Rehabilitation should begin as soon as the animal is stable, and before the onset of complications that are often associated with prolonged immobility. A proactive approach to rehabilitation requires less effort, and better outcomes may be obtained than one that is in response to a developing problem.

Preoperative Considerations

The importance of history taking, orthopedic examination, neurologic examination, and lameness and gait analysis has been discussed at length in other chapters. However, it is vital that the practitioner has a clear working knowledge of these assessment tools. One must also have a working knowledge of common orthopedic and neurologic conditions and the medical management and surgical procedures that apply. A review of the common conditions affecting dogs is warranted. With this information in mind one can apply it to nursing care.

Clean, Dry, Safe, and Comfortable Housing

Veterinary professionals have a responsibility to act as advocates for animals when people bring their pets for care and treatment. Providing a clean, dry bed in an appropriate cage or enclosure is vital (Figure 16-1). In some veterinary hospital environments housing is limited to cages elevated above the ground or runs enclosed by fencing, thus creating a "step" in and out of the cage or run. These circumstances provide challenges for the preoperative/postoperative patient and those who care for them. It may seem elementary to consider but patients should be cared for as if they were our own pets and we should occasionally stop to consider, "If I were my patient, how would I feel? What would make me feel better?"

Bedding and Decubital Ulcers

Rubber mats may be used to provide appropriate footing in cages and kennel runs (Figure 16-2). An essential factor with any orthopedic or neurologic patient that is unable to walk is preventing decubital ulcers. Decubital ulcers develop over bony prominences such as the olecranon process, acromion process, greater trochanter, tuber ischium, and medial and lateral malleoluses. Prevention is crucial and begins with proper bedding. A helpful rule of thumb is that 1 inch of bedding should be used for every 40 pounds of body weight. Bedding should be changed daily and the cage or run cleaned daily as well. It has been well documented that frequent position changes and ample bedding decrease the incidence of pressure sores, edematous limbs, and joint and muscle stiffness.[1,2] Recumbent patients should be turned every 4 to 6 hours at a minimum.

Urine/Fecal Scald

Providing absorbent pads underneath the patient to facilitate the absorption of urine and feces of recumbent patients is essential to keep the patient and cage from becoming excessively soiled. Alternatively larger patients that are incontinent may be placed on fenestrated grates to allow excess urine and feces to drain away from the patient. Urine- or fecal-scalded skin typically appears red, irritated with or without serous exudate, and is caused by prolonged exposure to urine or feces (Figure 16-3). Proper nursing care should be initiated immediately if scalding occurs and consists of cleansing the area, drying thoroughly, applying creams such as Desitin (Johnson & Johnson, New Brunswick, NJ) or Skin Sake (Skin Sake, Hamburg, AR) (Figure 16-4) to help protect the skin and baby powder to the fur



Figure 16-1 Patient in clean, dry, comfortable cage. Note placement of bedding, absorbent pad, and folded towel to support the head and neck. Pieces of foam can be used to position the patient in sternal recumbency if necessary.



Figure 16-2 Rubber mats are commercially available (½" Eco-soft tiles, Foamtiles.com, Chandler, AZ). They can be pieced together to fit a variety of cages and are also easily cleaned and sanitized between patients.

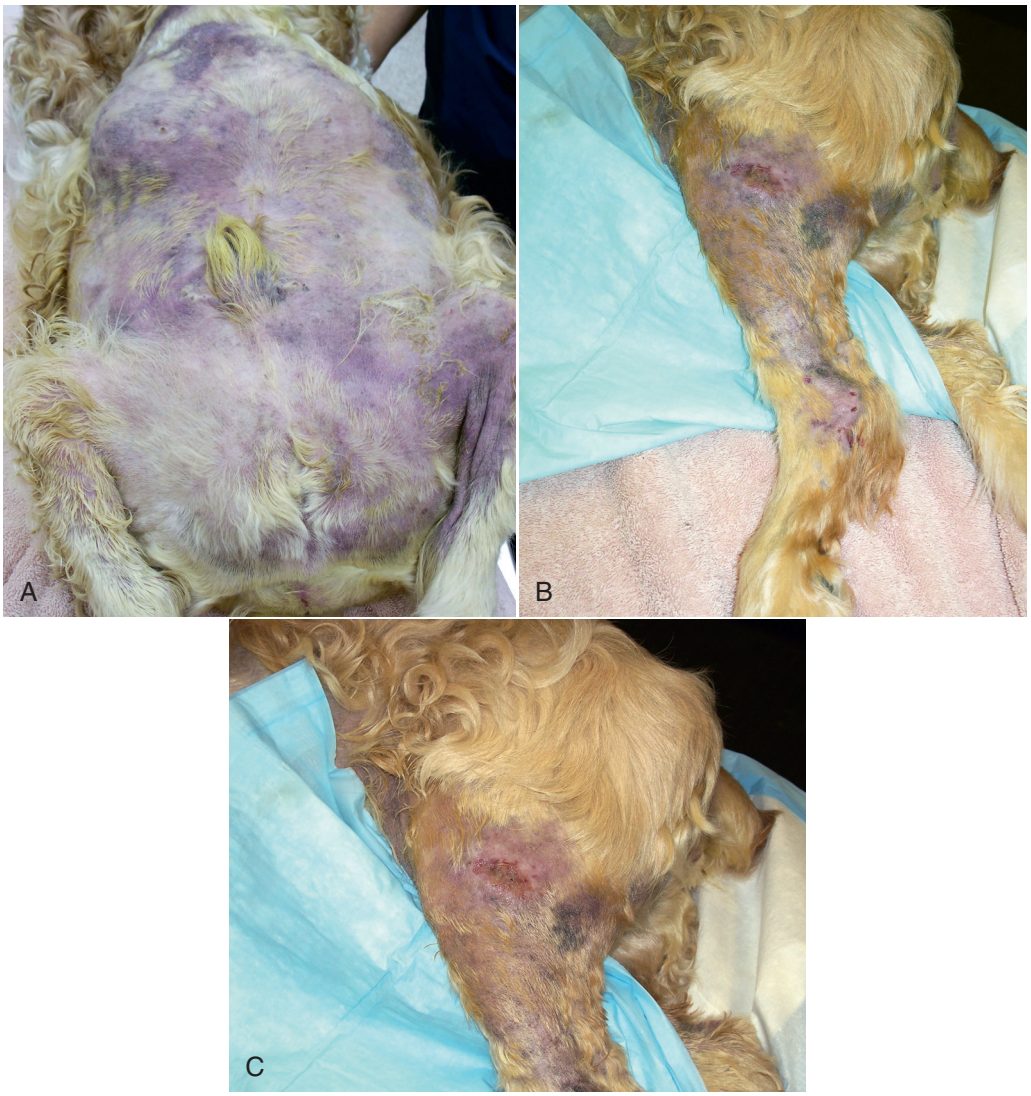


Figure 16-3 Patient with absence of deep pain with urine scald. **A,** View of abdomen. **B,** Lateral aspect of hindlimb. **C,** Close up view of lateral aspect of hindlimb.



Figure 16-4 Skin Sake (Skin Sake, Hamburg, AR) or other skin barrier cream can be applied to areas of skin with urine scald.



Figure 16-5 Cavilon (3M) is another product that may be applied as a skin protectant.

to absorb additional fluid. The importance of preventing the patient from licking its urine/perianal scald, wounds, and/or incisions is paramount. A no sting barrier film (Cavilon, 3M, St. Paul, MN) (Figure 16-5) also can be used to protect the skin.

Positioning of the Neurologic Patient

If the patient is unable to sit sternally, foam rolls, pads, wedges, or rolled-up towels can be placed beside the patient to help it maintain a sternal position and prevent it from falling over (Figure 16-6).

Elizabethan Collars

The Elizabethan collar, or e-collar, is very helpful to discourage patients from licking their incision. The art of a functioning e-collar lies in the sizing and fitting of the collar. Elizabethan collars are typically made of plastic, but softer cloth e-collars are also commercially available. When placed appropriately the brim of the e-collar should extend well past the patient's nose (Figure 16-7). This ensures the patient cannot lick the incision. The collar should be comfortably snug around the patient's neck. To check the tightness of the collar, two fingers are passed under the collar. If two fingers cannot be passed under the



Figure 16-6 Patient with an atlantoaxial subluxation, properly positioned to maintain a sternal position.



Figure 16-7 Properly placed e-collar, with clear sides and adjustable Velcro affiixing strip (3M). Note the brim of the collar extends beyond the patient's nose.

collar, it is too tight. If three or more fingers can be passed under the collar, it is too loose. Most commonly, e-collars are too loose and the patient has little trouble getting it off. Patients must become accustomed to daily tasks such as eating and drinking while wearing an e-collar. Food and water must be placed close enough to the patients for them to reach the bowls. Attention must be paid to the patient's appetite and willingness to eat. Many patients have difficulty learning to eat while wearing an e-collar. In this case the e-collar can be removed, feeding should be supervised, and the collar promptly reapplied after eating or drinking. In addition many patients refuse to eat or drink during their stay at the hospital. Hand feeding or assisted syringe feeding using pureed canned food is recommended, but feeding should be slow enough to allow ample time for swallowing. Patients should wear the e-collar at all times

in the hospital and at home until the sutures or staples are removed and any open wounds are adequately healed.

Medications

Although the decision regarding prescribed medications is ultimately the veterinarian's decision, the adept veterinary technician must always be aware of the current medications for each patient. Unflavored medications are best administered disguised, if possible, in food or a treat before mealtimes. The technical staff often is responsible for medicating patients, and this can be facilitated with soft, treat-like dog medication devices (Greenies Pill Pockets, Nutro Products, Inc., Franklin, TN). The technical staff is in a position to make the veterinarian aware of any discrepancies regarding medication, such as a history of concurrent nonsteroidal antiinflammatory drugs (NSAIDs) and corticosteroids, or an abrupt switch from one NSAID to another without a washout time in between. The veterinary technician is also essential to identify side effects a patient may be experiencing, such as vomiting, diarrhea, or melena. These classic NSAID side effects usually occur 2 to 3 days after beginning NSAID administration but may take up to 2 weeks to develop. If such signs develop the veterinarian should be notified immediately and the NSAID discontinued.

It is important to realize that many owners may give their pets aspirin, but do not consider this as a medication because it is easily obtained over the counter. It is critical to take a thorough medication history to prevent serious side effects from drug interactions. Aspirin is a nonselective COX inhibitor and may increase gastrointestinal side effects when given with other NSAIDs or COXIB class medications.[3]

Understanding When, What, and How

Understanding when a patient is a candidate for surgery, what procedure is recommended, and how it is performed comes with years of experience of the veterinary surgeon. For canine rehabilitation practitioners it is beneficial to be aware of certain factors regarding the patient's condition. For example if a patient has sustained a fracture several days before surgical repair, the injury will be more difficult to repair because of contracture of muscle, fibrosis, and early callus formation. This may make the initial rehabilitation period somewhat more challenging because of greater surgical trauma during fracture repair. It is also beneficial to know which fractures should be surgically repaired, and which may be managed successfully with external coaptation devices. Additionally knowledge of common orthopedic procedures is essential to the rehabilitation practitioner to fully understand postoperative considerations and the basic care of these cases.

For the neurologic patient with intervertebral disk disease or trauma, triage begins with the history. It is vital to collect information regarding the duration of clinical signs, the time elapsed since the patient became paretic/paralyzed, and any use of steroids or other antiinflammatory medication. After a patient has lost deep pain there is a 50% chance it will regain functional mobility if surgery is performed within 24 hours. In general every hour past 24 hours from the point when deep pain is lost decreases the prognosis for a full recovery exponentially.[4] It is good to remember that the loss of deep pain does not definitively preclude a dog from regaining motor function. However, dogs that do not recover deep pain, rarely regain conscious voluntary motor function and their ambulation is not normal. It is also important to remember that any dog that has intact deep pain in its affected limbs has the potential to recover function.

Postoperative Considerations

Postoperative Pain Management

Pain management is a priority in orthopedic or neurologic surgery. Multimodal analgesia has become the standard of care for postoperative pain control in veterinary medicine.

Many patients receive local or regional nerve blocks or epidurals before or immediately after surgery. It is extremely important to monitor the urinary status of patients after receiving epidurals. A high percentage of patients retain urine following epidural morphine (Duramorph) administration. There are three suggested mechanisms that may explain urinary retention following epidural morphine administration: (1) local detrusor inhibition through opiate receptors; (2) inhibitory action on the primary micturition center in the caudal brainstem; and (3) inhibition of parasympathetic nervous activity following binding of morphine with opiate receptors in the sacral spinal cord, resulting in detrusor muscle relaxation and increased bladder filling.[5,6] A 50/50 mixture of bupivacaine and Duramorph may be administered to provide more balanced analgesia.

Additional medications commonly used in multimodal analgesic protocols include NSAIDs such as deracoxib or carprofen; opioids such as hydromorphone, buprenorphine, or morphine; and other analgesics such as gabapentin, pregabalin, or amantadine. Physical modalities such as cryotherapy or transcutaneous electric nerve stimulation also may be used in postoperative pain management protocols. The veterinary staff must also be adept at monitoring and assessing pain and able to discern painful responses or behaviors in contrast to the dysphoria that can occur during anesthetic recovery.

Managing the Recumbent Patient

Some conditions require a lengthy recovery and animals may initially be recumbent. During this critical time, it is especially important to provide intensive nursing care to avoid serious complications.

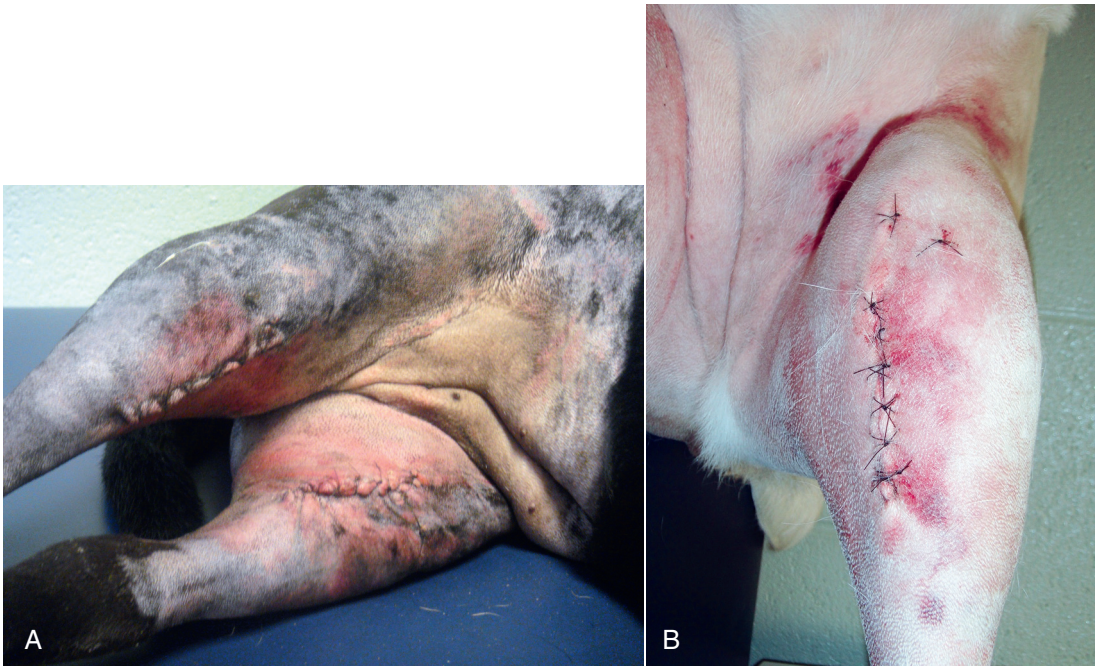


Figure 16-8 Incisions following surgery. **A,** One day postoperative bilateral TPLO incisions. Note that there is swelling and edema, resulting in constriction of the soft tissues by the sutures. There is also bruising. Bilateral procedures may be at greater risk for incisional complications. **B,** More normal one day postoperative TPLO incision.

Patients should be turned every 4 hours if they cannot turn themselves. They should be placed on a padded bed. An absorbent pad should be placed under the animal if incontinence is an issue. For patients that urinate frequently it may be necessary to have the animal on an elevated grate that will not allow urine to pool under the animal.

Animals that are recumbent for a very long time may be at risk for developing decubital ulcers. It may take as little as 1 hour of sustained pressure to develop a deep decubitus.[7] In particular, animals with thin skin and prominent bony protuberances, such as greyhounds and whippets, are especially prone to developing decubital ulcers. Prevention of ulcers is far superior to treatment of ulcers. Therefore the skin should be assessed each day for redness of the skin or other evidence of skin breakdown. If redness or irritation of skin is noted, a "doughnut" may be made to help distribute pressure away from the area.

Care of the Incision

The adept practitioner should be familiar with the characteristics and appearance of "normal" incisions, in order to properly identify questionable or compromised incisions (Figure 16-8). It is important to note the location of the incision(s), the number of incisions, number of individual sutures, and the type of incision closure(s). Effort should be made to ensure the incision(s) is clean and dry. Following surgical closure of the incision in the operating room the skin around the incision is cleaned with either warm sterile saline or hydrogen peroxide and allowed to dry. Although hydrogen peroxide may recruit free radicals and cause oxidative injury to tissue, the use of hydrogen peroxide to clean blood and other fluids from the skin around the wound has minimal effects on healing if not directly applied to the incision. For standard orthopedic procedures a nonadherent pad is applied with triple antibiotic ointment and affixed over the incision with wide adhesive tape (Figure 16-9). This temporary dressing serves to protect the incision and may be removed when the incision is sealed after 2 to 3 days if there is no drainage. Often the postoperative orthopedic patient's incision will have slight bloody or serosanguinous discharge the day of surgery and the day following surgery. This is considered to be normal and becomes less prominent as a fibrin seal is created approximately 24 to 72 hours[8] after surgery (Figure 16-10). When allowable a cold compress can be applied immediately after surgery for 15 to 20 minutes to the operative site to provide analgesia, and to decrease inflammation and edema formation.[9,10] The patient's incision should be monitored daily for any drainage or discharge, apposition of wound edges, excessive inflammation or swelling, and any missing staples or sutures (Figure 16-11). Before discharge the patient's incision should be evaluated and cleaned again if needed. The patient should not be discharged with an actively draining or open incision (Figure 16-12). Patients with edema in a single limb should have this limb elevated to help prevent swelling by lying on the opposite side and using a rolled towel or pillow to elevate the edematous limb if tolerated.



Figure 16-9 Placement of a protective dressing following surgery. **A,** Materials needed to apply a temporary dressing. **B,** A nonadherent contact dressing is placed on an adhesive bandage. Triple antibiotic ointment is applied to the pad. **C** and **D,** Application to the incision. **E,** Final placement of temporary protective dressing.



Figure 16-10 Bilateral incisions for luxating patella corrective surgery. **A,** 24 hours postoperatively. Note that the sutures are somewhat tight. **B,** 48 hours postoperatively. **C,** 72 hours postoperatively.

Cold Compresses

Cold compresses may be applied to the operative site immediately following surgery. Common practice is to affix a nonadherent sterile pad with adherent tape over the incision, and apply the compress directly on or around the temporary dressing. Studies of human patients have indicated that it is not effective to apply a cold compress through a thick padded bandage.[11] Thus cryotherapy should be performed at the time of bandage changes if a bandage must remain on the patient. When applying a cold compress, a barrier such as a nonadherent sterile pad or very thin towel may be placed between the skin and the cold compress to maintain patient comfort (Figure 16-13). It is important to use a compress appropriately sized for the patient. For example in a small dog with a distal radius and ulnar fracture that has been repaired with a bone plate, an extra-small to small cold compress should be used. Likewise for a mastiff with a femur fracture repair, a large to extra-large cold compress would be best. For more information on types of cold compresses, the reader is referred to the chapter on superficial thermal modalities.

Warm Compresses

It is important to remember that if the tissues are inflamed from disease, injury, or surgery, application of heat is contraindicated. Warm compresses may begin after the acute postoperative inflammatory phase has ceased, usually around 72 hours postoperatively or postinjury. The application of heat results in increased blood flow to the area, and possibly decreased muscle spasms. When applying a warm compress when drainage is present, always provide a barrier, such as a nonadherent sterile pad or an absorbent

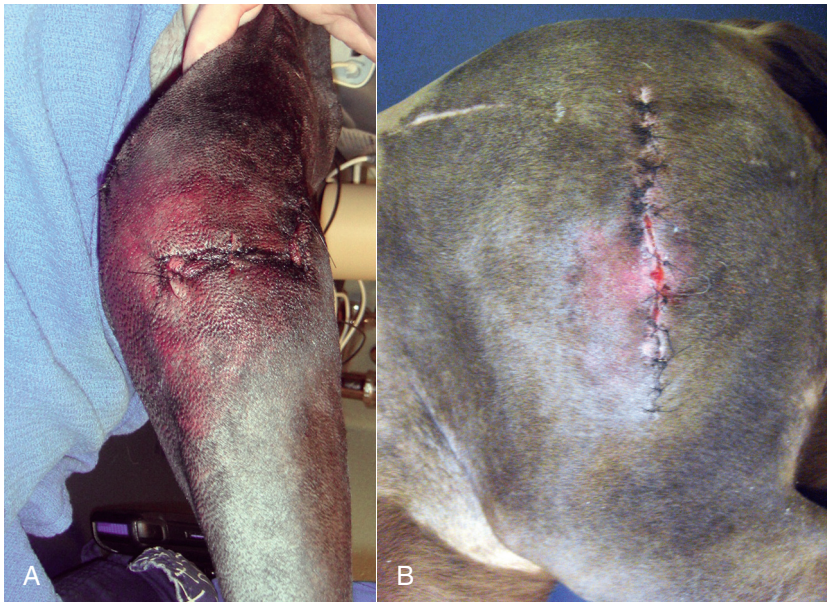


Figure 16-11 **A,** Postoperative stifle wound repair with marked increase in erythema and edema. **B,** Postoperative femur fracture repair with a compromised incision. Note area of missing sutures.



Figure 16-12 Postoperative TPLO surgery with seriously compromised incision, most likely secondary to self-trauma and infection. Note drainage and TPLO plate can be visualized through the open incision. Immediate wound management is warranted.



Figure 16-13 Crushed ice and water in plastic bag. Wrap in cold towel to protect skin for cryotherapy application.

pad with a plastic backing, between the skin and the compress to reduce disease transmission. It is important to always test the compress before application on the patient to be certain that it is not too hot. Several commercially made compresses are available. For more information regarding types of warm compresses, see Chapter 18, Superficial Thermal Modalities.

Massage

Soft tissue massage can be beneficial in reducing postoperative edema. The degree of limb edema can vary following orthopedic surgery depending on the size of the patient, the procedure performed and the severity of the injury. Ideally massage is performed following superficial thermal modalities and before assisted ambulation or controlled therapeutic exercises. It is important to begin the massage distally and work proximally to increase lymphatic and venous flow and thereby decrease edema (Figure 16-14). The edges of the incision should be avoided. The degree of pressure applied by the hand and fingers should be gentle but firm. Patients that have compromised incisions or skin grafts are not candidates for soft tissue massage. The reader is referring to the chapter on massage for more information regarding massage techniques.



Figure 16-14 Massage, specifically effleurage, performed distally to proximally on the hind limbs of a dog with pelvic fractures repaired surgically.

Assisted Standing

Standing exercises focus on strengthening shoulder, elbow, hip and stifle extensors and reeducating muscles needed for balance and proprioception. Several approaches can be used to facilitate standing. Particularly when a patient is large, assistive devices such as slings, physio rolls, mobility carts, wheelchairs, or lifts may be used to maintain a patient in a standing position (Figure 16-15). In smaller patients, the therapist can manually support the patient to maintain a standing position (Figure 16-16, *A*). Two common variations of this exercise are static standing and sit-to-stand exercises. Static standing is typically performed three to five times daily for 2 to 5 minutes or until the patient fatigues. It is useful to support the patient under the ischiatic tuberosity and on the cranial aspect of the stifles to facilitate a normal standing angle of the hind limbs (Figure 16-16, *B*). Conversely one often will need two people to maintain limb position in a tetraparetic patient even with abdominal support from a physio roll or sling system. It is important to maintain normal foot position at all times during this exercise (prevent knuckling and encourage weight bearing). It is recommended that one should perform standing exercises at the time of eating or drinking, because standing often improves while the patient is distracted by the act of eating and drinking.

Assisted Ambulation

Assisted ambulation is paramount with orthopedic and neurologic patients. It is necessary to assist postoperative patients getting into and out of cages, and to help them up from a recumbent position, as well as while ambulating. The caregiver provides assistance when needed and also prevents the patient from falling or overloading tissues and surgical repairs (Figure 16-17). Caution should be taken if the patient is excessively painful or ill tempered. To best help these patients begin early ambulation, the patient must trust the caregiver. Therefore some time must be invested in allowing the patient to become accustomed to the assistive device and the procedures involved in placement and assisting ambulation. Numerous assisted ambulation devices such as slings and harnesses are commercially available. The practitioner should be familiar with the devices, how they work, and how to graduate a patient from one device to a more challenging device. For more on this topic see Chapter 17, Assistive Devices.

Everyday Activities as Rehabilitation Exercises

Encourage owners to make everyday activities a rehabilitation exercise in disguise. For example, while a postoperative patient is standing to eat from a food bowl, an opportunity is presented to do some standing and weight-shifting exercises. Not only will the dog probably be more interested in the food than the standing or weight-shifting exercise, but the behavior is tolerated and positively reinforced with the food (Figure 16-18).

Nursing Care for the Orthopedic Patient

Postoperative Orthopedic Infections

Infection control measures begin with hospital admission and continue in the prep room and operating room. The cages and runs that house the patients should be properly cleaned between patients. Chlorhexidine or povidone iodine surgical scrub is used to cleanse the skin of patients. The presence of pyoderma, a skin infection, is a clinician-dependent precaution for surgery. The client should be informed if pyoderma is present before the surgical procedure and any risks should be thoroughly explained. The surgery proceeds depending on the surgeon's and client's decisions. There is some increased risk of postoperative infection if pyoderma exists, especially if implants are used. If surgery is postponed the procedure is performed when the patient is clear of infection after completing antibiotic therapy.

Other factors can also increase infection risk, such as prolonged operative and anesthesia times, number of personnel in the operating room, prolonged hospitalization, and the experience level of personnel. The level of microbes in the air of the operating room is directly proportional to the number of people moving in the room; thus *Staphylococcus* infections can be reduced if traffic is limited in the operating room during surgical procedures.[12] Effort should be made to reduce operative times and limit foot traffic in the operating room.

Perioperative antibiotic therapy is indicated in contaminated cases (open surgeries), patients with severe soft



Figure 16-15 Methods of manual assistance for patients. **A,** Assisted standing exercise in a critically ill patient provided via Hoyer lift, slings and therapy balls. **B,** Assisted standing over therapy roll by a dog recovering from a cerebellar infarction. **C,** Note manual placement of the left hind foot during assisted standing exercises. **D,** Note manual placement of the left forefoot during assisted standing exercises.



Figure 16-16 **A,** Assisted manual standing by a dachshund following hemilaminectomy. **B,** Another manually assisted standing technique is to support the patient under the ischiatic tuberosity and on the cranial aspect of the stifles to facilitate a normal standing angle of the hind limbs.



Figure 16-17 Assisted standing performed using a harness and black elastic therapy band.



Figure 16-18 Assisted standing exercise over a therapy roll, performed while eating to encourage standing behavior and provide positive reinforcement.

tissue swelling or bruising as a result of trauma, or in cases with lengthy operative times (longer than 2 hours).[13] Cefazolin is commonly used at a dose of 22 mg/kg given 20 minutes before surgery, and every 2 to 4 hours thereafter throughout the surgical procedure. The most common types of infectious agents seen in postoperative surgical wounds are *Staphylococcus* species and *Escherichia coli*.[13]

A factor contributing to antibiotic resistance is the overuse of antimicrobial therapy. Again, although is it the responsibility of the veterinarian to prescribe antibiotics, the adept therapist should know when and why antibiotics are administered and make the veterinarian aware of which antibiotics were prescribed, for what purpose, and the time period of treatment.

Infection can be a costly complication of orthopedic surgery and affect the ability of the patient to undergo physical rehabilitation. The patient in Figure 16-12 had a tibial plateau leveling osteotomy (TPLO) that developed a postoperative infection. Wound management was performed daily under sedation and consisted of flushing, wound debridement, and packing it with antimicrobial gauze. A drain was placed and the wound was surgically closed after 2 weeks.

In human medicine the incidence of methicillin-resistant *Staphylococcus aureus* (MRSA) infections has increased. The term *methicillin-resistant* was originally used in 1961

after a *Staphylococcus aureus* infection in the United Kingdom was found to be resistant to methicillin.[14] MRSA is a gram-positive bacterium that is resistant to methicillin and many other β-lactam antimicrobials; β-lactam antimicrobials include penicillins and cephalosporins. The major vector for transmission is unwashed hands, although MRSA can live on the patient's skin, in the environment, and on surfaces for extended periods of time. Approximately 25% to 40% of humans are carriers of the organism, mostly in the nasal passages.[15] The organism lives harmoniously with the host, but an immunocompromised individual or skin wounds can allow infection to occur. These infections can have dire consequences in humans, but so far do not appear to be life threatening in canine patients. Morbidity generally consists of incisional dehiscence, septic arthritis, and chronically draining wounds necessitating implant removal.

Methicillin-resistant *Staphylococcus aureus* was considered solely a human pathogen for many years until it was first isolated in a dairy cow in 1972.[16] Since then the veterinary medical community has seen an increase in the incidence of postoperative infections. Specifically infections caused by MRSA and more commonly, methicillin-resistant *Staphylococcus pseudintermedius* (MRSI), have been identified following both soft tissue and orthopedic surgeries, as well as cases of pyoderma. Although a standard method for typing and naming MRSA strains has yet to be developed, many methods of nomenclature are currently in use.[15] MRSA infections in small animals are predominated by the strain USA 100, whereas MRSA infections in large animals are predominately strain USA 500.[17] Although it was previously thought that MRSA could only be transmitted from human to animal, emerging evidence indicates that MRSA can be transmitted in both directions, from human to animal and from animal to human. After an animal becomes colonized it can act as a reservoir, transmitting the infection to humans or other animals.[17] Although identification of carriers is paramount to prevention of MRSA and MRSI transmission, routine screening of animals is not yet practiced; therefore the possibility remains that colonized animals will go undetected in veterinary settings.[18] Research is needed to determine adequate treatment regimens for MRSA- and MRSI-infected animals, control of MRSA and MRSI transmission, and environmental sanitation. In the interim practicing proper hand hygiene and sanitization is integral to the prevention of MRSA and MRSI transmission. In addition, disposable gowns, gloves, and masks should be worn to change bandages and wound dressings of afflicted patients. Signs of MRSA and MRSI in dogs include acute edema around the incision or wound, excessive scratching or licking, pimple-like lesions on the skin with larger "bumps" in the area, and drainage from the wounds.

Recognizing Signs of Infection

Monitoring for infection begins on day 1 postoperatively, with most infections occurring within 7 days after surgery. The classic signs of inflammation are heat, redness, swelling, pain, and loss of function; the cardinal signs of infection are often the same but they persist beyond the acute inflammatory period and may be accompanied by discharge from the incision, fever, lethargy, and poor to no appetite. However, local abnormalities are more commonly observed than signs of systemic illness with MRSI infections in orthopedic procedures. Often the patient is discharged, and the client calls with concerns a few days to 2 weeks after surgery. The most common clinical history is a sudden decrease in use of the surgical limb, or the limb may develop significant edema. The surgical limb may also have a seroma or active serosanguinous or purulent drainage from the surgical site. A seroma is a collection of serum in the body, producing a fluctuant to firm swelling. If clinical signs of a postoperative infection are noted by the client, the patient should be seen by the veterinarian as soon as possible.

A sample is obtained from the affected area and submitted for cytology, culture, and sensitivity. To aseptically drain a seroma, the site should be clipped and cleaned thoroughly with an aseptic scrub such as chlorhexidine. Wearing exam gloves or sterile gloves, a butterfly catheter or needle attached to a syringe is inserted into the seroma. The fluid is aspirated and collected into the proper tubes or material is placed on a culturette. A seroma does not necessarily indicate that an infection is present. The diagnosis of infection is based on cytology or culture results.

When patients have a postoperative infection, the area is often inflamed, edematous, warm to the touch, and painful on palpation. Postoperative infections may involve the joint, periarticular soft tissues, or the site of an implant such as a plate or pin. In any case, radiographs are indicated to help determine the extent of the infection. While the patient is sedated an arthrocentesis may be performed to determine if intraarticular infection is present. Septic arthritis is associated with greater postoperative morbidity and less favorable long-term outcomes.

To aseptically perform an arthrocentesis the site should be clipped and cleaned thoroughly with an aseptic scrub such as chlorhexidine. Wearing sterile gloves, a 20- or 22-gauge 1½-inch needle is inserted into the joint, usually just medial to the patellar tendon. In the event that the joint is not infected, caution should be used to avoid affected skin and subcutaneous tissues while obtaining joint fluid to avoid carrying infection into the joint. Fluid is allowed to drip from the needle into a sterile collection vial. If fluid does not drain a syringe may be attached and fluid gently aspirated and collected into the proper tubes or culturettes. Normal joint fluid is clear and viscous. Abnormal joint fluid may be cloudy and red-tinged, indicating the presence



Figure 16-19 Postoperative infected TPLO after several days of daily aggressive wound management. The sutures are for securing a tie-over dressing.

of blood; or cloudy white or yellow, indicating the presence of purulent material.

The fluid should be cultured for both aerobic and anaerobic bacteria. It generally takes 72 hours to obtain culture and antibiotic susceptibility profiles of organism(s). In this case, the client is instructed to continue on an empirical antibiotic or perhaps no antibiotic, until the results are available. During this time it is imperative that the patient be prevented from licking the wound. After culture and susceptibility results are available the patient is placed on the appropriate antibiotic therapy for 3 to 6 weeks. In serious cases the patient may need to return every other day or daily or be hospitalized for bandage changes and wound debridement (Figure 16-19).

In severe cases in which an implant may be harboring an infection, the implant may need to be removed after bony healing is completed. In such cases continued antibiotic therapy is often required. Radiographs are taken to confirm bony union and the implant is removed and infected tissue is resected and cultured. Following implant removal, postoperative care is the same as after the initial surgical repair.

External Coaptation Devices: Bandages, Splints, and Casts

The roles of coaptation devices are threefold; they can provide temporary support before surgical intervention, secondary support after surgical intervention, or primary support instead of surgical intervention. The primary functions of a coaptation device include protection, absorption, compression, and stabilization.[19] The device should protect the incision from bacterial contamination and from self-mutilation. It should be absorptive if a wound is present, to decrease accumulation of exudates. The contact layer should be appropriate for any wounds that may be present.

Coaptation devices should be well padded and comfortable, well tolerated by the patient, provide support and protection to the injured limb, provide compression of the soft tissues to aid in edema reduction, provide stabilization of the limb, should not cause morbidity, and be cost effective.

Splints and Casts

Basic coaptation guidelines should be followed. Splints or casts may be used to stabilize fractures after reduction. It is important to immobilize the joint above and below the fracture; otherwise the cast or splint may act as a fulcrum at the fracture site. Thus only fractures distal to the elbow and stifle may effectively be treated with external coaptation.

Splints and casts may be used with variable success in stabilization of distal limb fractures, such as the radius and ulna. Case selection depends on the type of fracture, and the signalment and demeanor of the patient. Malalignment of the fracture may result in limb deformities, gait abnormalities, and osteoarthritis.

When applying coaptation devices, the limb should be maintained in a neutral standing position with joints placed at a functional standing angle. Immobilizing the hind limb in an extended position and allowing weight bearing may produce cartilage changes similar to those of osteoarthritis,[20] and joint stiffness as a result of immobilization in extension may result in a poorly functional limb.

Components of a Bandage

The primary contact layer is a nonadherent sterile dressing (e.g., a Telfa pad, Covidien; Mansfield, MA). This layer is in direct contact with the incision, and a sterile triple antibiotic ointment can be applied between the incision and primary contact layer. The secondary layer of the bandage is the padding or absorptive layer, such as cotton cast padding. The tertiary layer of the bandage is the protective layer, such as Vet Wrap or Elastikon (Johnson & Johnson, Langhorne, PA) (Figure 16-20).

Principles of Bandage Application

There are several principles of bandage application that should be followed. The limb and joints should be placed in a neutral standing position for bandage application. Non–stretch adhesive tape should never be placed circumferentially around the limb. Doing so could lead to serious complications including compromised limb circulation, ischemia, and limb necrosis. Tape stirrups should be applied to either the medial and lateral or cranial and caudal aspects of the distal limb. These tape stirrups aid in preventing the bandage from sliding distally off the limb.

The contact layer, usually a nonadherent sterile dressing, is applied to the incision or wound. A doughnut can be custom made for a patient and is beneficial when placed



Figure 16-20 Materials for a soft padded bandage (modified Robert Jones bandage). **A,** Materials needed to start the bandage and materials for the contact layer. **B** and **C,** Materials for the secondary layer of bandage (padding and compression). **D,** Materials for the tertiary or outer layer of bandage.

over a pressure point, such as the tuber calcis, olecranon, or accessory carpal bone, to prevent pressure sores. The doughnut can be made from cotton cast padding or roll cotton. After folding several layers a hole may be cut in the center so that the doughnut fits over the pressure point. The purpose of the doughnut is to distribute the forces of bandage compression over a greater surface area rather than just over the pressure point. Just adding more cotton over a pressure point without a doughnut may actually increase the pressure over the point.

When beginning application of the bandage, cast padding or roll cotton is placed circumferentially around the limb, always rolling the material from distal to proximal (Figure 16-21, *A*). The tips of the middle digits should be barely showing from the end of the bandage. This will allow veterinary care providers and owners to check for coolness and swelling of the digits, which might indicate that the bandage is too tight. When applying material it is helpful to roll the material on from lateral to medial, overlapping by 50% and distributing pressure uniformly throughout all layers. Cast padding is often textured and the textured markings flatten when the material is pulled taut. If too much tension is applied the material will tear. If the secondary layer of cast padding is too loose the bandage may slip distally and irritate the tissues under the bandage. A compression layer is then applied over the cotton, using gauze material applied under tension while wrapping the gauze circumferentially from distal to proximal, and applying it in such a fashion as to cause slight internal rotation of the limb (Figure 16-21, *B*). The gauze should not be placed beyond the cotton padding, which could cause vascular compromise. It is very easy to apply the rolled gauze too tight, so careful application is necessary. When working on the proximal aspects of the bandage, it might be helpful to have the limb abducted to allow the cast padding, rolled gauze, and Vet Wrap to be applied as proximally as possible. When applying the tertiary outer layer the material should be loosely applied by either unwrapping it and then carefully re-wrapping it to loosen the elasticity of the material and ensure the material is



Figure 16-21 Placement of a soft padded (modified Robert Jones) bandage. **A,** Application of rolled cotton. **B,** Application of rolled gauze to provide compression. **C** and **D,** Application of the outer layer. Note: all layers are applied in a distal to proximal direction.

not applied too tight, or unroll some of the material, allow the material to undergo elastic recoil, and then roll it on the limb as it is applied in a distal to proximal direction (Figure 16-21, *C* and *D*). Some practitioners apply a strip of Elastikon at the most proximal aspect of the bandage to the skin to help prevent bandage slipping. Care must be taken not to apply adhesive tape over thin-skinned areas, such as the medial thigh, to avoid irritation of the skin by the adhesive.

Soft-Padded Bandages

The Robert Jones bandage is one of the most frequently applied coaptation bandages in veterinary medicine. It provides excellent temporary support for an injured limb before or after surgery. Overall the Robert Jones bandage is a safe bandage and few complications are seen. The main layer is rolled cotton, which yields the final diameter of the bandage, which is 40% to 50% larger than the original limb. The modified Robert Jones bandage is similar to

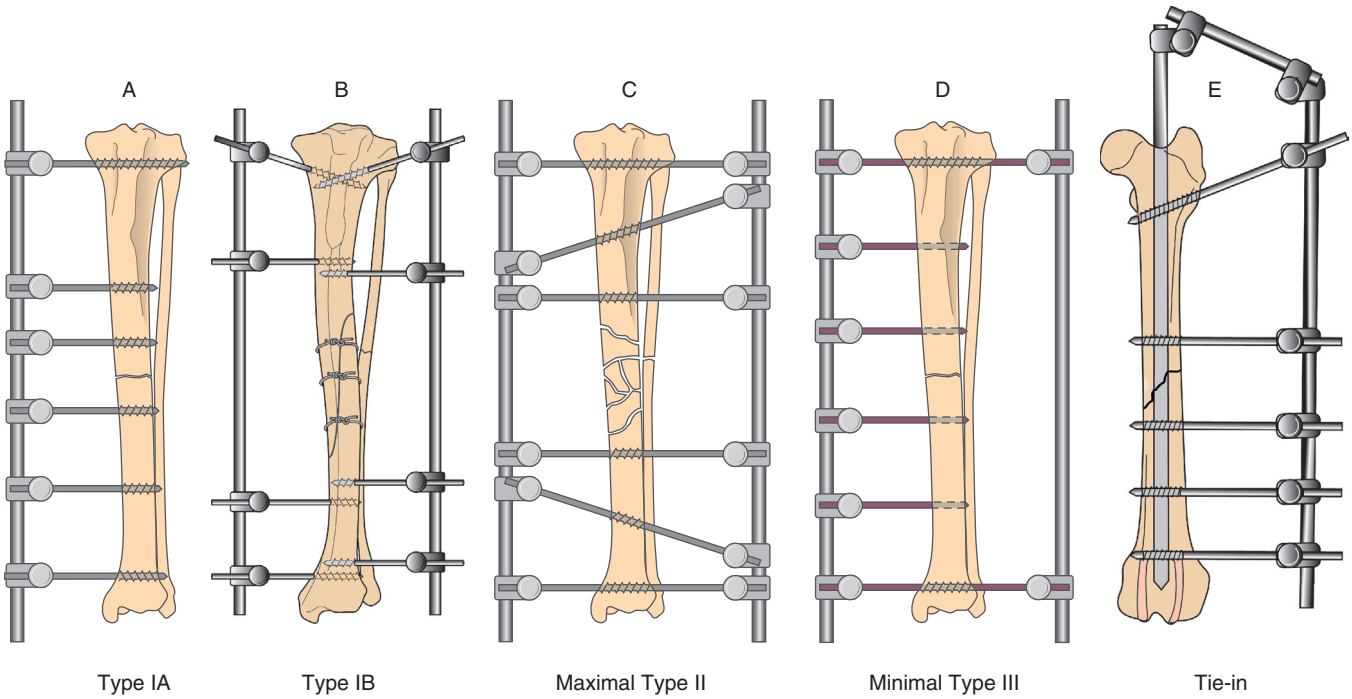


Figure 16-22 Classification of external fixator frame configurations. **A,** Type IA. One plane, unilateral. **B,** Type IB. Two plane, unilateral. **C,** Type II. One plane, bilateral. **D,** Type III. Two plane, bilateral. **E,** Tie-in configuration. (From Fossum, TW: *Small Animal Surgery*, ed. 4, St Louis, 2013, Mosby. Second and fourth images modified from Johnson AL, Dunning D: *Atlas of orthopedic surgical procedures of the dog and cat*, St. Louis, 2005, Saunders in Fossum, TW: *Small Animal Surgery,* ed. 4, St Louis, 2013, Mosby.)

the Robert Jones, except less cotton layer is applied, thus significantly decreasing the size of the bandage. The modified Robert Jones bandage provides the same advantages as the Robert Jones bandage, yet it is more economical and tolerable for the patient. The modified Robert Jones bandage is the most commonly applied bandage following surgeries of the elbow or stifle, or distal to these joints. However, neither the Robert Jones bandage nor the modified Robert Jones bandage provides adequate stabilization for fractures. Bandages should be changed daily up to weekly as directed by the veterinary surgeon.

Bandaging an External Fixator

External fixators are used to repair fractures or joint luxations and may be applied in various configurations. They can counteract the forces of rotation, bending, shearing, and distraction. External fixator configurations can be designed to meet the stabilization needs of a fracture and can be subsequently modified or destabilized throughout the healing process.[21] Pin factors, such as pin type, size, number, location and length, and frame configuration, affect the stiffness of the fixator and its ability to resist the forces associated with weight bearing, such as bending, axial loading, and rotation.

There are three main types of linear external fixators (Figure 16-22). The fixator configurations are named for the number of planes occupied by the fixator and the number of sides of the limb from which the fixator protrudes.

- Type 1A is unilateral-uniplanar; usually applied to the medial aspect of the radius or tibia and to the lateral humerus or femur.
- Type 1B is unilateral-biplanar; usually applied to the radius or tibia.
- Type 2 is bilateral-uniplanar; applied only to the radius or tibia, it cannot be applied to the humerus or femur because of the close proximity to the body on the medial side.
- Type 3 is bilateral-biplanar; applied only to the radius or tibia, it cannot be applied to the humerus or femur because of the close proximity to the body on the medial side.

The number of pins and planes directly affect the strength and stiffness of the external fixator. When an external fixator is used with an IM pin, it is called a tie-in configuration, and results in greater strength and stiffness of the fixation.

Pins may be placed in different ways. Half pins penetrate both cortices of the bone but only one skin surface. Full pins penetrate both cortices and both skin surfaces. Clamps and connecting rods adjoin fixator pins and connecting bars to each other.

Circular or Ilizarov ring external fixators are multiplanar and multilateral, facilitate distraction of long bones, and may be used to correct angular limb deformities.

Special wires or small pins are placed through the bone and connected to rings with clamps. Distraction osteogenesis induces new bone formation between two bone ends (after an osteotomy is performed) that are distracted over time. The rate of distraction osteogenesis is 1 mm per day and the rhythm is 0.25 mm four times a day.[21]

External fixators provide a bandaging challenge. Although there are many different methods to bandage an external fixator, certain principles should be followed. Each pin-skin interface should be covered with a primary contact layer. Often a sterile nonadherent pad is cut to accommodate the pin and trimmed to an appropriate smaller size. Sterile triple antibiotic ointment is placed on each pad before applying the contact layer to the patient (Figure 16-23, *A*). The pads can be held in place with recycled dry scrub sponges placed around the pins, connecting rods, and linkage devices (Figure 16-23, *B*). The sponges also provide padding for the patient and the apparatus. All sharp pins protruding from the apparatus should be covered to protect the patient. The ability to improvise is important; items used to cover pin points include rubber caps, injection caps, and sponges folded over the pin point and securely taped (Figure 16-23, *C*). Next the apparatus is wrapped with cast padding, roll gauze, and Vet Wrap, noting that the limb itself is *not* incorporated in the bandage (Figure 16-23, *D* through *I*) unless edema is present or wounds must be covered. In these cases, the bandage is placed similar to a modified Robert Jones bandage, incorporating the external fixator as previously described. An adequate but minimum amount of cast padding should be applied because if the bandage is too large it may slip off.

External fixator bandages should be rechecked weekly. The bandage is removed, the apparatus is assessed for loosening or drainage, and radiographs are taken at the veterinarian's discretion. While the patient is still sedated from the radiographs, the pin tracts are thoroughly cleaned

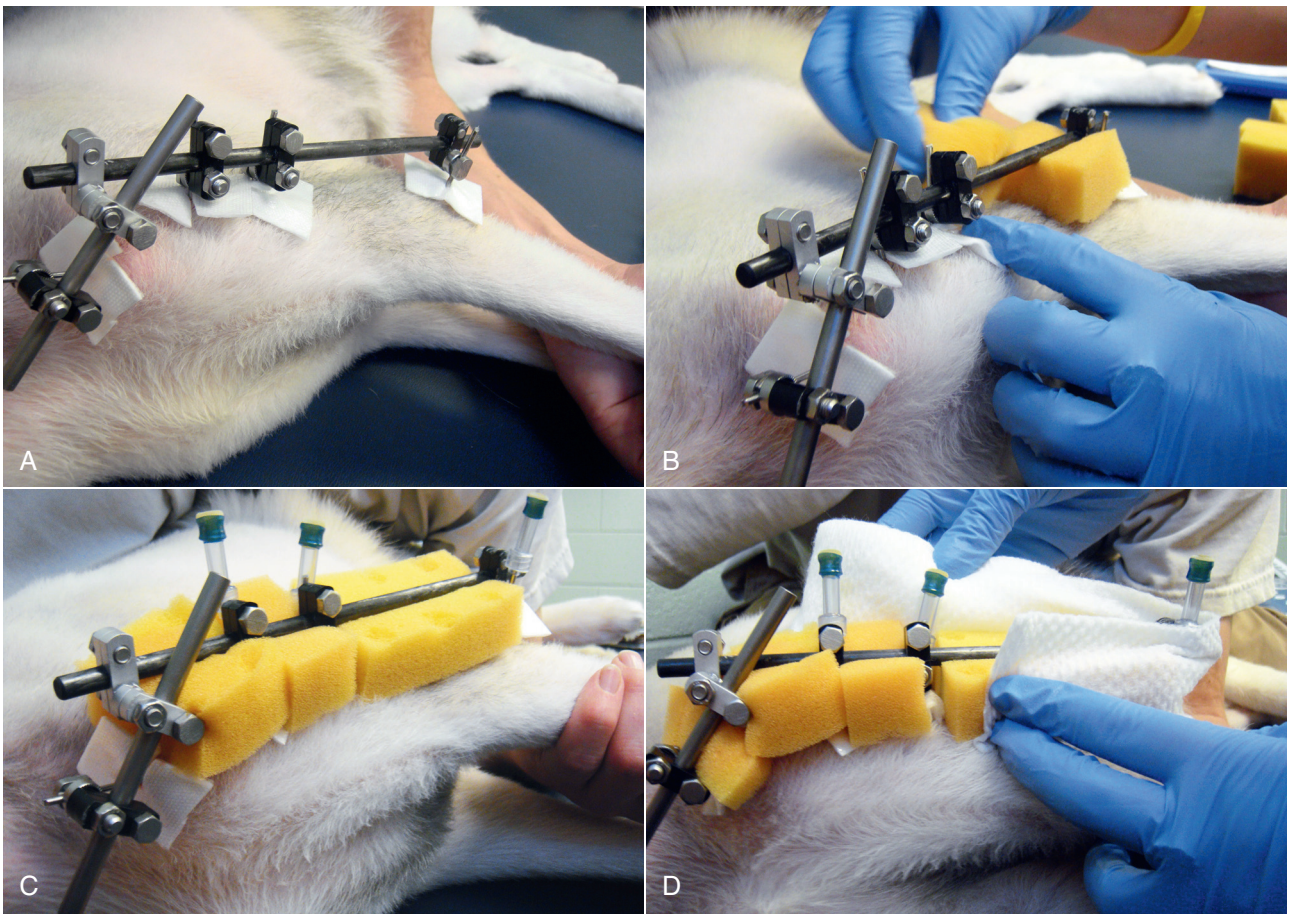


Figure 16-23 Bandage placement over an external skeletal fixator. **A,** Nonadherent pads cut to accommodate external fixator pins for treatment of a fracture of the humerus. **B,** Recycled scrub sponges, cleaned and dried, can be used to hold the nonadherent pads in place and provide padding. **C,** Injection caps can be used to cover pin points to protect the patient and environment. **D** and **E,** Cotton cast padding is applied to the apparatus.

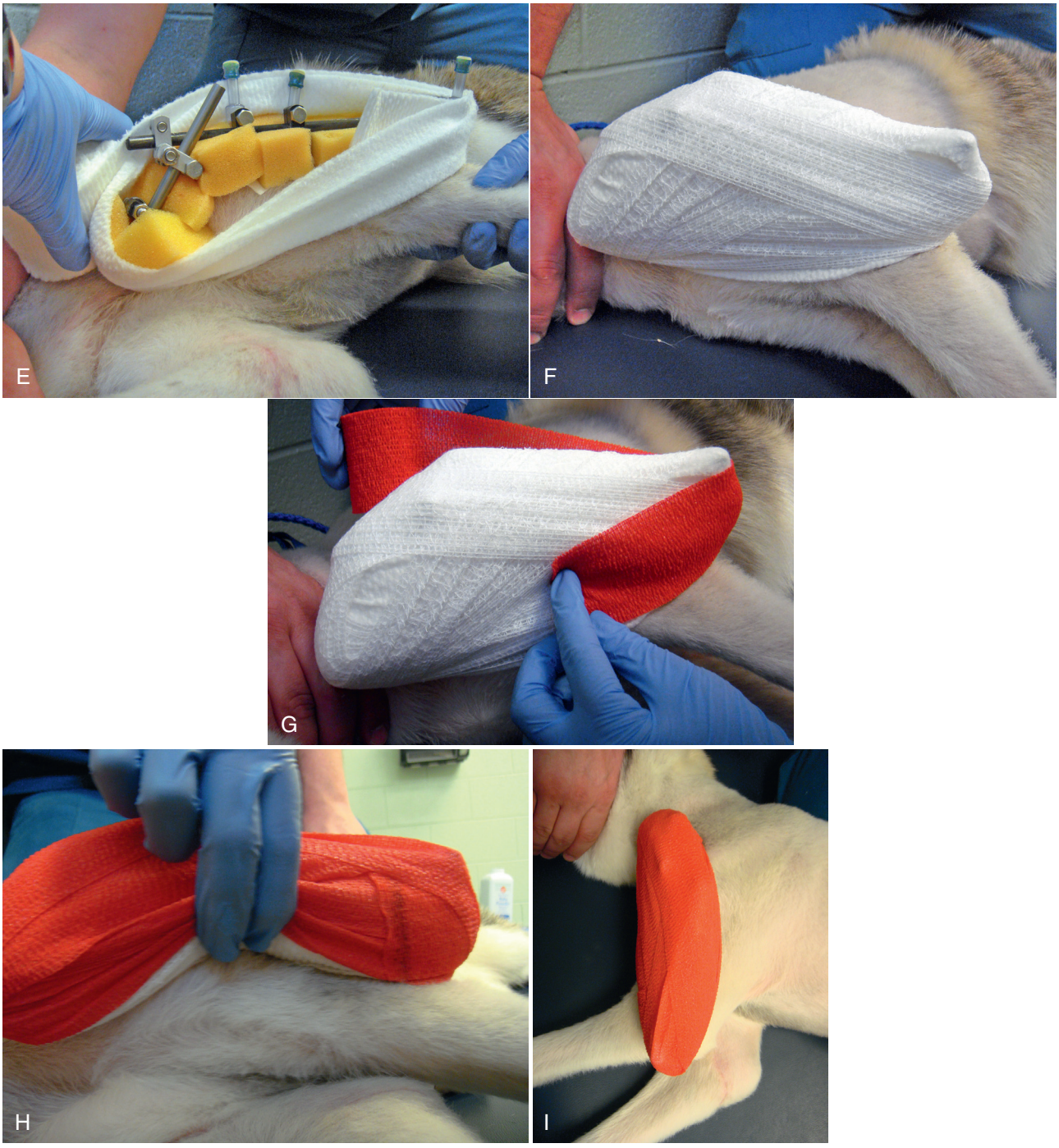


Figure 16-23, cont'd **F,** Roll gauze is applied in a similar manner. **G,** An outer wrap is applied in a similar manner. **H,** Note that the limb is not included in the bandage, unless edema is present or wounds must be covered. **I,** Finished product: bandage placed over an external skeletal fixator of the humerus.

and supplies are prepared for bandage change (Figure 16-24). Using a cotton ball or gauze sponge and chlorhexidine scrub, gently scrub around the pin tracts to remove dried exudate; sterile cotton tipped applicators also may be used. Gently rinse with warm saline on a cotton ball, gauze, or cotton tipped applicators. This process is repeated for each pin tract until all are free of dried exudates (Figure 16-25). When accessible it may be necessary to scrub and rinse portions of the limb. The limb is allowed to air dry or a hair dryer placed on the cool and low setting can be used to expedite the drying process as tolerated by the patient. Depending on radiographic healing the veterinarian may choose to remove some pins, exchange connecting rods, or remove the device entirely. Removing the fixator



Figure 16-24 Wound management and bandaging materials needed for bandage change of an external fixator bandage of the humerus.

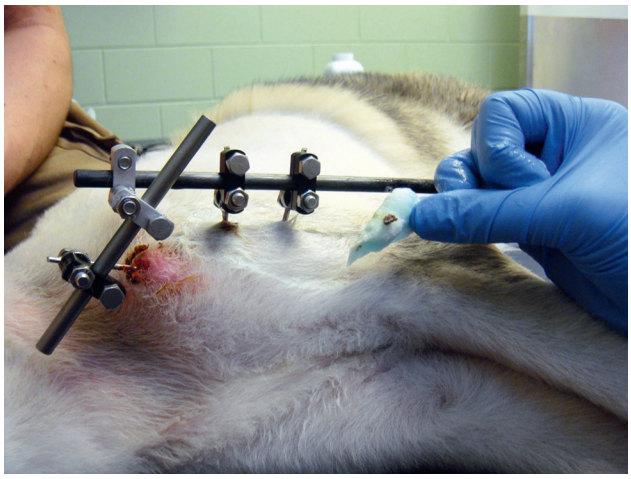


Figure 16-25 Pin tracts are cleaned with dilute chlorhexidine scrub and warm saline to remove exudate.

in stages gradually reduces the stability of the fixator and increases loading of the bone, which helps to stimulate callus development and maturation. The external fixator is rebandaged at each recheck or adjustment. The process of weekly rechecks continues as directed by the veterinarian until the injury has healed and the apparatus can be removed.

Pin loosening is the most common postoperative complication of external fixators. Factors that contribute to pin loosening are excessive micromotion, damage to the bone at the time of insertion, and fatigue failure of the bone cortex.[22] Pin tract infections can also occur and result in increasing purulent discharge from the pin-skin interface, reduced limb use, and pain with palpation over the site. In addition, delayed fracture healing, joint stiffness, and iatrogenic fracture are other complications that can occur.

Splints

A splint is essentially a bandage with incorporation of a stiff supportive device. Splints can be made from various materials. Commercial splints are available, such as spoon splints, Mason Metasplints, and Quick Splints (Jorgensen Laboratories, Loveland, CO). Some splints can be custom made using aluminum rods or casting material. Finding the appropriate size of commercially available splints for a particular patient can present a challenge. It is important that the splint fit properly. Mason Metasplints and Spoon Splints should cradle the caudal aspect of the limb and not extend proximal to the elbow or tarsus. Quick Splints should fit properly at every joint angle. It is important the splint is applied between layers of the bandage, most commonly after the compression layer has been applied.

Spica splints are used to immobilize all joints of the forelimb or hind limb, but are more commonly applied to the forelimb. The Spica splint can be applied in cases of humeral fracture repair, and closed or open reduction of an elbow luxation. The Spica splint envelops the affected limb and the torso of the patient. The limb is held in a functional standing position. After cast padding has been applied, the splint material, such as premeasured and prebent rods or fiberglass cast material, is applied from the distal digits over the dorsum to the proximal scapula of the contralateral limb. Roll gauze is then applied followed by an outer wrap. Monitoring the splint after application is important because Spica splints can cause abrasions in the axillary area.

Casts

Casts are a conservative method to stabilize fractures in young patients after closed reduction. Common fractures include simple fractures of the radius and ulna or fractures of the radius with the ulna intact. Similarly simple fractures of the tibia and fibula or fractures of the tibia with the fibula intact may be managed with a cast.

Cast application involves making sure the leg is clean and dry before application. Long hair should be clipped to a shorter length before cast placement, but not surgically clipped or shaved. To maintain patient comfort during reduction of the fracture and application of the cast, the patient is lightly anesthetized or heavily sedated with adequate analgesics. Tape stirrups are applied to either the medial and lateral or cranial and caudal aspects of the distal limb. An appropriate sized stockinette is applied to the leg. Traction is then placed on the limb to aid in fracture reduction. The fracture site may be palpated and manipulated to aid in reduction. It is important that the limb is not placed such that varus or valgus deformation of the limb is present. To avoid these deformations the limb may be placed so that the limb is hung from the ceiling during fracture reduction to make alignment of the limb easier.

Circular pads or doughnuts are applied over bony prominences. Cast padding is then applied, moving in a spiral



Figure 16-26 To maintain coverage and protection of a bandage, commercially made bandage covers can be used.



Figure 16-27 The Velpeau sling maintains the carpus, elbow and shoulder in a flexed, non–weight-bearing position.

pattern from distal to proximal, overlapping by 50%, with up to two to four layers applied. Fiberglass cast material is immersed in tepid water (70-75° F). The warmer the water, the faster the cast will harden. When the cast material is immersed bubbles will be released. After bubbling has ceased (10-15 seconds), the material can be removed from the water and shaken, but not squeezed, to remove excess water. A second person maintains the limb in traction with the fracture reduced while the cast is applied. After making a final assessment of the limb for proper alignment, the cast material is applied in a distal to proximal spiral pattern, overlapping by 50%. After the first layer is applied the tape stirrups and stockinette are turned up to incorporate them into the cast. Continue to apply cast material so that two to four layers are applied. Gently laminate the layers together and allow for adequate curing time. The cast material undergoes an exothermic reaction that creates heat during the curing process.

A saw may be used to cut the cast on the medial and lateral aspects of the limb after curing to bivalve the cast. When cutting the cast material with the saw, it is important to hold the saw stationary as it cuts. Moving the saw back and forth while cutting may result in cutting the patient's skin. Bivalving of the cast facilitates regular rechecks and assessment of the limb underneath. The patient may weight bear on a casted limb as early as 20 minutes after application is complete when using fiberglass casting material.

Maintenance of the External Coaptation Device

It is very important to keep all bandages, splints, slings, and casts clean, dry and intact. The external coaptation device must be protected from adverse weather. Plastic bags, recycled and dried fluid bags, or commercially made bandage covers (e.g., Medipaw, MediVet Products, Fogelsville, PA) may be used to cover the distal end of a bandage, splint, sling, or cast when walking outside (Figure 16-26). The patient should be housed inside while wearing an external coaptation device and the plastic covering should be removed when inside to prevent moisture buildup inside the bandage. Bandages should be checked several times daily for swollen toes, slipping of the bandage, moisture, soiling, drainage, or foul odor. The external coaptation device should be changed immediately if any of these is present, or if the device is damaged or loosened. If the bandage has been well tolerated and the patient begins to lick or chew at the bandage or toes, the bandage should be changed because there is generally a problem under the bandage that should be addressed as soon as possible, such as a wound infection, moisture that is irritating the skin, or wound dehiscence.

Complications of Bandaging

The complications of bandaging include pressure sores and ischemic injury, infection, damage to the surrounding soft tissues, neuropraxia, contracture, malunion, and angular deformities. The complications from a bandage can be serious. Therefore proper bandage application and aftercare cannot be overstated.

Slings

A sling is designed to prevent the patient from weight bearing on the affected limb. A Velpeau sling maintains the carpus, elbow, and shoulder in a flexed, non–weight-bearing position (Figure 16-27). The Velpeau sling can be applied in cases of scapular body fracture, bicipital tenosynovitis, and medial shoulder luxation. To apply a Velpeau sling: Flex the shoulder and elbow while adducting the limb against the body; start by applying two or three layers of cast padding around the patient's torso and affected forelimb, incorporating the forelimb such that it is secured to the thoracic wall. Some layers should go in a cranial to caudal direction around the limb to prevent slippage of the incorporated limb. Apply rolled gauze in the same manner to provide mild compression. Finally apply the outer wrap in the same manner to provide support.

An Ehmer sling maintains the hock, stifle, and hip in flexion, with internal rotation of the hip (Figure 16-28). The Ehmer is commonly used following closed or open

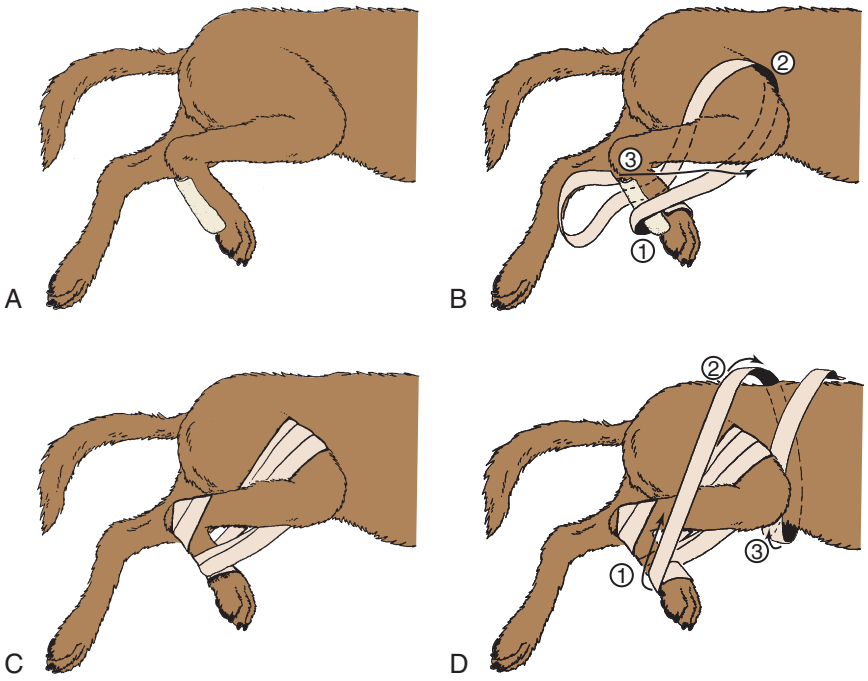


Figure 16-28 Ehmer (figure-of-8) sling. **A,** Application of the sling begins with placing some padding on the plantar surface of the metatarsus. **B,** Conforming roller gauze bandage is wrapped around the metatarsus (1) from lateral to medial, being sure to include most of the metatarsal pad. After several wraps to secure it, the gauze is carried medial to the flexed stifle (2) and over the cranial surface of the thigh. This internally rotates the limb at the hip joint. Finally (3) the gauze is brought medial to the tibia and tarsus and over the plantar surface of the tarsus. Several more circuits are made in the same manner. **C,** The gauze is continued in a figure-of-8 fashion around the flexed hock and paw to secure it. The entire bandage is then covered with elastic adhesive tape that overhangs the gauze to anchor the sling to the hair. Although some construct the bandage without any gauze by starting with adhesive tape applied to the skin, this invariably leads to considerable skin irritation on the cranial thigh region as well as the difficulty of removing the large amount of tape. **D,** It is difficult to keep the sling from slipping down over the stifle on short-legged breeds. One solution is to attach wide adhesive tape to the paw area of the completed sling (1), then carry the tape upward over the back (2) and around the belly (3). This is simple in the bitch or cat, but care must be taken to avoid the sheath in the male dog. (Adapted from Piermattei, DL, Flo GL, DeCamp CE: *Handbook of small animal orthopedics and fracture repair,* ed 4, St. Louis, 2006, Saunders.)

reduction of craniodorsal hip luxations, as well as some acetabular fractures, and can be maintained for up to two weeks. To apply an Ehmer sling, place several layers of cast padding around the metatarsal region. Next wrap roll gauze over it several times. Fully flex the stifle and wrap the rolled gauze around the thigh by bringing it medial to the thigh. Next, pull the gauze firmly and bring it over the front of the thigh to maintain flexion. Wrap the gauze over the lateral aspect of the thigh and then wrap it distally medial to the tarsus. The wrap is continued around the plantar aspect of the metatarsals, wrapping from medial to lateral to return to the original starting position. Repeat the pattern with rolled gauze three or four times. Finish with a layer of Elastikon applied in the same manner.

The pelvic limb sling, also known as a Robinson sling, prevents full weight bearing without maintaining the joints in a highly flexed position (Figure 16-29). A pelvic limb sling suspends the metatarsals to a bellyband and can be maintained for many weeks. Periodic recheck examinations are needed to maintain the integrity of the sling; replace it if the sling becomes soiled or wet. An e-collar is recommended to prevent the patient from removing the sling. A pelvic limb sling can be applied following fracture repair of the femur and tibia or in other situations where joint motion is desired without full weight bearing.

A carpal flexion sling prevents weight bearing on a forelimb and removes stress from flexor tendons (Figure 16-30). However, it is important to remember that prolonged application results in limitations in carpal extension and may require stretching and range of motion to reestablish normal extension of the carpus and digits. A carpal flexion sling can be applied following flexor tendon or fracture repair.

The 90/90 flexion sling is designed to maintain the tarsus and stifle in 90 degrees of flexion and is primarily used to prevent contracture of the quadriceps following repair of fractures of the distal femur in young dogs. To apply a 90/90 flexion sling, flex the stifle and tarsus to 90 degrees of flexion. With cast padding, wrap several layers around the metatarsal region and over the distal thigh. Adhesive tape or Elastikon is then wrapped around the flexed tarsal and stifle joints to maintain 90 degrees of flexion.

Hobbles may be applied to the distal antebrachii or cruses to allow full weight bearing and walking, but prevent

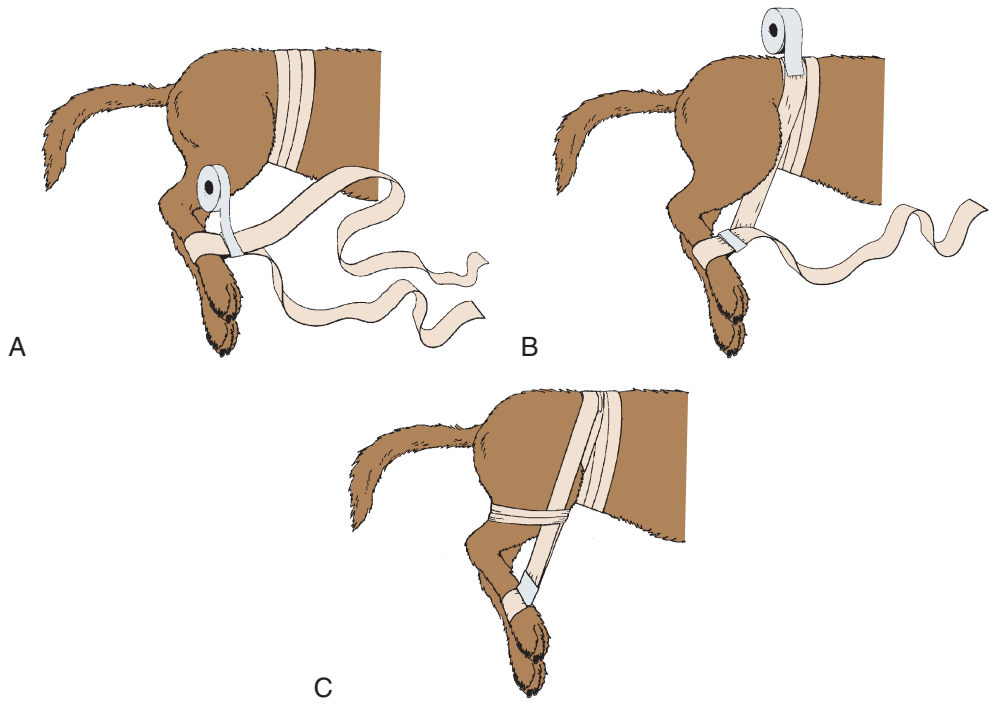


Figure 16-29 Robinson sling. **A,** The metatarsal region is padded with cotton or cast padding. A long piece of adhesive tape or Elastikon is wrapped around the metatarsal region, and the tape is stuck to itself near the foot. A belly wrap is also placed by wrapping the caudal abdomen with cast padding or roll cotton. Adhesive tape or Elastikon is then wrapped around the cotton. **B,** The medial strip of tape is then placed alongside the belly wrap, shortening the tape to an appropriate length so as to prevent weight bearing on the foot when standing. This strip is secured to the belly wrap with additional tape. **C,** The lateral strip of tape is then passed over the lateral surface of the thigh and attached to the belly wrap with additional tape. (From Piermattei, DL, Flo GL, DeCamp CE: *Handbook of small animal orthopedics and fracture repair,* ed 4, St. Louis, 2006, Saunders.)



Figure 16-30 Carpal flexion sling. This sling prevents weight bearing on a forelimb and removes stress from flexor tendons.

abduction. The most common use is in dogs with medial glenohumeral instability or ventral coxofemoral luxation. Cast padding is placed around both distal limbs. Adhesive tape may then be wrapped around the limbs to secure them in a standing position, but one which will not allow abduction to occur. More recently commercially fabricated hobbles that may be customized to the patient are available (Shoulder Stabilization System, DogLeggs, Reston, VA).

Aftercare and Complications of Slings

If care is not taken in placing slings, serious complications may arise such as tissue necrosis, neuropraxia, and severe limb edema. Toes should be checked at least twice daily for excessive swelling and warmth. If the toes are cool or swollen, the sling should be immediately removed and a new one placed after the clinician is certain that there are no circulatory problems. Other commercial alternatives have become available in recent years, using Velcro fasteners to make application easier (DogLeggs, Reston, VA). These slings are reusable and washable and allow for daily removal and reapplication for treatments such as passive range of motion exercises (Figure 16-31).

Postoperative Management of Bowel and Bladder

Patients often have decreased intestinal motility while in the hospital and thus may not defecate for days after surgery. Clients frequently express concern and want to know what they can do. Multiple factors contribute to decreased defecation, including discomfort when posturing to defecate or constipation or pain when defecating,



Figure 16-31 Removable and reusable slings are commercially available for the rear limbs. Slings are also available for the forelimbs (not shown).

such as following a pelvic fracture. Metamucil, DSS, or lactulose may be administered to soften stools. Another treatment that is simple and economical is the addition of cooked pumpkin to the food, which is high in fiber and serves as a source of insoluble fiber.

Urine present in the patient's cage is not evidence that the patient can urinate voluntarily or that the urinary system is intact after trauma. Appropriate evaluation of the urinary system should be made to be certain that it is intact after trauma. Urinary retention can lead to serious urinary tract infections, possibly even ascending to the kidney, which may induce acute renal failure. Overdistention of the bladder can lead to permanent atony of the detrusor muscle and incontinence. Regardless, knowledge of a postoperative patient's urinary status is paramount, and the patient should be directly observed urinating within 6 hours postoperatively. In addition, the patient's bladder should be evacuated immediately before and after surgery, especially if an epidural has been administered.

Advice for Clients

Client education begins before surgery. In discussing postsurgical follow-up care including cage confinement and restricted activity, patient limitations should be made explicitly clear. Also advisement about certain forbidden activities (running, jumping, playing with other dogs, etc.) and the risk of implant failure or reinjury if the patient is not made to follow recommendations must be clearly explained. Despite the veterinary staff's best efforts these instructions may still be ignored. Providing written instructions for the owners is important for good follow through. One alternative approach is to remind owners that the surgical procedure is an investment, and if they follow postoperative care instructions they would be protecting their investment and avoiding costly treatment for complications.

Tricks of the Trade

The following are a few notes regarding specific situations or operations.

- TPLO patients may develop edema around the hock a few days postoperatively, usually after discharge. The client may call and express concern. The veterinary staff can explain that this pocket of fluid around the hock has developed as a result of the patient's lymphatic system being disturbed as well as a decrease in circulation from reduced limb use. The client can be instructed to perform gentle soft tissue massage to move the fluid up the leg and increase lymphatic return. The fluid pocket will resolve with time. In addition, warm compresses may help the edema to resolve sooner. If edema continues and there is any drainage from the incision, the patient should be evaluated for possible infection.
- Also regarding postoperative TPLO patients, occasionally a client may call expressing concern that their dog was walking very well, then suddenly became lame on the operative leg and now is sensitive around the surgical site. It is prudent to recheck these cases as soon as possible to evaluate for postoperative complications. Complications include, but are not limited to, infection, fracture of the proximal fibula, screw breakage or loosening of the screw, patella ligament swelling, fracture of the patella or tibial tuberosity, or rarely, fracture of the tibia.[23] Radiographs and a good orthopedic exam are essential in diagnosing complications. Fractures of the fibula, patella, or tibial tuberosity usually result from too much activity, which applies excessive forces on bones, combined with the acute changes in biomechanics following rotation of the tibial osteotomy. Fractures of the fibula heal with proper conservative management consisting of pain medication and crate confinement. Fractures of the tibia, screw breakage, or plate loosening generally require additional surgery, and treatment should be determined by the veterinary surgeon. Patella ligament swelling also occurs as a result of overuse before a period of tissue adaptation. Enforced rest and symptomatic treatment generally result in resolution of patella ligament swelling.
- Dogs that have surgery of the foot, carpus, or tarsus or distal limb fractures may have marked distal limb edema following surgery. The veterinary staff should be diligent in checking the toes to ensure that the bandage, if any is used, is not too tight. Distal limb edema can be attended to multiple times a day with superficial thermal modalities and massage, in conjunction with antiinflammatory medication and bandaging.
- In the immediate postoperative period following patella luxation repairs, care should be taken with passive range of motion exercises, particularly stifle flexion. In some cases, reluxation may occur if passive range of motion is too aggressive. In complex repairs of patella luxation, passive range of motion may be contraindicated in the first 2 weeks following surgery.

Nursing Care for the Neurologic Patient

Bladder Care

Urinary bladder management is a cornerstone of neurologic nursing care. The type of bladder management necessary depends on the location of the lesion and type of spinal injury. Bladder management in the neurologic patient plays an extremely important role in the overall recovery of the patient. Recurrent bladder infections can complicate the health of neurologic patients. Patients with intervertebral disk disease localized to the T3-L3 thoracolumbar region develop an upper motor neuron (UMN) bladder, whereas those with lower lumbar or sacral region lesions develop a (LMN) lower motor neuron bladder. Upper motor neuron bladders have a hypertonic bladder sphincter and are difficult to express. Lower motor neuron bladders have a very relaxed, hypotonic sphincter, are easily expressed, and are frequently incontinent. Both types of bladders may have overdistention of the bladder with resultant bladder damage if the bladder is not evacuated regularly.

Many neurologic patients are able to urinate voluntarily but choose not to. If they are unable to ambulate, they sometimes will not urinate on their own. An opportunity to urinate outside should always be given first. If a patient has been given the opportunity to urinate outside and is unsuccessful, the bladder must be emptied either by expression or catheterization.

It is extremely important to understand the difference between voluntary urination and bladder overflow and distention. Urine present in the patient's cage is not evidence that the patient can urinate independently. Knowing the difference is critical to proper bladder management. Bladder overdistention can eventually result in permanent atony of the detrusor muscle. The bladder must be evacuated frequently and kept to a small size to avoid this problem. If urine is noted in the cage of a neurologic patient, bladder palpation should be performed to ensure the bladder has been emptied. If the bladder remains large and palpable, and attempts to urinate outside were unsuccessful, there is likely overflow and bladder expression should be performed.

With any spinal injury, cystitis or bladder infections can become a serious threat to the patient's health. Clinical signs of a urinary tract infection include foul odor to the urine, bloody or dark-colored urine, flocculent or purulent material in the urine, urine dribbling, constant licking of the genital area, depression, loss of appetite, and, in advanced cases, fever.

Bladder Expression

There are many options for bladder palpation techniques and patient positioning. Ensuring the patient is relaxed minimizes stress involved with bladder expression. Placing the patient in a standing position or lateral recumbency helps the caregiver to palpate the bladder more easily.

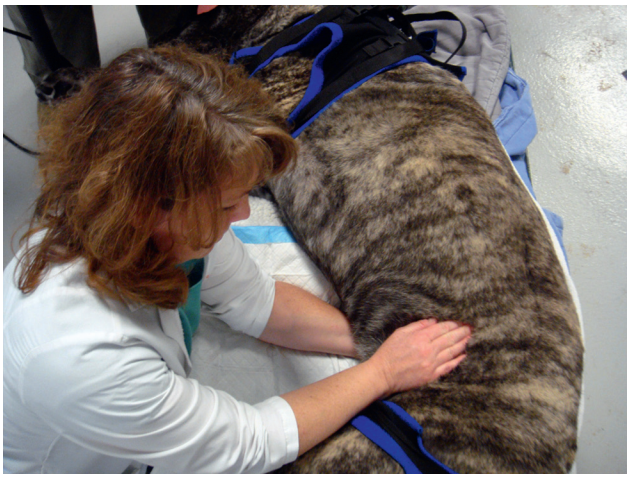


Figure 16-32 Bladder expression of the neurologic patient.

Place the hands under the abdomen between the last rib and the pelvis with one hand on either side of the patient. Slowly and gently palpate the patient's caudoventral abdomen for the presence of a bladder. Depending on the size of the bladder it may be located more cranially than one may anticipate. Place the hands cranially while applying gentle constant pressure to force the bladder to move caudally in between the caregiver's hands. It can be described as feeling somewhat like a water balloon that slips between your hands.

After the bladder is located, apply constant gentle pressure by compressing the two hands together, until urine is expressed. The bladder will gradually "deflate" as it empties (Figure 16-32). Bladder expression often can be extremely difficult, depending on the tenseness of the abdomen or if the patient is morbidly obese. Also, the difficulty to express may be caused by the underlying neurologic condition; with UMN bladders, spasm of the urethral sphincter may decrease or obstruct urine flow while expressing. Pharmacologic agents may be used to alter sphincter tone; the medication selected depends on whether bladder dysfunction is caused by UMN or LMN disease. Diazepam can be initiated if a UMN bladder is present to relax the skeletal portion of the urethral sphincter. This is especially important in male dogs. The initial dose of diazepam is 2 to 5 mg orally 30 minutes prior to bladder expression every 8 hours but can be increased as needed up to 10 mg. In addition, drugs may be used to relax the smooth muscle portion of the bladder, such as prazosin (1 mg/15 kg of body weight q 8 hours) or phenoxybenzamine (0.25 mg/kg po sid-bid, prn). Lower motor neuron bladders already have decreased sphincter tone, which makes it easy to express the bladder.

Bladder Catheterization

Most neurologic patients that have lost their ability to urinate can be managed with bladder expression alone. However, in some cases bladder catheterization is

appropriate and often necessary. In a large recumbent patient, even if the ability to urinate is not lost, bladder catheterization may be necessary because of the inability to rise and move around. It must be noted that an increased risk for urinary tract infections exists in catheterized patients. The choice of whether to intermittently catheterize the patient or use an indwelling system depends on the type of urinary catheter, the experience and availability of the veterinary staff, and clinician preference.

The type of urinary catheter and length are important to properly evacuate and measure urine output. If intermittent catheterization (two to three times daily) is warranted, a red rubber or a soft polyvinyl catheter can be used. If a more long-term indwelling system is desired, a softer, less irritating catheter such as a Foley catheter with a balloon device for anchoring within the bladder can be used. Whichever system is used, sterile placement is important.

Materials Needed for Female Catheterization

- Scrub solution
- Tiemann or Foley catheter, appropriate sizes
- Sterile gloves
- Sterile lubricant
- Sterile speculum
- Water and syringe for balloon device
- Extension set
- Urine collection system and bag

Procedure for Female Catheterization

Two people are required for catheterization of a female dog. The dog is placed in sternal recumbency. Sedation is often necessary to decrease patient discomfort during catheter placement. The vulva is cleaned with scrub solution. Sterile gloves are worn and the urinary catheter and lubricant used are also sterile. Lubricant is placed on the catheter end. The sterile vaginal speculum should be introduced vertically through the vulva lips and then turned horizontally. Two small indentations are on the floor of the vestibule. One is near the clitoral fossa; the other is the urethral opening set on the urethral papilla. The catheter should be carefully introduced through the urethral papilla. After urine starts to flow, the balloon is inflated with water as indicated on the catheter, and attached to a urine collection system. Most female urinary collection systems are indwelling because of the difficulty associated with repeated cathterizations.

Materials Needed for Male Catheterization

- Antiseptic solution
- Sterile red rubber or Foley catheter
- Sterile gloves
- Sterile lube
- Extension set (if indwelling)
- Urine collection system and bag (if indwelling)
- Syringe, three-way stopcock, and bowl (short-term)

Procedure for Male Catheterization

Two people are required for the catheterization of a male dog. The dog is placed in lateral recumbency. The person restraining the dog extrudes the penis. The prepuce and penis are gently cleaned with antiseptic solution, such as dilute chlorhexidine. With the penis extruded, the person placing the catheter wears sterile gloves and is sterilely handed the urinary catheter and sterile lube. Sterile lube is placed over the catheter tip. The catheter is introduced into the tip of the penis and the catheter is gently advanced into the urethra until urine starts to flow. If an indwelling catheter system is required, a sterile extension set is attached to a urine collection bag. To ensure the catheter is anchored in the bladder, the balloon of the device is filled with water, with the amount dependent on the size of the catheter. The amount needed is noted on the neck of the catheter. If the catheter is temporary, a three-way stopcock may be attached to the end of the catheter and a 60-cc syringe may be used to empty or measure the urine (Figure 16-33).

Catheter Care

Indwelling urinary catheter systems should be cleaned with a mild disinfectant solution at the prepuce of males and the vulva of females. Check four times daily to be sure the catheter is in place and urine is continuing to flow into the collection bag. The urine bag should be placed on a clean surface below the patient, but not on the floor. Exam gloves should be worn when emptying the urinary collection bag. The extension set clamp is closed, and the exit valve on the collection bag is opened. Urine is collected in a graduated cylinder or bowl to be measured. Care should be taken to maintain sterility of the collection system as much as possible. Remember to open the clamp on the extension set when finished to allow urine to flow.

Special Considerations

Cervical Neurologic Patients

Atlantoaxial subluxation occurs when instability between the first two vertebrae causes a compression of the spinal cord. This disorder usually occurs in young toy or small breed dogs but can also be seen in larger breeds. Clinical signs can be of congenital origin or traumatic. The severity of the clinical signs will determine the course of treatment. Clinical signs can vary from mild ataxia to severe neck pain, tetraparesis, or tetraplegia. Conservative management for mildly affected cases includes strict cage confinement and brace placement for 4 to 6 weeks. The brace can be made from Thermoplast or fiberglass cast material, and extends from mid sternum cranially to the chin. The brace prevents the patient from ventral flexion of the neck (Figure 16-34). The brace should be closely monitored for excessive tightness, areas of irritation, and wetness. If any of these occur, the brace should be removed and replaced. Assure that the brace is not too tight, to allow the patient



Figure 16-33 Urinary catheter placement in a male dog. **A,** The penis is extruded and the catheter is aseptically inserted. The catheter is passed until urine starts to flow. **B,** To anchor the catheter in the bladder, the balloon is inflated. **C,** For a closed collection system, a sterile extension set is aseptically connected to a collection bag.

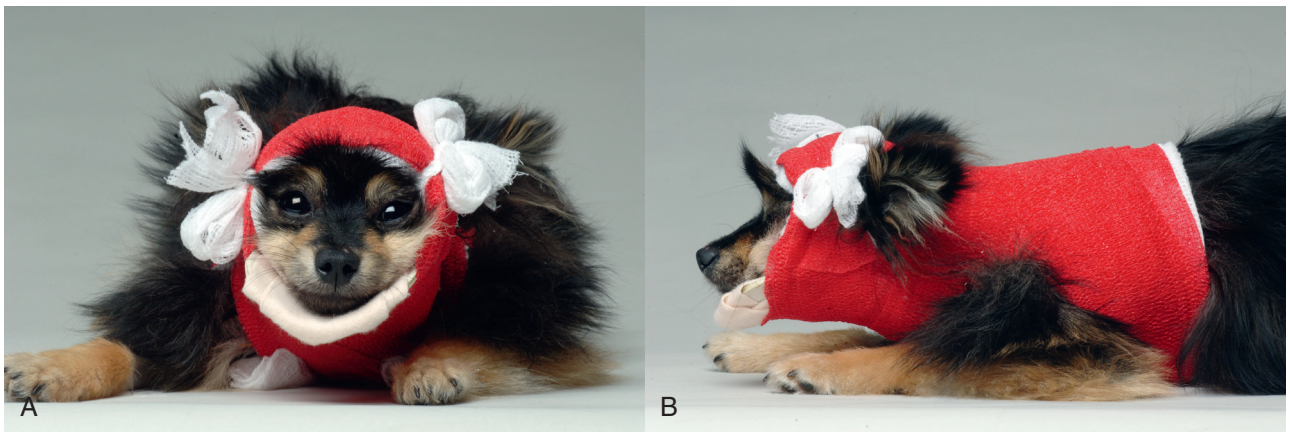


Figure 16-34 Coaptation for atlantoaxial luxation. **A,** Proper brace application to prevent the patient from ventral flexion of the neck. **B,** Lateral view of atlantoaxial brace. Note that the brace extends from the head to the cranial thorax.

to eat and drink. Many animals have difficulty eating, so syringe feedings with water and pureed food may be required. Keeping the patient in a comfortable position is very important. Positioning pillows and blankets around patients allows them to remain sternal, making it easier for them to eat, drink, and visualize the environment. For more severely affected cases, surgery is often required. Stabilization of the joint between C1 and C2 is performed. After surgical stabilization, a brace is still required for 4 to 6 weeks postoperatively.

Cervical spondylomyelopathy, also known as Wobbler's, is a condition primarily affecting large breed dogs. Clinical signs vary from ataxia to tetraplegia. Treatment options vary depending on the severity of the signs. Conservative management of mildly affected cases includes strict cage confinement for 4 to 6 weeks with only short walks outside with a harness. The use of neck leads or leashes should be avoided in cervical neurologic patients. Analgesics are administered as needed to control pain although many dogs with Wobbler's do not experience pain. Indications for surgery include substantial neurologic deficits or progression of signs despite conservative management. The surgical technique depends on the specific pathogenic changes identified on imaging, using MRI, CT, or myelography.

Postoperatively the cervical spinal cord patient can be very challenging. Frequently these patients are large to giant breed dogs that cannot be managed by the owner at home, requiring lengthy hospitalization until function returns. Early basic exercises focusing on activities of daily living are important for recovery. In severe cases patients may be so painful and weak that they are unable to lift their head. Foam supports and manual support or placement are required if the patient cannot maintain sternal recumbency. Nonambulatory tetraplegic patients should be assisted into sternal recumbency, or a standing position if capable, to eat and drink to prevent aspiration and regurgitation. Passive range of motion exercises to peripheral joints should be performed three to five times daily to prevent contracture and allow for joint nourishment. Assistive devices such as wheelchairs or a sling-lift system are frequently needed for postoperative cervical patients that are tetraparetic or tetraplegic (Figure 16-35).

Thoracolumbar Neurologic Patients

Intervertebral disk disease (IVDD) is the most common cause of thoracolumbar spinal injury in dogs. However, spinal tumors, fibrocartilaginous embolus, and trauma may also cause neurologic deficits. Hemilaminectomy to relieve the spinal cord compression is the most common surgical treatment of IVDD. Postoperative care of the IVDD patient focuses on pain control, bladder care, attention to bedding or housing, and postoperative rehabilitation. Often patient handling can be challenging if pain is not adequately controlled. In addition to oral and parenteral pain medications,



Figure 16-35 An assistive device, such a Hoyer lift, can be used to manage a large tetraparetic or tetraplegic cervical patient.



Figure 16-36 An assistive device, such as an abdominal sling, can be used to help a thoracolumbar patient stand and ambulate.

cryotherapy, electrical stimulation, gentle massage, moist heat (not within 72 hours postoperatively), and therapeutic laser may provide additional pain control for the patient.

Often patients with paraparesis drag themselves with the fore limbs. It is essential to discourage this behavior, because it can cause severe skin wounds and abrasions, increase potential for urinary tract infections (especially in females), and place significant stress on the forelimbs. Proper fitting with a sling (Figure 16-36) in the immediate postoperative period is recommended for paraplegic and paraparetic patients. A wheelchair or cart (Figure 16-37) is indicated if the disability is expected to last longer than 4 to 6 weeks (potentially earlier in large and giant breed patients).

Summary

Nursing care of the rehabilitation patient is an essential part of the rehabilitation program. Several factors,



Figure 16-37 An assistive device, such as a cart, can be used to help a thoracolumbar patient stand and ambulate.

including the procedure and owner expectations, must be considered when caring for the rehabilitation patient in order to have a successful outcome. Whether the patient has had an orthopedic or neurologic injury or surgery, the ultimate goal is the same: to assist and care for the patient to achieve a functional recovery.

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17 Devices for Ambulation Assistance in Companion Animals

Denis J. Marcellin-Little and David Levine

Independent locomotion is critically important in dogs and cats. In fact companion animals that have lost the ability to walk independently are at risk of being euthanatized.[1] Many devices have been conceived to provide ambulation assistance and independence from owner care.[2] Humans use canes, crutches, braces, orthoses, and prostheses to assist with locomotion.[3,4] In recent years the veterinary profession has been increasingly involved in physical rehabilitation for weak and paretic patients, and multiple devices aimed at providing ambulation assistance to animals have been created and used.[2,5] The devices used to assist companion animals may support or protect weak, unstable joints or feet (orthoses, boots); replace a missing portion of a limb (prostheses); or facilitate locomotion (slings, carts, ramps, steps). Assistive devices may also decrease the complications present in recumbent patients, including decubitus ulcers.[2] The purpose of this chapter is to present the range of ambulation assistive devices and discuss their purpose, benefits, construction, and functional use for companion animals.

General Considerations When Using Assistive Devices for Ambulation

Assistive devices present unique challenges to rehabilitation clinicians because their design, fabrication, fitting, and the patient training are most often unique. Their successful use requires an accurate assessment of the biologic situation and the mechanical needs of the patient, requiring knowledge in the fields of orthopedics, rehabilitation, biomechanics, and prosthetics.[6]

Dogs wearing assistive devices should not be left unsupervised because they may damage their assistive devices or injure themselves. Assistive devices may be tolerated differently by individual animals. They may present risks when they are partially dislodged. As a general rule hair protects the skin from abrasions and hair should be left intact unless it interferes with the performance of an assistive device. In patients with long hair, the hair may be trimmed or clipped to enhance the fit of a device. New devices should be worn under controlled conditions (i.e., indoors and under direct supervision) and for limited periods of time, starting with a few minutes. Devices should not interfere with blood flow. If a device is too tight blood flow to an extremity will be decreased, potentially leading to a loss of tissue oxygenation or impaired venous drainage with resultant swelling of the lower part of the limb. Devices should be stable on the limbs to avoid problems created by constant friction, slippage, or rotation. The condition of the limb should be assessed each time the device is removed. Skin redness, bruising, or focal hair loss should be noted early and immediately addressed, by potentially modifying the assistive device (removing hard material responsible for skin wear or adding foam protection). Devices should be inspected for wear and damage and cleaned daily. Cleaning is especially important if there are any incisions, wounds, or open sores in contact with the assistive device.

Boots

Boots are used to protect pads and feet from abrasion or to provide support. They may be used in normal dogs to protect against abrasions created by rough surfaces; for example, for search and rescue dogs working in areas with broken glass. Sled dogs may wear fleece boots to avoid the accumulation of ice on their feet or to avoid skin lesions from ice crystals, for example (Figure 17-1). Boots may be used to avoid abrasions of the dorsal surface of digits and metatarsal bones of dogs with sciatic nerve palsy, provided that they have some residual hock function. If the hock is flaccid, a support orthosis may also be necessary to maintain the lower limb in a functional standing position. Similarly boots may be used to protect the dorsal surface of the manus in patients with palsy of the branch of the radial nerve that innervates the extensor muscles of the antebrachium (Figure 17-2). Because most radial nerve palsies or brachial plexus injuries also affect the triceps brachii muscle and therefore severely limit weight bearing by the entire forelimb, boots may not provide adequate support alone. Boots may also be used in dogs with degenerative myelopathy and other conditions that lead to scuffing of the feet. Boots must be lightweight, particularly for patients that have weak motor function because the added weight of the boot on the foot may further decrease limb use. Boots may be made of nylon mesh, cotton, fleece,

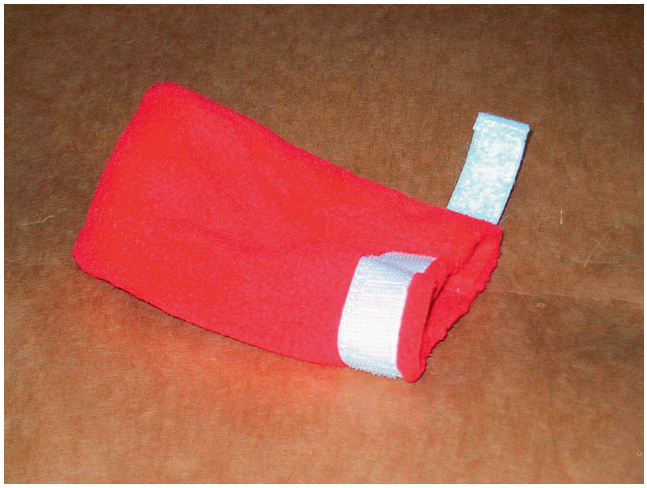


Figure 17-1 Fleece boot made for a sled dog. The boot has a simple design, with a single hook-and-loop band used to secure it in place. These boots are placed on all feet during sled dog races. They protect the pads from abrasions and other injuries caused by snow and ice.



Figure 17-2 Golden retriever with a partial brachial plexus avulsion sustained as a result of a fall from a moving vehicle. A palsy of the radial nerve is present. Function of the triceps brachii is weak and antebrachial extensor function is absent. The dog is seen wearing a sock, a folded bandana taped on the caudal aspect of the carpus, and a support orthosis (*inset*) placed inside a boot, stabilized with athletic tape. Although gait abnormalities remain (the affected forelimb is placed under the chest by abducting and externally rotating his humerus at a walk), the dog is nevertheless able to bear weight on his affected limb.

neoprene, rubber, or other materials. They may have a rubber sole for increased traction.[2] They may be held in place through the use of straps or hook-and-loop fasteners. Bootstraps are most often placed immediately proximal to the distal portion of the metacarpal or metatarsal bones in order to secure the boot.

Orthoses, Including Sleeves and Braces

Orthoses are devices applied to limbs for support and protection. They are applied on limbs in order to assist or limit joint motion. The most common orthoses are used to restrict the excessive joint motion present in patients with ligamentous or tendinous problems, including patients with ruptures of their palmar fibrocartilage or common calcaneal tendon (Figure 17-3). Orthoses function by placing the limb segments proximal and distal to unstable joints in a foam-lined rigid shell. Orthoses are most often made of rigid thermoplastics. They may be lined with foam, fleece, or terry cloth. They are most often held in place with hook-and-loop fasteners. Orthoses may be commercially available premade or they may be custom made. Premade orthoses are affordable and are best suited to patients with normal anatomy and healthy skin (i.e., patients with rupture of the common calcaneal tendon). Custom orthoses are usually necessary in patients with abnormal anatomy (limb deformity, abnormal skin



Figure 17-3 Siberian husky with ectrodactyly (lobster claw deformity) of his right forelimb. The right carpus collapses when bearing weight (*upper inset*). A custom orthosis has been made that supports his carpus (*lower inset*). The stability of the limb within the orthosis could be optimized by placing a third strap across the distal aspect of the radius.

contours) and in very small or very large patients. They are generally based on a cast of the abnormal limb made by use of semirigid or rigid fiberglass or plaster of Paris. They may also be based on a computed tomography–based replica of the limb. Solid replicas may be made of cyanoacrylate-impregnated plaster, ABS plastic, or laser-cured photopolymers.[7] Orthoses may be hinged to enhance their functionality.[8] The hinges most often connect rigid shells placed around the pes (or manus) and the crus (or antebrachium). These hinges may be passive or dynamic (i.e., spring loaded). Passive hinges may be made of nylon or metal (Figure 17-4).[8] Orthoses with passive hinges may enhance limb function because part or all of the motion of the tarsus (or carpus) may be maintained. Orthoses may have dynamic hinges that may be used to enhance the support of weak joints or to stretch contracted joints. Hinges are made dynamic by using springs embedded in the hinges or elastic bands.

Sleeves may be placed around limbs to provide protection or support, but they do not cover the feet. Although some sleeves offer protection of abnormal skin surfaces, for example in dogs with elbow hygromas or in recumbent patients at risk of developing decubitus ulcers, little is known about the benefits of sleeves for ambulation assistance. Sleeves may be reinforced, essentially becoming semirigid orthoses, by creating a pocket along their long axis and sliding a plastic or metal strip in that pocket.



Figure 17-4 Labrador retriever with a nonunion fracture of the metatarsal bones, hyperextended digits (*lower inset*), and a chronic nonhealing wound on the plantar surface of the foot. A hinged orthosis with a nylon hinge (*upper inset*) restores the dog's mobility.

Braces are placed across a joint, usually to limit its range of motion or promote its stability. Braces may be elastic or rigid. Rigid braces may also be used to modify the position of joints in nonfunctional limbs. This could be considered in patients with contractures, for example in patients with chronic grade IV medial patellar luxations that may have severe internal rotation of their tibia in relation to their femur. These patients may lose 60 degrees of stifle joint extension or more because of the shortening of their hamstring muscles as a result of the deformities and poor function of the quadriceps mechanism. A rigid brace may be considered in these patients with severe patella luxation and loss of stifle joint extension, when surgery is not a consideration or has failed (Figure 17-5). Braces may be made of foam-lined metal or thermoplastic, neoprene possibly reinforced with metal strips, or nylon. Most braces are hinged to allow joint motion. Hinged braces may be made of thermoplastic, neoprene, nylon, or other material.

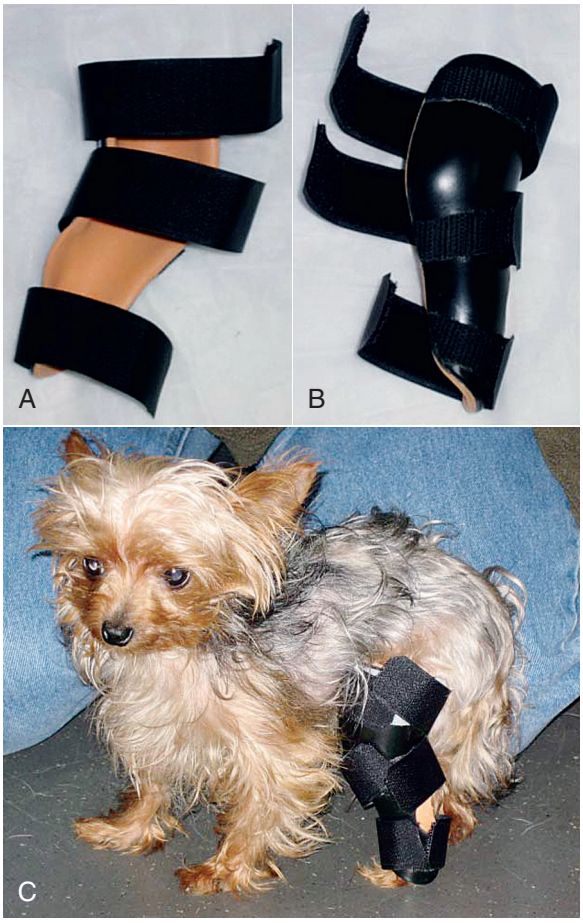


Figure 17-5 **A,** Yorkshire terrier with medial patellar luxation, internal rotation of the tibia in relation to the femur and lack of stifle joint extension, making the leg nonfunctional. He has been fitted with a rigid orthosis (**B, C**). The orthosis matched the dimension of his leg, and is made of thermoplastic, foam, and three hook-and-loop straps fitting around the thigh, crus, and pes.

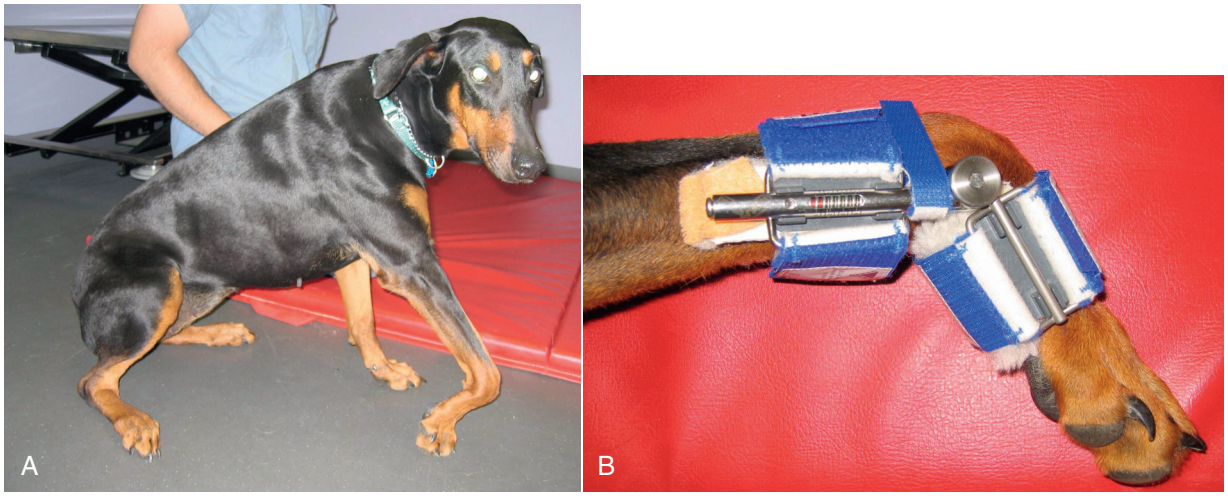


Figure 17-6 **A,** Doberman pinscher with an idiopathic loss of carpal extension. **B,** The antebrachial flexor muscles are stretched using a dynamic hinged brace that is worn for several hours at a time. The torque placed on the carpus is slowly increased over a period of weeks.

The hinges may be passive or dynamic (Figure 17-6). For example in the canine stifle joint, a brace theoretically could be used to decrease the stress placed on healing ligaments after traumatic stifle luxation, to limit the cranial thrust occurring while weight bearing in cranial cruciate deficient joints, or to improve the tracking of the patella. Despite efforts to design and commercialize several stifle joint braces, there is no brace that appears to predictably achieve any of these theoretical goals at this time. Knee braces are commonly used in humans for a variety of reasons, including protecting healing ligaments or improving patellar tracking.[9] Functional knee braces do not prevent joint subluxation in humans.[10] They appear to have a role in the anterior cruciate deficient knee, but only in conjunction with activity modification.[10] One problem in stabilizing the cruciate ligament deficient stifle is the conformation of the rear limb, and the slope of the tibial plateau at a standing angle. To eliminate subluxation of the tibia relative to the femur, it would be necessary to completely compress the soft tissues of the femur and tibia with the splint, severely compromising these tissues.

Prostheses

Prostheses are external devices used in most instances to replace a missing manus or pes. Like custom orthoses, prostheses are often fabricated based on casts of the affected and opposite limbs. The layer of the prosthesis that contacts the skin, named *contact layer*, may be a silicone liner or foam adhering to a shell. Standard pediatric silicone liners may be used for long antebrachia. Custom silicone liners may be used for smaller or larger patients and for other body parts (i.e., thighs). Liners may be secured to shells using locking pins embedded in their distal portions or they may be held in place by friction within the shell. Most prostheses have custom shells made of rigid thermomoldable plastic, fiberglass, or carbon fiber. Thermoplastics are cheapest, and their relative lack of strength and stiffness relative to other materials does not represent a significant drawback in smaller patients. Fiberglass or carbon fiber may be preferred for large patients. Little is known about the optimal shape of external prostheses for partially amputated forelimbs or pelvic limbs in companion animals.[5] However, it is likely that a longer residual limb will provide better stability and reduce chances of skin breakdown. The stability of the prosthesis can be defined as axial, angular, and torsional. Axial stability is the displacement of the prosthesis along the long axis of the limb (i.e., the lack of movement of the prosthesis when it is being pulled on and pushed on). Axial stability is for the most part the consequence of the shape of the bone and surrounding soft tissues. It is also influenced by skin mobility in relation to underlying soft tissues. Axial stability often relies on the presence of bone prominences that are used to *lock* the prosthesis in place, creating *suspension*. Companion animals, as a general rule, have more discrete bone prominences than humans and the process of locking the prosthesis in place is harder to achieve. Angular stability is the lack of movement of the prosthesis in relation to the residual limb when mediolateral and craniocaudal forces are applied to it. Angular stability is most often the consequence of the length of a residual limb segment: the longer the better. Torsional stability is the lack of motion of the prosthesis in relation to the residual limb when internal or external torsion is applied. Torsional stability may be hard to achieve when a limb segment has a small diameter and is relatively round. This is particularly relevant in the antebrachium. Axial and torsional stability of prostheses are increased when the prostheses span a joint (i.e., the hock, stifle, or elbow joint). This is often



Figure 17-7 Sheltie who lost his toes after surgical resection of a soft tissue sarcoma of his thigh. The distal portion of his stump is sensitive to touch. His stump is placed in a silicone-lined glove finger and fitted in a hinged prosthesis held in place with four hook-and-loop fasteners.

achieved through the use of hinges placed over the center of rotation of the joint proximal to the end of the residual limb.

Adjustable devices may facilitate the function of the distal portion of these prostheses.[7] The weight-bearing surface of prostheses is most often rounded and made of low-wear, nonskid rubber. The rubber stiffness is chosen based on patient size, mobility, and anticipated level of activity. Some prostheses are hinged at the carpus or tarsus. These hinges may enhance the stability of the prosthesis on the limb by increasing the contact area between the shell and the skin without excessively interfering with joint motion. Hinges may also be included in the prosthesis to promote a joint position more compatible with weight bearing. Hinges add complexity and cost to prostheses, but they may greatly enhance limb function (Figure 17-7).

Straps and Bands

Straps and bands may be used for ambulation assistance. Elastic bands designed to assist patients with compromised hock flexion, for example in patients with sciatic or peroneal nerve palsy, are commercially available (Figure 17-8). These elastic bands are attached to the mid-metatarsal region and to a ring on a chest harness. Elastic bands or tape may also be used to form hobbles, limiting forelimb or pelvic limb abduction. Pelvic limb hobbles are most often used in the management of patients recovering from ventral hip luxations. Forelimb hobbles are most often used to manage patients with injuries of the medial glenohumeral ligament.[11]



Figure 17-8 Labrador retriever with a sciatic neuropathy, weak hock flexion, and scuffing of hind limb toes. Elastic bands within nylon sleeves are connecting his pes to his dorsal midline. The bands promote hock flexion and eliminate scuffing.



Figure 17-9 Dalmatian is supported by a chest and pelvic limb sling. The sling has a longitudinal chest handle and a transverse pelvic handle. It may be used to control the dog and to avoid falls.

Slings

Slings may be used to support weak patients with neurologic or musculoskeletal conditions. They may also be used to control dogs or avoid falls. Examples include patients recovering from orthopedic procedures of their pelvic limbs, such as total hip arthroplasty. Slings may be made of neoprene or other material, including cotton or nylon. Slings often have straps with an adjustable length for comfort of the therapist and should also be washable (Figure 17-9). Thin support sling bands may be used to control dogs recovering from injuries or surgery

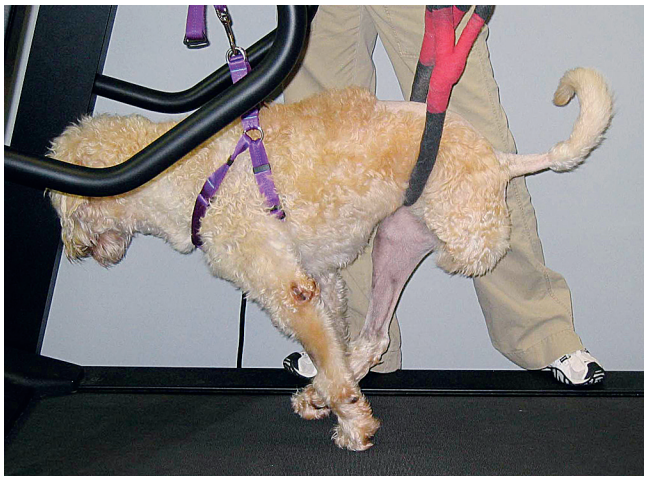


Figure 17-10 Labradoodle with an amputation of his left pelvic limb. The dog is recovering from a total hip replacement of his right hip joint preformed to manage a femoral malunion with severe osteoarthritis. The dog is trotting on a treadmill, controlled and supported by a chest harness and a belly sling. The sling is made by wrapping self-adherent bandaging tape around a surgical gown.



Figure 17-11 Saluki supported by a paracart. The pelvis is supported by a rigid base made of foam-covered tubing. The nonfunctional pelvic limbs are held in a slightly flexed position, off the ground. The dog's back is horizontal, and weight distribution between forelimbs and pelvic limbs appears optimal. (Courtesy of Mari Hiruma, Dog Will Project, Tokyo, Japan.)

(Figure 17-10). Slings supporting the abdomen may be used in conjunction with slings supporting the pelvic limbs and thoracic limbs. Subjectively abdominal slings do not appear to assist with locomotion as effectively as pelvic limb slings in dogs with pelvic limb weakness. Specialized slings are available to assist forelimb amputees (Appendix). Slings may be combined with exercise bands to improve the rehabilitation and locomotion of neurologic patients recovering from injuries or surgery. Patients who cross their pelvic limbs may benefit from the placement of a rolled cotton towel within a pelvic limb sling.[2]

Carts

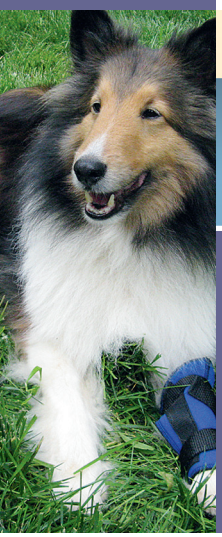
Carts provide temporary or permanent assistance to nonambulatory patients through support and prevention of the deleterious effects of recumbency. Several companies in North America have commercially available carts (Appendix—Manufacturers of Supplies and Equipment). Carts may be made of wide variety of materials. Their frames may be made of aluminum rods or tubing, or PVC piping.[12] Tires may be rigid or pneumatic. Wheels may be connected using ball bearings. As a consequence, the performance, weight, durability, and cost of ambulation carts are quite variable.

Paraparetic or paraplegic patients are usually fitted with carts that have two wheels or, possibly, three wheels if a small wheel is used on the caudal aspect of the cart. These paracarts support the lumbar spine and pelvic limbs by offering a rigid resting surface for the pubic and ischial symphyses or by slinging the pelvic limbs (Figure 17-11). Rigid resting surfaces usually are foam-covered aluminum tubing. Cart slings are most often made of neoprene with nylon straps and side release clips but they may be made of nylon mesh, cotton canvas, or other material. Optimal support materials are strong, breathable, washable, soft, and have some elasticity. Subjectively, rigid resting surfaces may be ideally suited to dogs without motor function in their pelvic limbs, and slings appear ideally suited to dogs with residual or intact motor function that need pelvic limb support. Dogs can spend several hours each day in properly fitted and balanced carts. Forelimb paracarts may also be used to support the chest of dogs with forelimb problems. This may be done using a special balanced two-wheeled cart or a quad cart (one with four wheels). Two-wheeled carts are more compact, more maneuverable, and cost less than quad carts. Tetraparetic or tetraplegic patients are usually fitted with "quad" carts. Like para carts, quad carts are available with rigid resting surfaces or slings.

Most carts are custom made. Cart measurements are specific to each manufacturer. The standing measurements required for cart constructions include: height of the dorsal midline, caudal to the scapula and at the lumbosacral joint; forelimb circumference proximal to the elbow joint; chest length and circumference, thigh length and circumference at the flank fold; height at the groin; width of the chest, abdomen, and pelvic regions; and width between the front legs. Some carts, however, are adjustable in height, width, and length.

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SECTION IV

Therapeutic Modalities

18 Superficial Thermal Modalities

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Thermotherapy is the use of superficial heat and cold as a therapeutic modality for the treatment of disease or trauma, and it may be applied using many different methods.

Superficial heat and cold have been used for centuries to manage soft tissue and joint injuries with the specific goals of relieving pain, altering the physiologic processes underlying tissue healing, and affecting the elasticity of connective tissue, including muscle, tendon, ligament, and joint capsule.[1] The primary goal of any thermal modality is to facilitate the ultimate therapeutic modality of exercise.

A dog adjusts its temperature based on the information supplied by the peripheral receptors, and, based upon the information, activates mechanisms such as trembling, sweating, and modifying the local circulation. Thermotherapy works on this latter process.[1]

Thermotherapy applications may be simple or sophisticated, being delivered through hot or cold water, hot or cold packs, hot or cold whirlpools, ice massage, contrast baths, cold compression units, and other methods.[1]

Before the application of heat or cold, the skin should be checked for the level of sensation, and if decreased or absent, extreme care must be used when applying thermal modalities to prevent injury. The skin should also be checked for any redness, burns, scabs, or wounds because these may alter the thermal transmission or affect the method of treatment. The area being treated should be cleaned to remove dirt or any other substance on the skin that could decrease conductance or alter the uniformity of the modality. The skin should also be inspected a short time after applying the modality to check for excessive temperature changes, and the skin should be monitored throughout the treatment. Cryotherapy is most effectively used immediately following trauma (accidental or following surgical procedures) to provide analgesia, control bleeding, and reduce inflammation, edema, and muscle spasm.

Cryokinetics combines cryotherapy with motion (passive, active-assisted, or active) to facilitate normal, pain-free movement, and to reduce edema through a muscle pumping action to facilitate the return of lymphatic fluid to the vascular system. The primary benefit of cryokinetics is to facilitate the patient's ability to perform pain-free exercise early in the rehabilitation process as long as the level of exercise remains below that which causes further injury. As signs of acute inflammation begin to subside, the clinician may elect to transition to the use of heat if indicated.

Superficial heat is best applied before performing stretches or exercises to improve range of motion (ROM). This is to take advantage of the benefits of tissue temperature elevation, including alteration of tissue viscoelastic properties, which results in increased soft tissue extensibility and decreased joint stiffness. In addition heat causes vasodilation, which increases tissue oxygenation and transport of metabolites to the exercising tissue, and increases the rate of enzymatic and biochemical reactions that may facilitate tissue healing. However, heat applied too early in the healing process may result in increased inflammation, increased enzymatic activity detrimental to cartilage (e.g., collagenase and gelatinase), and increased local metabolism.

The Physics of Thermotherapy

Thermotherapy applications fall in the infrared portion of the electromagnetic spectrum just beyond the wavelength and frequency ranges for visible light.[2] The shorter the wavelength, the greater the frequency and the more shallow the depth of penetration. The thermal electrophysical modalities discussed in this chapter fall within the category of "near infrared" and possess shorter wavelengths than electrical stimulating currents. Therefore, infrared electrophysical agents penetrate to a shallower depth compared with electrical stimulating currents. The biologic effects of

electromagnetic radiation depend on the frequency used, duration of exposure, tissue characteristics, and power density.[2]

Electromagnetic energy and its transmission through the body are governed by several physical laws. The first law is the Arndt-Schultz Principle[1]: Tissue must absorb the energy produced by the thermal agent to stimulate the tissue's normal function. If the energy absorbed is insufficient to stimulate the tissue, there is no effect. If too much energy is absorbed, tissue damage may occur. The second law, related to the first, is the Law of Grotthus-Draper, which determines the fate of the energy. If a tissue does not absorb the energy, it is transmitted to deeper tissue layers. If the energy is absorbed more superficially, less energy is transmitted to deeper layers and less penetration of the energy occurs. When electromagnetic energy encounters the body's tissues, it has three possible fates: it may be reflected (e.g., reflected from the skin's surface), it may be refracted (e.g., at the interface between the dermis and subcutaneous fat), or it may be absorbed (e.g., by muscle). The estimated depth of penetration for most infrared thermal modalities (cold or hot packs, whirlpools, paraffin baths, or luminous infrared devices) is approximately 1 to 2 cm.

Heat may be transferred by the following mechanisms:

- **Conduction**—the transfer of heat by the direct interaction of the molecules in one area with those in another area (assuming the areas have a temperature difference). (Example: cold/hot packs)
- **Convection**—heat transfer by movement of air or fluid from a warm area to a cooler area. (Example: the circulatory system, air temperature)
- **Radiation**—the exchange of electromagnetic energy that occurs when there is a difference in temperature between two objects. Heat transfers from a warmer to cooler area. (Example: sun, infrared lamps)
- Evaporation—vapocoolant sprays are used to cool the skin and underlying tissues by causing evaporation and extracting heat from the tissues.
- Conversion—the conversion of a nonthermal form of energy (mechanical, chemical) that penetrates the tissues and is converted to heat. (Example: therapeutic ultrasound; sound waves being converted to thermal energy within the tissues)

Most thermal techniques transfer energy by conduction. Energy travels down a thermal gradient, with energy (heat) being removed from an object, rather than cold being added. Heat always travels from the warmer object to the cooler.[3] According to Knight,[4] the rate at which heat transfer occurs depends on the following factors:

- *The temperature difference between the body and the modality:* The greater the difference, the more quickly the transfer of heat occurs.
- *Regeneration of body heat and modality cooling:* As tissue gives up heat, the heat lost is replaced by circulating blood and surrounding tissues. Cold application tends to result in greater tissue temperature changes at greater tissue depths as compared with superficial heating modalities. For example, after a 20-minute ice pack application to the thigh muscles, the intramuscular temperature declined more slowly than the subcutaneous temperature, and there was less cooling at deeper tissue levels (and never reached as cold a temperature as the subcutaneous tissue). When the ice pack was removed the subcutaneous tissue temperature rose steeply, whereas the intramuscular temperature continued to decline for a period of time, possibly because heat was transferred from the deeper tissues to warm the superficial tissues, resulting in continued tissue temperature decreases at the greater tissue depths (Figure 18-1).
- *The heat storage capacity of the modality:* The more energy it takes to convert a solid to a liquid (e.g., ice to

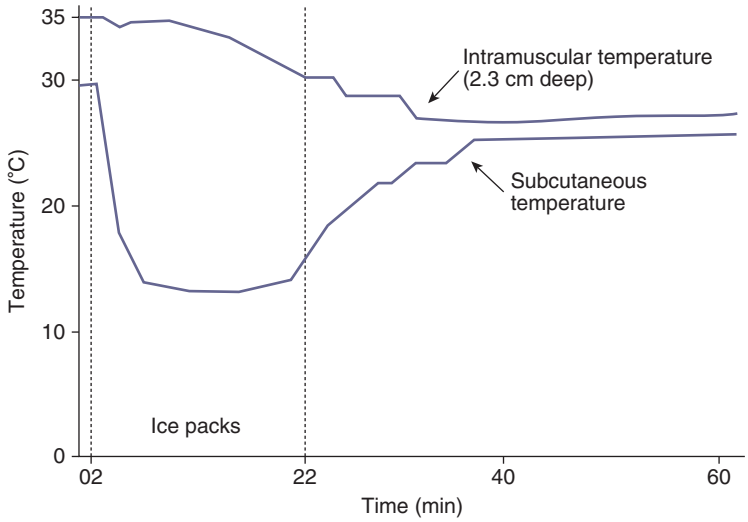


Figure 18-1 Intramuscular versus subcutaneous tissue temperature changes with cryotherapy. Intramuscular (gastrocnemius) and subcutaneous temperatures during and after a 20-minute ice pack application. Note that intramuscular temperature declined much more slowly than subcutaneous temperature and continued to decrease after the ice pack was removed. (Redrawn from Hartviksen K: Ice therapy in spasticity. *Acta Neurol Scand* 38(suppl 3):79-84, 1962.)

water), the greater its latent heat of fusion and its capacity to remove heat from a tissue. For example crushed ice packs require more energy for this conversion to occur as compared with semisolid gel packs. Therefore ice packs cool tissues for a longer time and to a greater extent than gel packs.

- *The size of the modality:* The larger the modality (e.g., large cold pack), the greater the energy storage capacity.
- *The area of the body in contact with the modality:* For example cold water immersion results in the greatest tissue temperature decline compared to other methods of cold delivery because a greater surface area is in contact with the cooling modality.[5] Vapocoolant sprays result in the least temperature decline and provide only superficial cooling because of the relatively small surface area cooled.
- *The application duration:* The longer the contact time between the two surfaces, the more opportunity for energy exchange to occur.
- *Individual variability:* Individuals with lower body fat percentage exchange heat more quickly than those with greater amounts of subcutaneous fat.

Cryotherapy

Physiologic Effects of Cryotherapy

One of the oldest methods of physical therapy is cryotherapy, the application of cold after trauma or surgery. Cryotherapy is used not only during the acute phase of tissue injury and healing to mitigate the effects and sequelae of tissue injury, but also after exercise during rehabilitation to minimize adverse secondary inflammatory responses.[5-7]

The most important physiologic effects are:

- Vasoconstriction
- Decreased blood flow
- Decreased swelling
- Reduced enzyme-mediated tissue damage
- Analgesia

The decrease in blood flow slows edema formation after injury or after surgery. The cooling of the tissue reduces the metabolic rate of the treated tissue, decreases the rate of reactions related to the acute inflammatory process, causes the inhibition of enzymatic effects related to inflammation and minimizes the release of histamine, which reduces the tissue damage.[6,7] At a temperature of 30° C or lower, cartilage-degrading enzymes (protease, hyaluronidase, collagenase) are inhibited. The vasoconstriction of blood vessels also may help to decrease pain by decreasing pressure on nociceptors, which may be stimulated as a result of increased tissue swelling and edema, as well as by other physiologic means such as decreased nerve conduction velocity.

The primary effect of cryotherapy is the vasoconstriction mediated by a local reflex and the central nervous system. The resulting reduced blood flow is useful not only to decrease swelling, but to control hemorrhage and the flow of inflammatory cells as well. The Hunting reaction, first described by Lewis in 1930, refers to the cyclical temperature oscillation of 2° to 6° C every 8 to 15 minutes after the tissue temperature approaches 2° C. This effect begins 20 to 40 minutes after cryotherapy application. He attributed these oscillations to cold-induced vasodilation (CIVD) as an attempt to protect tissues from cold-induced damage. However, his results have been incorrectly and widely applied to the sports medicine literature in an attempt to explain the success of cryokinetics.[1] Knight et al.[8] replicated Lewis' experiment and concluded that Lewis' observation during cold immersion and subsequent rapid increase in temperature after cryotherapy are caused by temperature effects, not CIVD and subsequent increased blood flow. Based on these studies, the primary benefits of cryotherapy are twofold: decrease in cellular metabolism and analgesic effects to permit cryoexercise to facilitate rehabilitation.[4,8]

The analgesic response to cryotherapy treatment is mediated by the peripheral nervous system. Peripheral thermal receptors are categorized as cutaneous myelinated type III or A delta, which are sensitive to pricking or sharp pain and cold; unmyelinated type IV or C receptors for aching pain; and unmyelinated type IV or C cutaneous receptors for pain and temperature (Table 18-1).

The unmyelinated cutaneous receptors respond to absolute temperature and the rate of temperature change.[9] The cold-sensitive receptors begin firing at 36° C and increase their firing rate until reaching their maximum at approximately 25° C. The firing frequency drops sharply at temperatures below 20° C and is minimal by the time the receptors are cooled to 10° to 12° C. Conversely, the warm receptors also begin firing at 33° to 36° C, rapidly reach their maximum firing frequency at 43° C, and drop to a minimal firing rate at 45° C.[9,10] Both the hot and cold receptors rapidly adapt to temperature changes.

Central thermosensitive neurons in the preoptic and anterior hypothalamus respond to temperature changes with autonomic responses to stimulate heat retention by the body, by altering cutaneous blood flow and thermoregulatory behavior such as panting. Figure 18-2 illustrates the regulation of temperature by the hypothalamus. The anterior hypothalamus also controls thermoinsensitive neurons, such as those that are sensitive to osmolarity and glucose concentration.[9,11] Stimulating the posterior hypothalamus by heating does not result in the autonomic responses characteristic of the anterior hypothalamus, but behavioral responses to the heat load occur. Local warming of the medulla increases respiratory frequency. Autonomic thermoregulatory responses also take place at the spinal cord level.[11] The sympathetic nervous system response to

Table 18-1 Sensory Receptors: Anatomy, Location, and Function

Receptor	Type	Sensory Nerve Classification	Location	Diameter (μm)	Conduction Velocity (m/sec)	Sensory Function
Sensory (afferent) myelinated	A	Ia	Muscle	12-20	72-120	Muscle spindle primary ending (or 1-degree ending)
	A	Ib	Tendon	12-20	72-120	Golgi tendon organ Rate of muscle shortening
	A	II	Muscle	6-12	36-72	Muscle spindle, secondary ending (or 2-degree ending) muscle length changes
	A	II	Skin	6-12	36-72	Vibration, discriminatory touch, pacinian corpuscles
	A	III	Skin	1-6	6-36	Pricking, sharp pain, temperature (cold), light touch
Unmyelinated	C	IV	Muscle	~1	0.5-2.0	Aching pain
	C	IV	Skin	~1	0.5-2.0	Pain, temperature

From Guyton AC: Sensory receptors and their basic mechanisms of action. In *Textbook of medical physiology*, Philadelphia, 1986, Saunders.

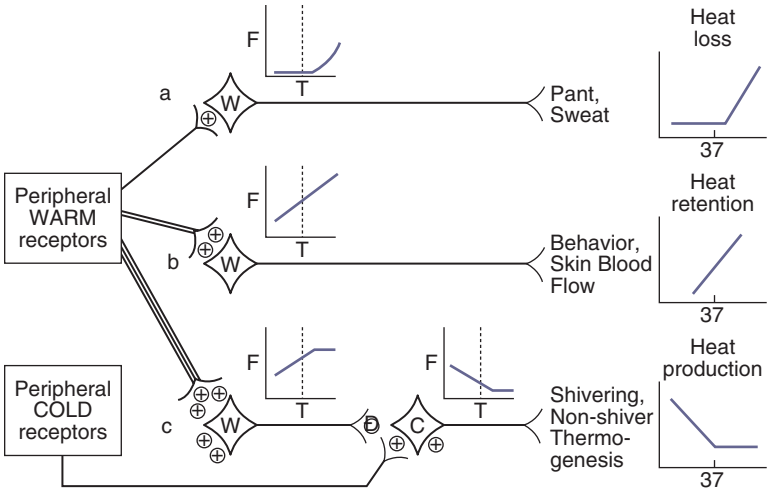


Figure 18-2 Central hypothalamic control of thermoregulation. Diagram shows neuronal control of various thermoregulatory responses. +, excitatory input; –, inhibitory input; dashed line, thermoneutral temperature; C, cold sensitive neuron; F, neuronal firing rate; T, hypothalamic temperature; W, warm sensitive neuron. (Redrawn fro Bouland JA, Dean JB: Temperature receptors in the central nervous system, *Ann Rev Physiol* 48:639-654, 1986.)

thermal load is also chemically mediated through the release of the neurotransmitters epinephrine and norepinephrine from the adrenal medulla to induce cutaneous vasoconstriction.[9]

A primary effect of cryotherapy is analgesia and the concomitant reduction of reflex muscle spasm. This may be caused by the decreased nerve conduction velocity that occurs when nerves are cooled. This relationship, known as the Q10 effect, is thought to be linear until 10° C, when neural transmission is blocked.[12,13] Nerve cooling also increases the duration of the refractory period, the time when a nerve cannot be stimulated by a second impulse.[14] Conversely raising the subcutaneous tissue temperature increases sensory nerve conduction velocity, with the greatest effect occurring during the initial 2° C temperature elevation.[15] Another mechanism postulated for the analgesic effect afforded by cryotherapy is that the cold receptors are overstimulated by cryotherapy, resulting in pain control

at the spinal level by preventing pain transmission to higher centers via the spinal gate control theory of pain transmission.

Cryotherapy decreases muscle spasm through a number of possible mechanisms. Muscle spasm compromises venous return and promotes lactate accumulation and acidosis. The muscle spindle receptors and Golgi tendon organ receptors fire more slowly when cooled, although the effect is less pronounced on the Golgi tendon organs.[16] Sudden cooling has an excitatory effect on the muscle spindle, resulting in increased alpha motoneuron activity and increased muscle guarding. As the temperature continues to decrease, primary spindle afferent activity diminishes.[12] Muscle spasm, resulting from the pain-spasm-pain cycle, stimulates the static stretch response of the type II tissue afferents. Applying superficial heat until the temperature is above 42° C decreases the muscle spindle discharge rate while it increases the firing rate of the Golgi tendon organ.[16] This sequence of events results in a decrease in the firing rate of the alpha motor neuron. Thus cold may raise the threshold stimulus for muscle spindle activity, decreasing muscle spasm.[17]

Thermal effects on strength and endurance have also been observed by a number of authors. The force-velocity curve of muscle contraction shifts downward with decreasing temperature.[18] For a given concentric contraction velocity, cooler muscles have a lower force output. Interestingly, some studies suggest that proprioception, joint position, and balance remain largely unaffected by cryotherapy, whereas others indicate that joint position awareness is affected by joint cooling.[19-21]

Although most authors agree that changes occur in muscle torque production with cooling, it is less clear whether those effects are specific to a particular contraction mode (concentric acceleration or eccentric deceleration). One group of researchers reported that eccentric muscle action is augmented following cryotherapy,[22] whereas another group found increases in concentric, but not eccentric, torque.[23] Still others found no effects on peak torque, but increased muscle endurance following cooling.[24] Functionally, vertical jump performance, an indicator of lower extremity explosive power, is adversely affected by cryotherapy.[25,26] These findings suggest that caution must be used in making recommendations for performance following cryotherapy. Clearly the literature on this subject is undecided as to the specific effects of cold (both immediate and delayed) on muscle strength. These findings have the most implication for dogs who are engaged in work or competitive athletic pursuits. It also requires that the clinician understand the physiology and biomechanics of muscle actions and sport demands. Cold and exercise are an effective combination in the treatment of acute musculoskeletal dysfunction unless restrictions in flexibility are present.[27,28] Combining movement with cold allows for relatively greater pain-free exercise and assists in muscle pump activity to reduce acute injury-related edema.

Because of these effects, cold is most effective when applied during the acute phase of trauma, typically in the first 72 hours after injury or surgery. Cryotherapy may also be useful in chronic pain situations such as arthritic joints. With this condition it can be used to reduce pain, edema, collagenolysis, synovial inflammation, and joint destruction.[1,4,5] As a result of its analgesic effect, cryotherapy may permit decreased use of drugs for pain. This was noted in human medicine in a study examining numerous patients after anterior cruciate ligament surgery. Cryotherapy is currently used in postoperative treatment for swelling and pain, in musculoskeletal injury, muscle spasm, and after exercise to prevent edema or pain.

This thermal modality may also have a significant influence on the effectiveness of laser therapy, given its effects on blood flow and the saturation of blood chromophores in the tissue. Where tissue perfusion is reduced with cryotherapy, the absorption of laser energy may be improved, so that deeper structures may receive greater laser energy. This use of cryotherapy before laser therapy treatment could be useful for large dogs in which the target tissue is deeply located.[29]

Cryotherapy Applications

Cold application may be applied in several ways: cold water-circulating blankets, reusable ice packs, ice cubes wrapped in a towel, ice cups, cold immersion, cold compression devices, vapocoolant sprays, or contrast baths (Figure 18-3). Selection of the method depends on the desired effects, depth of penetration desired, stage of tissue healing, treatment area, and physiologic goals.

Cryotherapy may be achieved by the following physical mechanisms:

- Conduction (ice/cold packs, iced towels, ice massage, contrast bath, cold compression units)



Figure 18-3 Cryotherapy options (*clockwise starting top right*): Popsicle-type molds for ice massage, alcohol-water slush plastic bags, cotton wraps, commercial cold wraps, fluoromethane cold spray.

- Convection (cold baths)
- Evaporation (vapocoolant sprays)

Local application of cold may increase connective tissue stiffness (sometimes with decreased tensile strength) and may temporarily increase muscle viscosity (with decreased ability to perform rapid movement).

Generalized application of cold over the whole body, or large portions of it, causes decreased respiratory rate; generalized vasoconstriction in response to cooling of the hypothalamus; increased muscle spindle bias, which can increase spasticity (opposite to the effect of localized cold application); and increased muscle tone, which may be accompanied by shivering. Knowing this, if a patient has lower motor neuron flaccidity, cryotherapy may be used to temporarily facilitate muscle contraction.

For sanitary reasons and to improve patient comfort and prevent damage by cryotherapy to the treated tissues, a thin towel or other material (e.g., towel or pillowcase) is commonly placed between the cold source and the skin. It is very important to observe the skin for response to cold during the treatment. A normal response at the end of a cryotherapy treatment is reddening of the skin, and an abnormal response indicating that damage may have occurred is whitening or blanching of the skin. Because of skin pigmentation in some dogs, these color changes may not be apparent, so it may be better to err on the side of caution and reduce the length of application time if there is any question. The expected changes in sensation during a treatment follow the acronym IBAN: intense burning, aching, and numbness.[8]

Cryotherapy is a superficial treatment, but effects of up to 1 to 4 cm of tissue depth may be noted. The depth depends on the amount of adipose tissue and local blood flow. For these reasons, cryotherapy may be more effective in the distal area of the limbs where joint capsules, tendons, and ligaments are more superficial and there is less adipose tissue.[3,15,30]

Ice Packs/Cold Packs

Examples of ice packs/cold packs are crushed ice placed in a moist towel; a plastic bag wrapped in a moist towel; or water and rubbing alcohol of a 3:1 ratio in a sealed plastic bag and placed in a freezer (this should result in a slush-type consistency). Numerous types of cold packs are available commercially (Figure 18-4). The treatment time is generally 10 to 20 minutes for an application of cryotherapy.

The latent heat of fusion is lower for the alcohol-water mix than for water alone, therefore it performs similarly to crushed ice alone but at a lower temperature. Use caution with the water-alcohol mix because the potential for cold-induced injuries is greater. Never apply this type of cold pack directly to the skin. Instead, apply using a layer of toweling, either wet or dry (wet conducts cold more rapidly), between the cold pack and skin. Commercial cold packs may also be applied. Packs that freeze solid (e.g., DuraKold) perform better than frozen gel packs, which contain gelatin or antifreeze to make them more pliable (Figure 18-5).[4] Cold packs containing ethylene glycol antifreeze should not be used in animals because the contents are toxic if ingested. As with the alcohol-water mixture, use caution because the risk for frostbite is greater than when using a crushed ice bag (see Figure 18-5).

Bocobo et al studied the effects of cryotherapy on intraarticular temperature in dogs' knees.[5] Four treatment protocols were examined on 24 dogs: ice pack applied for (1) 5 minutes, (2) 15 minutes, (3) 30 minutes, and (4) local ice bath immersion for 15 minutes. Intraarticular temperature decreased in a time-dependent fashion, with temperatures decreasing 2.2° C, 4.1° C, and 6.5° C after 5, 15, and 30 minutes of ice pack application, respectively. Temperatures continued to drop for a short period of time after the ice pack was removed, and required 21.7, 33.2, and 27.8 minutes to return to pretreatment temperatures, respectively. Ice bath immersion resulted in the greatest decrease in joint temperature, 20.2° C and had the greatest rewarming time of 59.8 minutes. Interestingly, rectal temperature did not decrease with 5 or 15 minutes of ice pack application, and only decreased 0.5° C with 30 minutes of treatment. Ice bath immersion caused a 1.6° C drop in rectal temperature, however. These data indicate that under normal circumstances, local application of ice packs results in tissue cooling in a time-dependent manner with minimal effects on core body temperature. Furthermore, the cooling effect appears to plateau after 30 minutes, suggesting that treatments longer than 30 minutes offer no further benefit in regards to intraarticular temperature decrease. Immersion in an ice bath results in more rapid and greater cooling than ice pack treatment. There was a prolonged rewarming period in all tissues after the removal of cryotherapy, and these results should be considered before beginning cryotherapy treatment.

Vannetta et al. also examined the effects of a 20-minute cold pack treatment to dogs on skin and muscle temperature as well as intramuscular blood flow.[31] Temperatures and blood flow ratings were recorded for each minute of the application and for 80 additional minutes following the removal of the cold pack (Figure 18-6). They found that the temperature of the skin and subcutaneous tissues decreased to a greater degree than did the tissues at depths of 1 and 3 cm. The temperature of the superficial tissues rose sharply following removal of the cold pack but the temperature of the deeper tissues continued to decrease for 10 minutes and remained decreased for 60 minutes before beginning to return to baseline. Blood flow was decreased in the superficial tissues and remained decreased after the cold pack was removed. Blood flow at 1- and 3-cm depths, however, were initially decreased but began to rise before the treatment ended and remained above baseline levels for the 80 minutes following the cold pack removal. This study



Figure 18-4 **A,** Example of a cryotherapy treatment using a cold pack wrapped around a dog's hind limb. **B,** Canine stifle icer. **C,** Carpal icer.

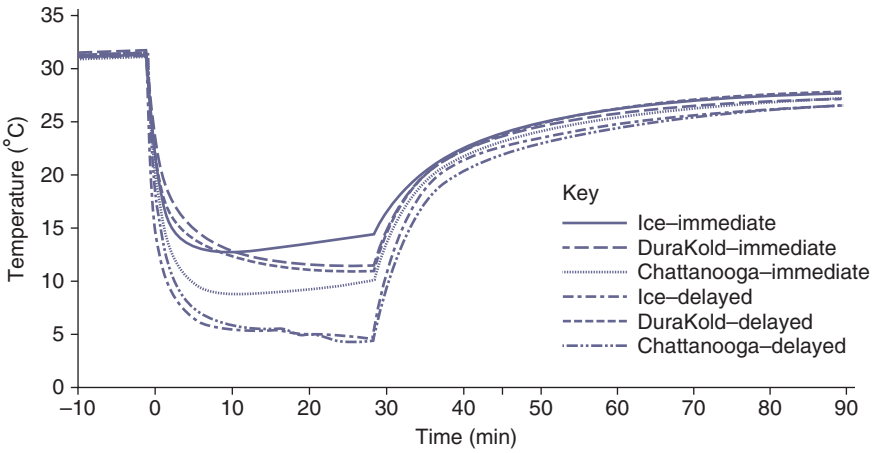
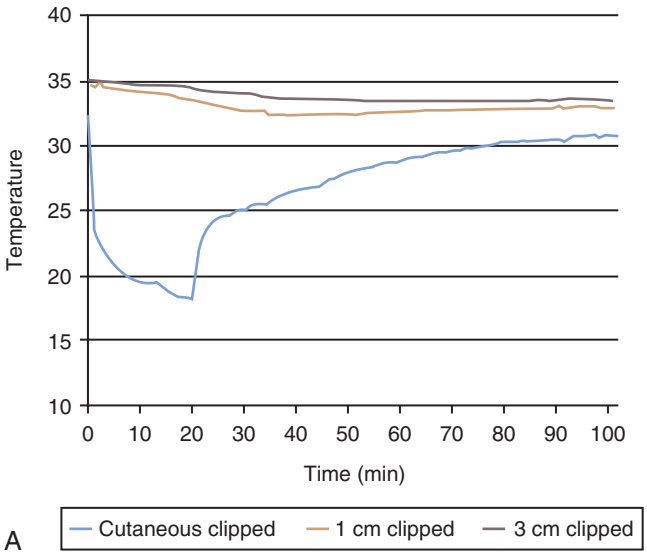
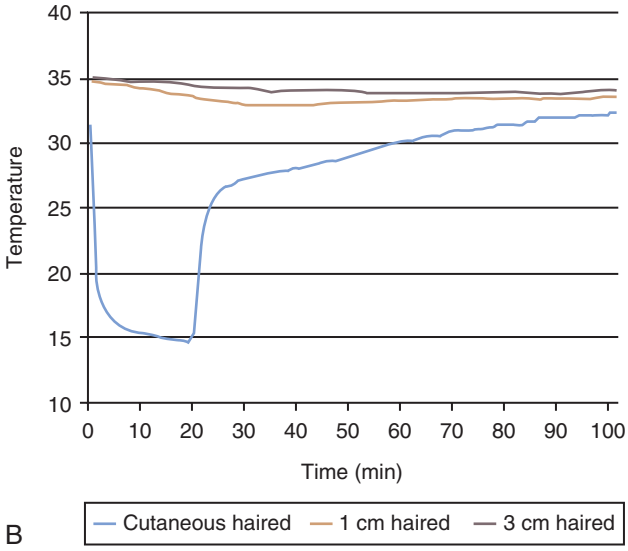


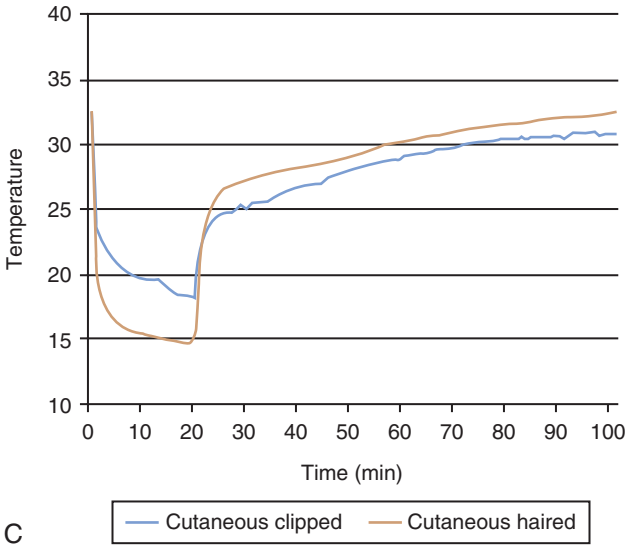
Figure 18-5 The effect of the heat of fusion can be seen in this comparison of several forms of cold packs. The crushed ice and DuraKold packs cool the skin more than the Chattanooga frozen gel pack. The effect is even greater when application is delayed for 20 minutes after the packs are removed from their cooling devices. (Knight KL, A re-examination of Lewis' cold-induced vasodilation in the finger and ankle. *J Athl Train* 15:238-250, 1980.)



A



B



C

Figure 18-6 **A,** Temperature of caudal thigh muscles during cryotherapy application for 20 minutes and rewarming in dogs with the hair clipped. Note that the most dramatic temperature changes occur in the skin. Changes in the deeper tissues occur slower, but the effects are more prolonged, presumably as a result of heat transfer from regions of higher temperature to lower temperature. **B,** Temperature of caudal thigh muscles during cryotherapy application for 20 minutes and rewarming in dogs with hair. Note that the most dramatic temperature changes occur in the skin. Temperature decrease is greater in dogs with hair during cryotherapy application, and rewarming occurs more quickly, likely because of the insulating effect of the hair. Changes in the deeper tissues occur slower, but the effects are more prolonged, presumably as a result of heat transfer from regions of higher temperature to lower temperature. **C,** Temperature of the skin over the caudal thigh muscles during cryotherapy application for 20 minutes and rewarming in dogs with the hair clipped or with hair intact. Note that greater changes occur in haired dogs, suggesting that hair provides an insulating effect.[31]

concluded that the temperature of deeper tissues may remain decreased for up to 70 minutes following a cold pack treatment. Also blood flow increases following the removal of a cold pack in deeper tissues, perhaps in an attempt to rewarm the tissues back to their baseline levels.[31]

The effects of cold −16°C (3°F) compresses were studied on epaxial muscles in dogs. The gel compress was applied for 5, 10, and 20 minutes in random order and temperature changes at 0.5, 1.0, and 1.5 cm of tissue depths were measured. At 10 minutes, the cold decreased tissue temperature at 0.5, 1.0 and 1.5 cm depths 7°C, 4.7°C, and 4°C, respectively. At 20 minutes the cold further decreased tissue temperature to 8.2°C, 6.5°C, and 4.7°C at the respective depths. The dogs were of ideal body condition making the results a good guide, but not applicable to all dogs. The differences between 10- and 20-minute applications were significant but applications of 10 minutes were sufficient for cooling.[32]

Cold-Compression Units

Cold-compression units alternately pump cold water and air through separate tubing in a sleeve wrapped around a patient's limb. The temperature of the water is usually set between 2° and 10°C (35 to 50°F) to provide cooling. Compression is applied by intermittent inflation of the sleeve with air. Units manufactured for humans may be noisy, and the sleeves for human use do not typically conform well to dogs. Commercially available units are now made for use in dogs (Figure 18-7) and can be a highly effective treatment during the acute phase of inflammation or tissue healing.

Cold Immersion

Cold immersion treatments consist of immersion of an affected body part in a container of cold water. The temperature of the water for this type of treatment is usually 2° to 16°C (35° to 60°F), and the treatment generally lasts for 10 to 20 minutes. Cold water immersion results in the greatest decrease in tissue temperature because the largest surface area is in contact with the cold source. This is a difficult form of cryotherapy to use in veterinary medicine because of compliance issues, and it requires the patient to stand in the immersion vessel. The target tissues should be the only areas immersed in the water. Cold water immersion should not be used in the presence of large wounds because of the risk of infection, and delayed healing may result.[26]

Contrast Baths

Contrast baths are applied by alternate immersion of an area in warm and cold water. This modality produces alternating vasodilatation and vasoconstriction and is sometimes used to decrease edema. Contrast baths are appropriate not only in the acute phase, but during the early subacute phase of tissue healing as well. During the acute phase of tissue healing, the cycles of alternating immersion should have greater emphasis on cold water. An example of this would be immersion in the cold water for 3 minutes and then immersion in warm water for 1 minute. As the stage of tissue healing progresses, the times spent in warm water may increase as the time spent in the cold decreases. Treatment time for this method of cryotherapy may last from 15 to 30 minutes. Contrast bath treatment for edema should end with cold. The varying sensory stimulus is also thought to promote pain relief and desensitization. This treatment should be considered for conditions such as chronic edema, subacute trauma, sprains, strains, and tendonitis. Alternating hot and cold packs has also been proposed as an alternative to contrast baths.

Ice Massage

Freezing water in cups or any other form of cylinder can be used to form an ice massage medium (Figure 18-8). To



Figure 18-7 Game Ready cold compression device with stifle sleeve, http://www.gamereadycanine.com.



Figure 18-8 Ice massage being performed on dog's hind limb.

apply the massage the therapist holds onto the cup, exposes the ice surface, and puts it in direct contact with the patient's skin. Treatment time is generally 5 to 10 minutes or until the affected area is erythematous, slightly pink or red, and numb. This technique can be useful for small, irregular areas. For large areas, a cold pack is more effective regarding efficient use of time.

Vapocoolant Sprays

These sprays are liquids used to achieve a brief and rapid cooling effect when applied to the skin. The spray absorbs heat as it hits the skin, and the increase in temperature causes it to change from a liquid into a vapor. To use the vapocoolant spray to treat trigger points, place the affected muscle in a position of slight stretch and spray from proximal to distal at a rate of 10 cm/sec from a distance of 30 cm (12 inches). Repeat four or five times until the affected area has been covered. It is important to cover the entire length of the muscle with the spray, and the muscle should be stretched during and after the application. This type of treatment is frequently applied directly over the skin just superficial to a trigger point in human populations.[33] This can be a difficult modality to apply to dogs because of the hair covering the skin surface. Also, some of the commercially available sprays may be harmful to animals if they contact an animal's eyes or are ingested.

Treatment, Duration, and Frequency

Cryotherapy treatment times depend on the modalities chosen and the treatment area, but the application time is usually 15 to 20 minutes and may be repeated throughout the day. A common method is to repeat the cold application every 2 to 4 hours during the first 24 to 48 hours after the injury, or treat 10 to 15 minutes on and 10 to 15 minutes off twice. An ice treatment should not be applied for more than 20 minutes per session to avoid tissue damage. Local hypothermia should never exceed 30 minutes per treatment session for the same reason, as well as to avoid stimulating unwanted local vasodilatation and edema formation.

Precautions and Contraindications

The therapeutic effect of cryotherapy is believed to occur when the tissue temperature reaches 15 C° to 19°C, but this is dependent on the tissues normal temperature. Previous regions of frostbite should be avoided. Although it may be difficult to check skin color of dogs for redness or blanching because of pigmentation and hair coat, it should be done after the first 5 to 10 minutes to be certain that continued treatment is safe. Apply cryotherapy with caution over superficial nerves; open wounds (because of the vasoconstriction that occurs); fractures; areas where there may be sensory or motor nerve impairment; previous frostbite; in patients with hypertension (cold may increase blood pressure with systemic application); to areas with decreased or absent sensation; and in very old or very young patients.[34]

Cryotherapy Applied with Coaptation Devices in Place

Dogs and cats frequently have external coaptation devices, such as bandages or casts, placed over limbs following injury or surgery. Because cryotherapy is useful during the acute postinjury phase, it would be beneficial to apply cryotherapy over coaptation devices to achieve the beneficial effects of the cryotherapy without the need to remove and reapply coaptation devices. In the case of cast application following fracture treatment, it would not be practical or advisable to remove and replace a cast several times daily.

In general it is believed that a cast or bandage prevents or reduces the effects of cold on the skin when applied over a cast or bandage. Several studies have addressed the use of cryotherapy over casts or bandages in people, and although specific studies are not available in animals, these findings may serve as guidelines for use in animals.

One study evaluated the magnitude of temperature changes at the skin of the ankle after the application of frozen ice packs to the surface of various casts and bandages both in normal and swollen ankles.[35] A Robert Jones bandage, elastic support bandage, a below-knee plaster cast, or synthetic below-knee cast were applied to patients, and two frozen ice packs were placed around the cast or bandage. Although skin temperature under dressings and casts decreased significantly relative to the baseline temperatures with application of two frozen ice packs in all groups, less skin temperature decrease occurred with the Robert Jones bandage (32° to 23°C) as compared with casts (31° to 16°C with fiberglass casts). In addition, the length of time to achieve the lowest skin temperature required 46 minutes compared with 36 minutes for the Robert-Jones bandage and fiberglass cast, respectively. Furthermore this study only evaluated skin temperature decrease, so the effects on deeper tissues are unknown.

A related study found similar results.[36] Over a 90-minute period of ice pack application, the average final skin temperature was 16.5°C under a plaster cast, 18.8°C under a synthetic cast, 21.2°C under an Ace wrap dressing, and 28.7°C under a Robert-Jones dressing. The authors concluded that ice application effectively decreased skin temperatures through the plaster cast, synthetic cast, and Ace wrap dressing, but the Robert-Jones dressing prevented adequate cooling. Other studies have similarly concluded that a synthetic or plaster cast does not eliminate tissue cooling when crushed ice packs are applied to the surface of the casts, but the time required for maximum cooling is approximately 1 hour,[37] and bulky dressings inhibit adequate cooling.[38]

Postoperative Cryotherapy

A recent study compared cold compresses, a modified Robert Jones bandage, cold compresses plus a Robert

Jones bandage, and microcurrent electrical stimulation in combination with a Robert Jones bandage. The use of cold compresses alone or with a modified Robert Jones bandage, or using microcurrent electrical stimulation in combination with a modified Robert Jones bandage decreased soft tissue swelling over 72 hours more than bandaging alone after extracapsular repair of CCLR.[39] A similar study in patients following TPLO surgery found that cold compression therapy decreased signs of pain, swelling, and lameness, and increased range of motion during the first 24 hours after surgery.[40]

Heat Therapy

Few therapeutic procedures have been historically used for as long as heat; it is one of the oldest forms of physical therapy. Superficial heating agents penetrate to tissue depths up to approximately 2 cm, whereas deep heating agents elevate tissue temperature to depths of 5 cm. Although skin temperature may increase 10° C or more, tissues at 1 cm in depth are typically raised less than 3° C and tissues at 2 cm less than 1° C.

Physiologic Effects of Heat

The effects of heat are opposite those of cold, except that heat and cold both may relieve pain and muscle spasm. With an increase in tissue temperature, there is a sedative and analgesic effect, increased leukocyte migration into the heated area, increased metabolic rate, and arteriolar dilation that results in increased capillary blood flow (which can promote edema) and decreased blood pressure (if heat is applied for a prolonged time or over a large surface area).[1,34,41] Other benefits of superficial heat may include reducing edema (however, heat should not be used during acute inflammatory periods because edema may be exacerbated) and increasing patient comfort. The most important beneficial physiologic effects of heat are:

- Increased circulation (vasodilation)
- Pain relief
- Increased soft tissue extensibility
- Relaxation of muscle spasm

Heat causes vasodilation and increases the rate of blood and lymph flow, thus potentiating the reabsorption of edema, which helps to remove tissue metabolites, and increase tissue oxygenation and metabolic rate. Normally the metabolic rate increases two to three times for every 10° C (18° F) increase in temperature. Increased enzymatic and metabolic activity has been observed in tissues at 39° to 43° C (102° to 109° F), and the activity rates continue to increase until 45° C (113° F). Beyond this temperature, the protein constituents of enzymes begin to denature and enzyme activity rates decrease, ceasing completely at about 50° C (122° F).[17,34] Any increase of enzymatic activity results in an increased rate of cellular biochemical reactions. This can increase oxygen uptake and accelerate healing but may also increase the rate of destructive processes.

Thermotherapy may cause vasodilation by a variety of mechanisms, including direct reflex activation of the smooth muscles of the blood vessels by the cutaneous thermoreceptors, indirect activation of local spinal cord reflexes by the cutaneous thermoreceptors, or by increasing the local release of chemical mediators of inflammation (histamine, prostaglandin, bradykinin). These cause an increase in capillary permeability as well.

Superficial heating agents stimulate increased activity of the cutaneous thermoreceptors. It is proposed that transmission from cutaneous thermoreceptors, via their axons, communicate directly to nearby cutaneous blood vessels to cause the release of bradykinin and nitrous oxide. Bradykinin and nitrous oxide act as vasoactive mediators stimulating relaxation of the smooth muscles of the blood vessel walls causing vasodilation. This vasodilation occurs locally, in the area where the heat is applied. Cutaneous thermoreceptors also project, via dorsal root ganglia, to synapse with interneurons in the dorsal horn of the gray matter of the spinal cord. These interneurons synapse with sympathetic neurons in the lateral gray horn of the thoracolumbar segments of the spinal cord to inhibit their firing and thus decrease sympathetic output. This decrease in sympathetic activity causes a reduction in smooth muscle contraction, resulting in vasodilation both at the site of heat application and in the cutaneous vessels of the distal extremities. This distal vasodilative effect of thermotherapy may be used to increase cutaneous blood flow to an area where it is difficult or unsafe to apply a heating agent directly. The vasodilation of the vessels of the distal extremities can promote the reabsorption of edema, but in the same way can promote edema if applied during times of acute inflammation.[34]

Local application of heat increases sensory and motor nerve conduction velocity. An increase or decrease in conduction velocity reduces pain and muscle spasm. Nerve conduction velocity increases by approximately 2 m/sec for every 1° C (1.8° F) increase in temperature.[34] The clinical implications of these effects are not well understood. They may contribute to reduced pain perception or improved circulation that occurs in response to the increasing tissue temperature. Nerve firing rate (frequency) also changes in response to changes in temperature. Elevation of muscle temperature decreases the firing rate of type II muscle spindle efferents and gamma efferents and increases the firing rate of type Ib fibers from Golgi tendon organs. These changes in nerve firing rates are thought to contribute to a reduced firing rate of alpha motor neurons and thus, to a reduction in muscle spasm. The decrease in gamma neuron activity causes the stretch on the muscle spindles

to decrease, reducing afferent firing from the spindles. The decreased spindle afferent activity results in decreased alpha motor neuron activity and thus, relaxation of muscle contraction.[11,22]

Another effect of heat is to relieve pain. Several studies demonstrate that the application of local heat can increase an individual's pain threshold. The inhibitory control of pain at the spinal cord level was explained in the gate control theory of Melzack and Wall in 1965.[42] This theory suggests that the perception of pain is not a direct result of activation of nociceptors, but instead is modulated by the interaction between different neurons, both pain-transmitting and non–pain-transmitting. The theory asserts that activation of nerves that do not transmit pain signals can interfere with signals from pain fibers and inhibit an individual's perception of pain. The gate control theory suggests that sensory inputs of pain are modulated through ascending and descending pathways in the central nervous system. Descending neural pathways potentiate or attenuate pain signals influencing the amount of neurotransmitter released by the incoming neurons or by changing the sensitivity of the ascending nerves in the spinal cord to these neurotransmitters. Afferent pain-receptive nerves, those that bring signals to the brain, are comprised of at least two kinds of fibers: a fast, relatively thick, myelinated A delta fiber that carries messages quickly with intense pain and a small, unmyelinated, slow C fiber that carries the longer-term throbbing and chronic pain. Large-diameter A beta fibers are non-nociceptive (do not transmit pain stimuli) and inhibit the effects of firing by A delta and C fibers.

The peripheral nervous system has centers at which pain stimuli can be regulated. Some areas in the dorsal horn of the spinal cord that are involved in receiving pain stimuli from A delta and C fibers also receive input from A beta fibers. The non-nociceptive fibers indirectly inhibit the effects of the pain fibers, "closing a gate" to the transmission of their stimuli. In other parts of the laminae, pain fibers also inhibit the effects of non-nociceptive fibers, "opening the gate." An inhibitory connection may exist with A beta and C fibers, which may form a synapse on the same projection neuron. The same neurons may also form synapses with an inhibitory interneuron that also synapses on the projection neuron, reducing the chance that the latter will fire and transmit pain stimuli to the brain. The inhibitory interneuron fires spontaneously. The C fiber's synapse may inhibit the inhibitory interneuron, indirectly increasing the projection neuron's chance of firing. The A beta fiber, on the other hand, forms an excitatory connection with the inhibitory interneuron, thus decreasing the projection neuron's chance of firing. (Like the C fiber, the A beta fiber also has an excitatory connection on the projection neuron itself.) Thus, depending on the relative rates of firing of C and A beta fibers, the firing of the non-nociceptive fiber may inhibit the firing of the projection neuron and the transmission of pain stimuli. The gate control theory thus explains how a stimulus that activates only non-nociceptive nerves can inhibit pain. One area of the brain involved in reduction of pain sensation is the periaqueductal gray matter that surrounds the third ventricle and the cerebral aqueduct of the ventricular system. Stimulation of this area produces analgesia by activating descending pathways that directly and indirectly inhibit nociceptors in the laminae of the spinal cord. It also activates opioid receptor–containing parts of the spinal cord.[27,34,42] Therefore the control of pain produced by thermotherapy may be caused by gating of pain transmission by activation of cutaneous thermoreceptors, but may indirectly be the result of improved healing, decreased muscle spasm, or reduced ischemia.

Muscle strength and endurance may decrease during the initial 30 minutes after the application of deep or superficial heating agents. It is proposed that this initial decrease in muscle strength is the result of changes in the firing rates of type II muscle spindle efferent, gamma efferent and type Ib fibers from Golgi tendons organs caused by heating of the motor neurons. Beyond 30 minutes after the application of heat and for the next 2 hours, muscle strength gradually recovers and then increases to greater than pretreatment levels.[11]

Increasing the temperature of soft tissue increases its extensibility. When soft tissue is heated before stretching, the length gained is maintained for a greater amount of time after the stretching force is applied, less force is required to achieve the increase in length, and the risk of tissue damage is reduced. If heat is applied to collagenous soft tissue such as tendon, ligament, scar tissue, or joint capsule before prolonged stretching, plastic deformation, whereby the tissue increases in length and maintains most of the increase after cooling, can be achieved. In contrast, if collagenous tissue is stretched without prior heating, elastic deformation, whereby the tissues increase in length while the force is applied but lose most of the increase when the force is removed, may occur.[34,43] For this reason, thermotherapy can be used clinically to increase joint ROM and decrease joint stiffness. Both of these effects are, in fact, the result of the increase in soft tissue extensibility that occurs with increased soft tissue temperature. Increasing soft tissue extensibility contributes to increasing joint ROM, especially if stretching is performed immediately after the application of heat or during it. If the tissues are allowed to cool before being stretched, the effects of prior heating on tissue extensibility are lost.[34]

Decreased muscle spasm may be a result of relieving the ischemia caused by the chronic contraction of a muscle and partial occlusion of the blood vessels. Superficial heating also decreases muscle spindle activity by decreasing gamma efferent activity, which may also be responsible for the decrease in spasm as well as breaking the pain-spasm cycle.[34]

Heat Applications

Superficial heating agents typically heat the skin and subcutaneous tissues to a depth of 1 to 2 cm. Although skin temperature may increase 10° C or more, tissues at 1 cm in depth are typically raised less than 3° C and tissues at 2 cm less than 1° C. The tissues are usually heated for 15 to 20 minutes to promote hyperemia.[34]

Heat therapy may be transmitted by:

- Conduction (hot packs, paraffin, hydrotherapy)
- Convection (hydrotherapy, air temperature)
- Radiation (UV lamps, infrared)

Heat therapy (Figure 18-9) may be used to relieve pain, relax muscle spasm, increase the blood supply to an area, and increase soft tissue extensibility. Subacute and chronic inflammation or traumatic conditions such as contusions, sprains, strains, and myositis can also benefit from heat therapy.

Superficial heating agents may include: hot packs, heat wraps, hosing with warm water, whirlpools, paraffin baths, circulating warm water blankets, electric heating pads, and infrared lamps. The last two are considered to have a higher risk of burns in animals. Another common form of superficial thermotherapy is heated beds for dogs, which have been used for comfort or conditions such as arthritis.

Hot Packs

Different forms of hot packs include heated sacks with a canvas covering that may be filled with cracked corn, beans, bentonite (hydrophilic silicate gel), or other inert materials; electric heating pads; damp microwaved towels; or circulating warm water blankets. There are different kinds of hot pack shapes and sizes designed to fit different areas of the body, but it is important to consider the depth of tissues that may be affected by the modality in relation to the tissues intended to receive benefit.



Figure 18-9 Example of a hot pack placed over a dog's hip.

Hot packs may be reusable and usually can maintain an elevated temperature for 20 to 30 minutes. The therapist should be certain that the hot pack is not too warm. A good rule of thumb is that if the hot pack is comfortable when it is placed on the back of the therapist's neck, it should be safe for the patient. Placing the therapist's hands under the hot pack and against the animal's skin is also a good way to check for appropriate temperature. To further avoid burns to the treated tissues, the therapist should place a towel between the heat source and the skin. It is very important to observe the skin's response to the treatment and whenever the skin feels very warm or hot or looks red, another layer of toweling should be added between the pack and skin. If overheated, dogs with normal sensation will try to move away from the pack but dogs with impaired sensation may not realize the area is overheating and are especially susceptible to burns. Hot packs are a relatively safe form of thermotherapy because they cool during treatment, minimizing the risk of burn.

The effects of warm 47° C (116° F) compresses were studied on epaxial muscles in dogs. The gel compress was applied for 5, 10, and 20 minutes in random order and temperature changes at 0.5, 1.0, and 1.5 cm tissue depths were measured. The compress significantly increased tissue temperature of the lumbar region (>2° C) at 0.5 and 1.0 cm depths, but heating was minimal at 1.5 cm depth. The compresses in this study were not as hot as many commercial products and the dogs were of ideal body condition making the results a good guide, but not applicable to all dogs and all hot packs.[44]

Warm Water

Warm water is another way of applying superficial heating. Water may be applied directly to the area being treated, towels heated with warm water may be used, or the affected segment may be immersed in a warm water bath or a whirlpool. If the last method is chosen, be mindful that systemic heating with a warm bath may decrease blood pressure and increase the heart rate.[34] Whirlpools have the advantage of also providing increased hydrostatic pressure to submerged body parts, which may reduce edema. Increased hydrostatic pressure helps to increase lymphatic and venous flow from a distal to proximal direction. The temperature of a whirlpool is based on the needs of the individual animal. For example, patients with chronic conditions may be treated with warmer water than patients with more acute disorders. When using an underwater treadmill in conjunction with thermotherapy, the temperature may range from approximately 27° to 35° C (80° to 95° F), depending on how vigorously the dog is exercising. The animal may be immersed in water at approximately 30° C after exercise to have a moderate heating effect, which continues to increase blood circulation and promote

the removal of lactic acid and other metabolites produced following intense exercise.[34]

Paraffin Baths

Warm, melted paraffin is another method of thermotherapy. For this application, the paraffin is mixed with mineral oil in a 6:1 or 7:1 ratio of paraffin to oil to reduce the melting temperature of the paraffin from 54° C (129° F) to between 45° and 50° C (113° to 122° F). Paraffin can safely be applied at this temperature because of its low specific heat and thermal conductivity. Paraffin is usually used for heating the distal extremities because it can maintain good contact with these irregularly contoured areas.[34] Treatment time for this method is 15 to 20 minutes. The use of paraffin baths in veterinary medicine is messy, and therefore uncommon.

Infrared Lamps

Infrared lamps emit electromagnetic radiation in the frequency range that gives off heat when absorbed by matter. The tissue temperature increase produced by infrared lamp radiation is directly proportional to the amount of radiation that penetrates the tissue. This is related to the power and wavelength of the radiation, the distance of the radiation source from the tissue, and the absorption coefficient of the tissue. Higher power infrared delivers more radiation to the skin.[15] Infrared lamps emit infrared light that penetrates the skin to promote increased blood flow and circulation. This method of thermotherapy is used to warm large areas of the body. The lamps are positioned 30 to 40 cm from the affected area. The therapist should place his or her hand under the heat lamp for several minutes at the desired height to be sure that the temperature is comfortable. Because of the dog needing to remain stationary during treatment, this method of heating is uncommon.

Treatment Duration and Frequency

The duration and frequency of thermal treatment depend on the severity of the injury, the stage of tissue healing, the area of the injured tissues, and the desired outcome. Heat treatments generally last from 15 to a maximum of 30 minutes and may be repeated three or four times daily. If white areas (due to rebound vasoconstriction) or red mottled areas appear on the skin, the treatment should be stopped.[30] The most desired effects of heat are achieved when the temperature is increased between 2° and 4° C in the tissues. Tissue temperatures greater than 45° C can be painful and cause irreversible tissue damage.[18] Hot packs have their greatest application in combination with other physical procedures. They may be applied before or even during massage, exercise, or electrical stimulation. It is recommended that heat be applied no earlier than 48 hours after an injury, because earlier application may potentiate swelling. The treatment times depend on which method is chosen.[34]

Precautions and Contraindications

Electric heating pads and infrared lamps are forms of thermotherapy that have a higher risk of burns. Electric heating pads should never be placed under an anesthetized or heavily sedated animal or one with decreased superficial skin sensation. In general animals should never be left unattended during treatment and the skin should be monitored frequently.

The patient should be protected from burns by insulating hot packs with towels and by examining the skin every few minutes. If the skin feels excessively hot, more towels should be added to the insulation layer or the hot pack should be removed and allowed to cool before reapplying. Heat therapy should be modified or abandoned if it causes pain or there is an exacerbation of pathologic signs.

When hot packs are applied to an infected site or abscess, the suppurative process is enhanced. Because of the increased circulation, more phagocytes and antibodies enter the site, which is a positive response. Too much heat may increase pain in these cases, however, and it is better to use packs that are only moderately warm.

Heat promotes tissue extensibility of joint capsules, tendons, and scar tissue and decreases joint stiffness, so precautions must be taken to not overstress these tissues because joints are less stiff and more mobile during treatment. Although thermotherapy may reduce pain of any etiology, it is not recommended as a treatment for pain caused by acute inflammation because an increase in tissue temperature may aggravate other signs of inflammation, including heat, redness, and edema.[34]

Precautions are needed for patients that are pregnant, extremely obese, have impaired circulation, poor thermal regulation, or cardiac insufficiency. Also, animals with bursitis, tendonitis, impaired circulation, or noninflammatory edema have decreased resistance to heat and should be given heat therapy with caution.[45] The natural thermoregulatory mechanisms are less efficient in very young animals. In older patients, the temperature-regulating systems may function poorly or the patient's sensitivity to pain may be impaired. The lessened cardiovascular and respiratory reserves in older animals may cause them to be less tolerant of heat.[45]

Local application of heat is contraindicated in the following situations: active bleeding, acute inflammation, thrombophlebitis, cardiac insufficiency, fever, malignancy, presence of swelling or edema, and poor body heat regulation.[34] It should be used with extra caution in cases of decreased or impaired sensation and in cases of decreased or impaired circulation in the area to be treated (to avoid overheating). If there is a wound and heat cannot be applied to this area, a hot pack may be applied to a remote area with an excellent blood supply in the hope that reflex

vasodilatation will occur at the treatment site, which may promote better wound healing.

Conclusion

Heat therapy and cryotherapy are powerful adjuncts in the rehabilitation of musculoskeletal injuries in dogs. Successful management depends on an accurate assessment of the dog's presenting problems at the beginning of each treatment session. Some effects and clinical indications for the use of cryotherapy and heat therapy are the same, but others are very different. There are some situations in which either may be used, and others in which only one would be appropriate. Careful consideration should be taken to choose the proper therapeutic modality to achieve an appropriate effect and understand how the modality chosen may facilitate or hinder a positive outcome.[45,46]

Cryotherapy and cryokinetics are most useful during the acute inflammatory stages of tissue healing. Two recent papers on cruciate repairs have demonstrated the effectiveness of cryotherapy to minimize inflammation.[39,40] Because tissue healing is initially established with relatively weak tissue strength, it is susceptible to injury from exercise that is too aggressive or from changing from cold to heat too early, thus exacerbating the inflammatory response. After new tissue has been established and the tensile strength of the tissue continues to increase with healing, the emphasis shifts to promoting a mobile scar, normal range of motion, and normal flexibility. In patients with limitations in these areas, concurrent application of superficial heat and flexibility exercises results in beneficial plastic changes to the tissues that may be relatively permanent. Superficial cold and heat modalities may be a very useful adjunct in a rehabilitation program to facilitate nature's ultimate therapeutic modality, controlled exercise.

Selection of the appropriate modality depends largely on an understanding and accurate assessment of the stage (acute inflammatory, proliferative, or tissue remodeling) of tissue healing, an accurate clinical assessment of the dog's functional abilities, the established treatment goals, and continued reevaluation as the patient's (and tissues') status changes.

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19 Therapeutic Ultrasound

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The aim of physical rehabilitation is to reestablish normal function using modalities such as heat and electrical stimulation, mobilization, and therapeutic exercise.[1] Superficial heating agents penetrate soft tissues to a depth of approximately 1 cm. Deep heating agents can elevate tissue temperatures at depths of 2 cm or more. Deep heating agents commonly used include therapeutic ultrasound (US) and diathermy. Although diathermy is an effective deep heating modality, it requires the patient to remain stationary for 15 to 20 minutes and is not practical in dogs.

Therapeutic US is considered an effective treatment modality for rehabilitating musculoskeletal conditions such as restricted range of motion (ROM) resulting from joint contracture, pain and muscle spasm, and wound healing.[2] There are advantages and disadvantages of US therapy (Box 19-1). Many protocols for the administration of US are based on tradition or extrapolated from basic science research and remain to be tested in controlled clinical trials.[3-6] This chapter is based on a review of current literature pertaining to the basic research findings (in human and dogs) and the clinical application of US therapy in dogs.

Physical Principles

Energy within a sound beam decreases as it travels through tissue, because of scatter and absorption. Scattering is the deflection of sound out of the beam when it strikes a reflecting surface. Absorption is the transfer of energy from the sound beam to the tissues. Absorption is high in tissues with high protein content[7,8] and relatively low in adipose tissue.[7,9]

For more detailed information regarding the components of US machines, piezoelectric effects, sound generation, and sound propagation through tissues, the reader is referred to other sources.[10,11]

The authors acknowledge Janet Steiss and Laurie McCauley for their work on this chapter in the previous edition.

Coupling Techniques

The therapist must first consider the site of soft tissue involvement, such as muscle, joint capsule, tendon, bursa, or scar tissue, and position the animal appropriately. Positioning is a compromise between patient comfort and access to the target tissue.

The US beam is attenuated in air and reflected at air-tissue interfaces. Consequently a coupling medium must be placed between the sound head and the skin.[12] Methods for direct coupling, immersion, and use of coupling cushions have been described for dogs.[13]

Direct coupling is preferred when the surface to be treated is relatively flat and is larger than the transducer head surface. A layer of water-soluble US gel is spread over the skin, and the sound head is placed in contact with the gel. Elimination of as much air as possible maximizes the US energy entering the tissues. If there are bony prominences in the treatment area or the surface is very small, smaller applicators (transducer heads) are preferable.

Commercially prepared water-soluble US gels are the best and most practical coupling method. The gel may be preheated. Coupling agents that are not recommended include[1] substances that may irritate or penetrate through the skin[2]; electroconductive gels, such as for electrocardiography or electromyography, which contain salts that can damage the transducer face[3]; lanolin-based compounds, which would cause heating of the transducer head with poor transmission to tissue[8]; and mineral oil,[4] which is messy to remove.[13] Over-the-counter creams and lotions should not be used because they generally do not provide effective transmission of US.

Other methods of coupling have been used. However, research results on these indirect coupling methods are inconclusive, and further investigation is warranted.[14] The underwater method was popular before smaller transducer heads became available.[8] Immersion should be considered only when the surface to be treated is so uneven that direct contact is too difficult. The part of the body to be treated, usually a distal limb, can be immersed in a container of tap

Box 19-1 Advantages and Disadvantages of Therapeutic Ultrasound

Advantages

Produces local heating of deeper tissues
Treatment time is short, approximately 10 minutes

Disadvantages

The dosage is difficult to monitor
Transducer contact with the skin may aggravate preexisting tenderness of the treated area

water at room temperature. The water and the animal's skin must be clean.[15] Water in a whirlpool is not recommended if it has been agitated because air bubbles form in the water. Deionized, degassed water is not necessary, but the water may be boiled and cooled or allowed to stand to remove air bubbles. Even so, small air bubbles tend to accumulate on the transducer face and the skin during treatment. The therapist can wipe these off quickly, but should try to minimize his or her own exposure. With regard to container type, metal containers reflect some of the US beam, which could increase the intensity in areas near the metal; rubber or plastic do not cause as much reflection. During treatment, the transducer should be held underwater 0.5 to 3.0 cm from the skin surface. The intensity may be increased by 0.5 W/cm^2 to compensate for absorption by water.[7]

Immersion has not compared favorably with direct coupling in several studies that examined the tissue temperatures achieved with these two methods. One study compared immersion to topical US gel on temperature rise at a depth of 3 cm in the gastrocnemius muscle of humans.[12] The treatment consisted of continuous US at 1.5 W/cm^2 for 10 minutes, with the sound head moved at a speed of 4 cm per second. With direct coupling, tissue temperature increased 4.8°C compared with 2.1°C with immersion. In another study the lateral epicondyle of pigs was treated with the transducer in direct contact with the skin and with the limb immersed in a water bath with the applicator held 2 cm from the skin surface. The temperature in the tendons of the extensor muscles originating at the lateral epicondyle rose into the therapeutic range (an increase of 2° or more) with direct coupling, but not with immersion.[16]

Another alternative to direct coupling is the use of a coupling cushion.[13] Placing a water-filled balloon between the transducer head and the skin, with coupling gel at the interfaces, has been described.[10] A recent in vitro study compared the transmission properties of commercial gel pads, bladder techniques, and water bath immersion, through pig skin from which the hair had been clipped.[14] Insufficient acoustic energy transmission occurred with degassed tap water in latex gloves, gel in a latex glove, degassed tap water baths, and gel-filled condoms. The authors recommended that if the direct method cannot be used because of the contour of the surface to be treated, clinicians should consider the use of commercially available gel pads. If other direct techniques are used, transmission is considerably less than with the direct coupling technique or commercial gel pads.[14]

The Effect of the Hair Coat

The hair coat of dogs treated with US presents a problem not encountered with human patients. Because US energy is absorbed by tissues with high protein content, and deflection of the US beam occurs at tissue interfaces, it would be expected that US penetration through the hair coat into underlying tissues would be poor. For horses, one author has specifically recommended clipping the hair and using adequate coupling gel or having the horse stand in water to reduce any air interphase.[17]

A study was conducted in dogs to determine temperatures in underlying tissues when delivering US through intact hair coats.[18] Despite application of a thick layer of gel on the skin, US delivered through short or long hair coats produced only minimal temperature increases in the underlying tissues, compared with therapy after the hair had been clipped, although there was considerable warming within the hair coat and on the skin (Figure 19-1).

Treatment Variables

The following list contains treatment variables that must be considered for each patient. These items should be documented in the patient's record:

1. Frequency
2. Intensity
3. Duty cycle
4. Treatment area
5. Treatment duration
6. Speed of the sound head
7. Treatment schedule

Sound waves above a frequency of 20,000 hertz (20 kHz) exceed the upper range of human hearing and are termed US. The higher the frequency, the less divergent the sound beam. Ultrasound beams are considered to be collimated, and frequencies in the megahertz (MHz) range expose a limited target area.

Frequency, not intensity, determines depth of penetration. The frequency most often used is 1 MHz because it represents a compromise between deep penetration and adequate heating. A frequency of 1 MHz frequency heats at depths between 2 and 5 cm.[19] As the frequency increases, penetration decreases. A frequency of 3.3 MHz (commonly referred to as 3 MHz) heats at depths between 0.5 and 3 cm.[8,19,20] Therefore for superficial lesions, a frequency of 3 MHz is preferred; for deeper lesions, a setting

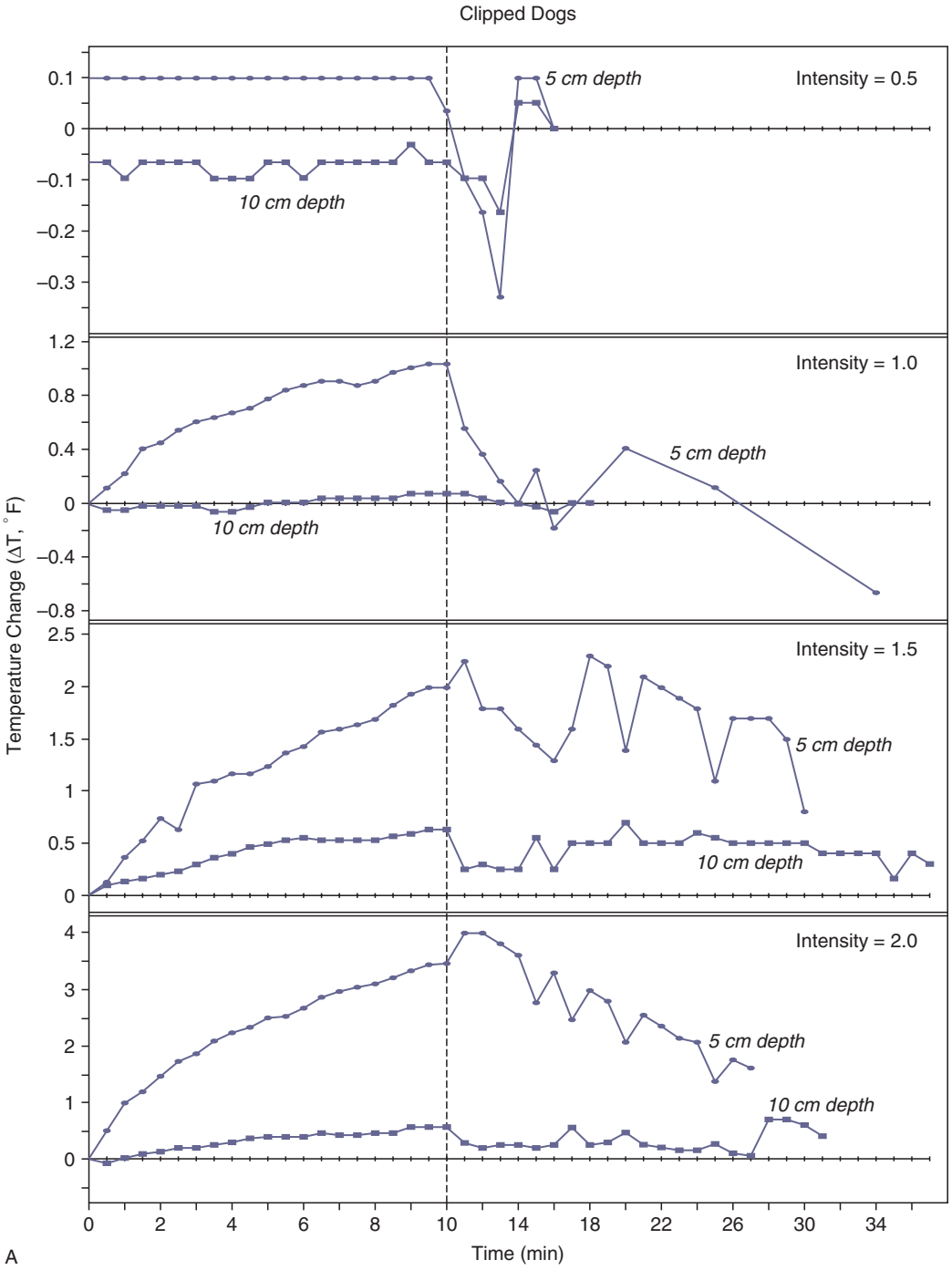
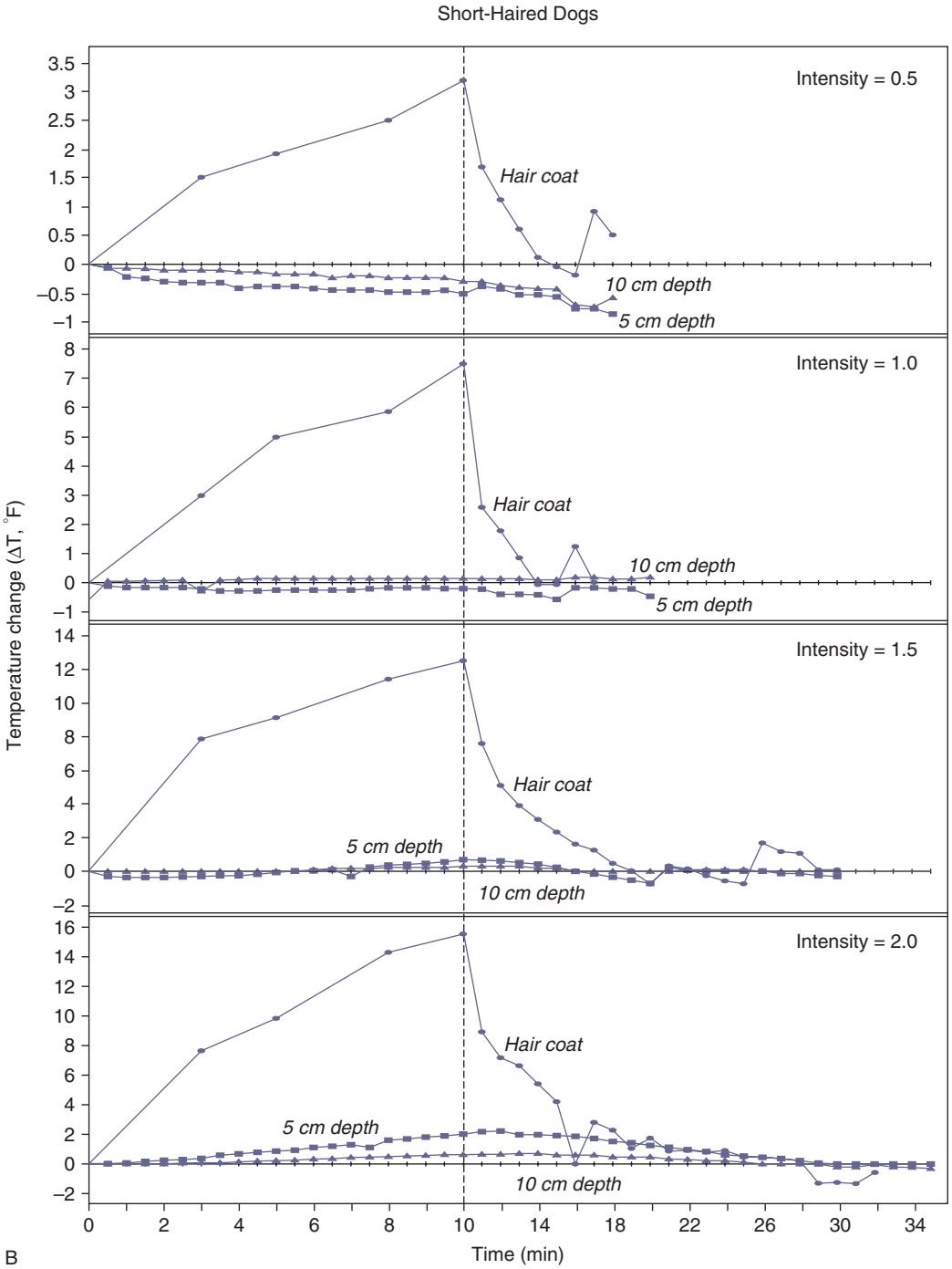


Figure 19-1 **A,** Intramuscular temperature changes measured at 5- and 10-cm depths during 1-MHz ultrasound at four intensities (W/cm^2) applied to clipped skin over the caudal thigh in dogs. The vertical dashed line represents the end of 10 minutes of ultrasound exposure. The units on the y-axis differ for each intensity.



B

Figure 19-1, cont'd **B,** Temperature changes measured intramuscularly at 5- and 10-cm depths and within the hair coat, during 1-MHz ultrasound at four intensities (W/cm^2) applied over the caudal thigh in dogs with short hair coats (greyhounds). The vertical dashed line represents the end of 10 minutes of ultrasound exposure. The units on the y-axis differ for each intensity. (From Steiss JE, Adams CC: Effect of coat on rate of temperature increase in muscle during ultrasound treatment of dogs. *Am J Vet Res* 60:76-80, 1999.)

of 1 MHz is needed.[8] If there is underlying bone, most of the US transmitted to that depth will be absorbed by the bone and may cause periosteal pain.[19-21] The intensity should not be turned up very high to heat the underlying soft tissue if the bone is not adequately covered by soft tissues.[19] Low frequency or "long wave" US machines have also been investigated.[22] These units emit US at a lower frequency, around 45 kHz. In an in vitro study using fibroblasts, osteoblasts, and monocytes, both 45 kHz and 1 MHz exposure enhanced cell proliferation, protein synthesis, and cytokine production.[22]

Intensity refers to the rate of energy delivery per unit area. Most US machines indicate both watts (W) and watts per centimeter squared (W/cm^2). Available intensities typically range from 0.25 to 3.0 W/cm^2. Measurement of the total output (W) of the transducer divided by the transducer surface area (cm^2) is referred to as the spatial average intensity. The greatest intensity anywhere within the beam is termed the spatial peak intensity. The beam nonuniformity ratio (BNR) compares the maximal intensity from the transducer to the average intensity. This ratio should be low (between 2 and 6) to indicate that the energy distribution is relatively uniform, with minimal risk of tissue damage from areas of concentrated US energy ("hot spots"). This risk is minimized when the therapist keeps the US transducer moving during treatment.

The higher the intensity, the greater and faster the temperature increases. The depth of tissue penetration is not affected to a large degree. Generally intensities required to increase tissue temperature 2° C or more vary from 1 to 2 W/cm^2 continuous wave US for 5 to 10 minutes. To heat an area with substantial soft tissue, intensities as high as 2 W/cm^2 may be used. If there is less soft tissue, or if bone is close to the skin surface, lower intensity and higher frequency are appropriate. After selection of an initial intensity, the patient's tolerance to the heat produced by the US is the final determinant of intensity.[7] This may be difficult to determine in pets. Many dogs lie quietly during US treatment. However, dogs sometimes begin to whine or otherwise seem uncomfortable within 5 to 10 minutes after commencing US. It should be assumed that any distress demonstrated by a patient may indicate pain, and the intensity of the US should be reduced or the session interrupted or discontinued.

Most therapists tend to use intensities that produce no detectable sensation in human patients. However, some clinicians advise that the intensity be increased until the person feels vigorous heating, after which time the intensity should be decreased if the patient experiences pain.[19] This is impractical in dogs. The US dosage that a patient is receiving usually cannot be monitored. In the past, in an effort to avoid tissue damage, the dosages have been low and the patient experiences no sensation. However, there is the possibility that the patient is not receiving a sufficient dose of US energy. More recent recommendations seem to address this issue.

When the treatment goal is to heat periarticular structures, as is done when treating limited ROM, higher intensities are chosen. Lower intensities may be used for the relief of pain and muscle spasm, in which the mechanism of action may be nonthermal.

Duty cycle refers to the fraction of time that the US is emitted during one pulse period. With continuous mode the US intensity is constant, whereas a pulsed wave is interrupted so that energy is delivered in an on/off manner.

$$\text{Duty cycle} = \frac{\text{Pulse duration (time on)}}{\text{Pulse period (time on + time off)}}$$

Typical duty cycles for pulsed mode US range from 0.05 (5%) to 0.5 (50%). For pulsed mode, the terms temporal peak intensity and pulse average intensity are used. Obviously pulsing decreases the pulse average intensity and decreases tissue heating. Pulsing may be used when the desired effect is based on a nonthermal mechanism or when minimal heating is desired, such as treating near bone.[8] Low-intensity continuous mode could also be chosen for these purposes.

The treatment area should be two to four times the size of the effective radiating area of the transducer head.[19] Increasing the total area beyond the recommended area decreases the dosage and the heating effect.

Experimental evidence indicates that a duration of 5 to 10 minutes is necessary to produce adequate tissue heating in an area equivalent to two to three times the diameter of the sound head.[21,23] For instance when using direct coupling with topical US gel, continuous mode at 1.5 W/cm^2 for 10 minutes, and moving the sound head at 4 cm per second, it took nearly 8 minutes for the temperature to reach therapeutic levels when temperature was measured at a depth of 3 cm in the gastrocnemius muscle of humans.[12] For an intensity of 1.5 W/cm^2 Draper recommends a duration of approximately 5 minutes at 3 MHz and at least 10 minutes at 1 MHz, assuming that an area twice the size of the effective radiating area of the transducer is treated.[19] The findings in dogs (see Figure 19-1, *A*) support these recommendations.

The recommended speed at which the sound head is moved over the skin is approximately 4 cm per second, or slower, to achieve uniform distribution of energy to the target tissues.[10] Moving the transducer too quickly diminishes the heating and the therapist may have a tendency to cover too large an area. Various longitudinal and circular patterns may be used. The transducer should never be stationary when direct coupling is used. Because the US beam is nonuniform, some target areas within the beam could receive a large amount of energy. This predisposes target

areas to hot spots and tissue damage, including endothelial damage and platelet aggregation. Keeping the transducer moving is not as critical with underwater applications because hot spots occur in the near field of the beam. Hot spots are situated in the water if a sufficient distance is maintained from the tissue. However, the distance may compromise the heating effect. Problems with achieving adequate tissue heating with the immersion technique have been previously described.

Treatment schedules may include daily treatment initially, followed by less frequent sessions as the condition improves.[7] Bromiley[8] states that treatment can be given daily for up to 10 days, but should not exceed two 10-day courses without a 3-week rest. Similar recommendations have been made for horses.[17] However, the scientific rationale for these recommendations is not clear.

Precautions and Contraindications

Tissue burns are a major concern of treatment.[13] These may occur if the intensity is too high, treatment times are too long, or the transducer is held stationary, thereby concentrating energy in a small area. These same factors may put the patient at risk for cavitation, a phenomenon in which bubbles of dissolved gas form and grow during each rarefaction phase.[10] Also if the transducer is inadvertently held in the air while emitting US, the face of the transducer may overheat. The transducer may become damaged, and the animal could be burned if the overheated surface of the transducer contacts the skin. Some units have a built-in system to prevent the crystal from overheating. Regarding safety of the therapist, some US energy is unavoidably propagated through the housing of the sound head to the hand of the therapist, but the effects of this "parasitic" exposure are unknown.

Avoid direct US exposure to the following:

- Cardiac pacemakers.
- Carotid sinus or cervical ganglia. Ultrasound could alter the normal pacing of the heart or stimulate baroreceptors.[7]
- Eyes. Because blood supply to the lens is poor, heat is poorly dissipated and could result in cataracts. Retinal damage could also result.[10]
- Gravid uterus.
- Heart. Electrocardiographic changes have occurred in dogs.
- Injured areas immediately after exercise.[15,17]
- Malignancy. Ultrasound applied to murine tumors resulted in larger tumors compared with controls.[24]
- Spinal cord if a laminectomy has been performed.[7] However, US therapy has been advocated in the area around the spinous processes in horses[25] and would also be acceptable in dogs if the bone is intact.
- Testes. Temporary sterility could be associated with heating.
- Wounds that are contaminated. Ultrasound could drive bacteria into the tissues[26]; infectious processes may be accelerated by heat.[7]
- Recent incision sites. It has been recommended not to treat incision sites with continuous US for the first 14 days to avoid dehiscence.[17]
- **Exert precaution in the following situations:**
- Bone fracture. This is controversial. Concern exists that high doses retard callus formation, delay calcification,[15] or cause pathologic fractures, subperiosteal damage, or demineralization.[26] Others believe that US over fracture sites is acceptable unless sensation is impaired.[9] Experimentally, low-intensity pulsed US may accelerate fracture healing.[10]
- Bony prominences. Treat around bony prominences rather than directly over them, to avoid concentrating energy at the periosteum, or use an immersion technique. Treat bony areas with low dosage levels to avoid the danger of pathologic fractures.[8]
- Cold packs or ice before US. Cold alters pain and temperature perception, limiting the patient's ability to respond if the US intensity is too high.
- Decreased blood circulation. Normal blood flow dissipates the heat that is generated.
- Decreased pain and temperature sensation. Pain response serves as a sensitive monitor of excessively high intensities or faulty equipment[17] that could cause burns or other tissue damage.
- Animals that are overly sedated, restrained, or under local anesthesia.
- Physeal areas in immature animals.[8,10,15] High intensities could affect physes and bone growth.
- Injuries in the acute stage that should not receive heat therapy.[17]
- Acute inflammatory joint disease. Intracapsular heating may accelerate destruction of articular cartilage in acute inflammatory joint disease.[7]

Note that metal implants are not necessarily a contraindication for US therapy. The effects of US on cementing compounds such as methylmethacrylate are unknown.[10]

Equipment Safety

Ultrasound equipment falls under the rules and regulations of the FDA's radiation safety performance standards.[15] Verification of such features as output accuracy and timer accuracy, as well as safety relating to the electrical components, is advised semiannually.[10] Ultrasound power meters are available for testing. The manufacturer establishes the BNR of a transducer. Most equipment problems originate in the transducer or cable assembly. Recalibration is necessary when a transducer is replaced, and yearly

calibration by the manufacturer is recommended even if the transducer is not replaced. Some machines feature self-calibration tests, output power control in response to tissue loading, and automatic shutoff in case of transducer overheating.

Thermal Effects

Early applications of therapeutic US were those for which tissue heating was the goal.[26] More recently, attention also has been drawn to low-intensity US, which appears to stimulate physiologic processes, such as tissue repair, without significant tissue heating.[27]

The thermal effect is a major indication for the use of this modality. Increased tissue temperatures may increase collagen extensibility, blood flow, pain threshold, and enzyme activity, as well as mild inflammatory reactions, and changes in nerve conduction velocity. Heating is thought to alter the viscoelastic properties of collagen and collagen molecular bonding. Changes in blood flow in response to US have been measured with variable results.[28,29] It is likely that with treatment for 10 to 20 minutes at high intensities, skeletal muscle temperature and blood flow increase.[10] Inconsistent changes have been found with lesser intensities and durations.

The degree of heat production depends on the intensity, frequency, duration, and size of the treatment area. In addition, the tissue type must be considered. Ultrasound penetrates skin and subcutaneous fat relatively well, with little attenuation, whereas tissues with high collagen content absorb more of the US energy and are affected to a greater extent. The order of tissue attenuation, from least to most, is as follows: blood (approximately 3% attenuation), fat, muscle, blood vessels, skin, tendon, cartilage, and bone (approximately 96% attenuation).[10]

To achieve the thermal effects of US, the tissue temperature must be raised 1° to 4° C, depending on the desired outcome. Obviously this is difficult to judge because thermistors are not inserted in clinical patients to monitor the temperature. Experimental studies in dogs and humans can provide guidelines. In healthy adult people thermistors were inserted into muscle at depths of 2.5 and 5.0 cm for 1 MHz treatment, and at depths of 0.8 and 1.6 cm for 3 MHz treatment.[30] The rate of temperature increase per minute at the two depths for the 1 MHz frequency ranged from 0.04° C at 0.5 W/cm^2 to 0.38° C at 2.0 W/cm^2, whereas the corresponding values for the 3 MHz frequency ranged from 0.3° C at 0.5 W/cm^2 to 1.4° C at 2.0 W/cm^2. The 3 MHz frequency heated significantly faster than 1 MHz at all doses.

One method for treating humans consists of applying ice on the treatment area before US. Adding ice to the water for US delivered underwater has been recommended for horses also.[25] However, if the aim is to increase tissue temperature, then it appears that US preceded by ice treatment yields little benefit. Experimentally, US alone increased tissue temperature an average of 4° C, whereas US preceded by 5 minutes of ice increased tissue temperature only 1.8° C.[2]

One prospective randomized experimental study examined the tissue temperature changes that occur at various depths during 3.3 MHz US treatments of the caudal thigh muscles in dogs.[20] Dogs received two US treatments (selected randomly), one at an intensity of 1.0 W/cm^2 and one at 1.5 W/cm^2. Needle thermistors were inserted in the caudal thigh muscles below the skin surface at depths of 1, 2, and 3 cm, directly under the US treatment area. Both intensities of US treatment were performed on each dog over a 10-cm^2 area for 10 minutes using a sound head with an effective radiating area of 5 cm^2. Tissue temperature was measured before, during, and after US treatment until tissue temperature returned to baseline. At the completion of the 10-minute US treatment the temperature rise at an intensity of 1.0 W/cm^2 was 3° C at the 1-cm depth, 2.3° C at 2 cm, and 1.6° C at 3.0 cm (Figure 19-2, *A*). At an intensity of 1.5 W/cm^2 tissue temperatures rose 4.6° C at the 1.0-cm depth, 3.6° C at 2 cm, and 2.4° C at 3 cm (Figure 19-2, *B*). Tissue temperatures returned to baseline within 10 minutes after treatment in all dogs. This study demonstrated that significant heating occurs in the superficial thigh muscles of dogs during 3.3 MHz US, but the effect is relatively short-lived.

Increasing tendon extensibility with the use of US was also studied in dogs.[31] This study evaluated tendon temperature of the calcaneal tendon following US treatment, and quantified hock flexion following US treatment of the tendon. Dogs were anesthetized and the calcaneal tendon region was clipped free of hair. One leg was used to measure temperature increase of the tendon with four modes of US: continuous and pulsed mode US, at both 1.5 W/cm^2 and 1.0 W/cm^2. The other leg was used to measure the change in hock flexion using a standard amount of force, pre- and post-US treatment of the calcaneal tendon using 1.5 W/cm^2 continuous-mode US. Continuous-mode US resulted in significantly greater tendon heating than pulsed US ($P < .001$). In addition, 1.5 W/cm^2 continuous US resulted in significantly greater tendon heating than 1 W/cm^2 continuous US ($P < .001$). Mild increases in tendon temperature occurred with pulsed mode US (all < 1.5° C), but there were no differences between 1 and 1.5 W/cm^2 pulsed mode US. The pattern of heating appears to be different between tendon and muscle. Muscle appears to have a relatively steady increase in tissue temperature during US treatment, whereas tendon temperature in this study increased relatively rapidly within the first 2 minutes, and then stayed relatively constant for the remaining 8 minutes of treatment time. Continuous mode 1.5 W/cm^2 US

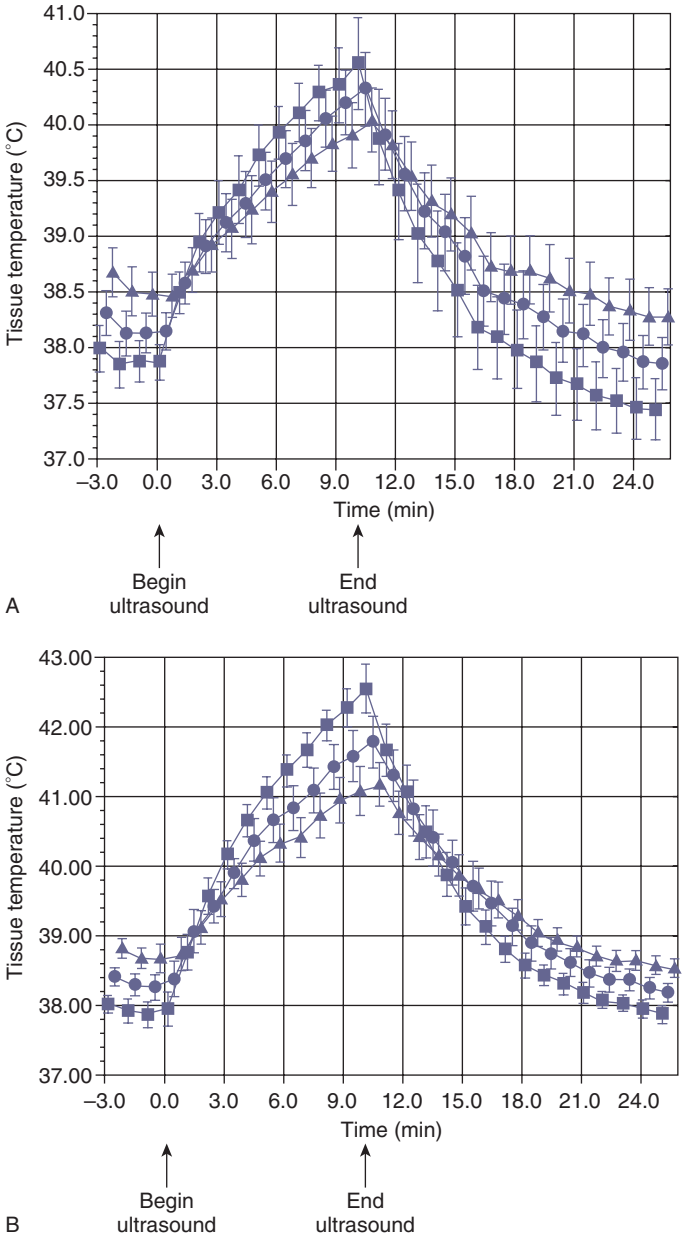


Figure 19-2 **A,** Baseline, US treatment, and posttreatment tissue temperatures for 1.0 W/cm^2, 3.3-MHz ultrasound at 1-, 2-, and 3-cm tissue depths. Values are means ± SEM. **B,** Baseline, US treatment, and posttreatment tissue temperatures for 1.5 W/cm^2, 3.3-MHz ultrasound at 1-, 2-, and 3-cm tissue depths. Values are means ± SEM. (From Levine D, Millis DL, Mynatt T: Effects of 3.3 MHz ultrasound on caudal thigh muscle temperature in dogs. *Vet Surg* 30:170, 2001.)

treatment of the calcaneal tendon resulted in significantly increased hock flexion immediately after treatment, but by 5 minutes post-US, hock flexion returned to near baseline.

Clinical Conditions Treated with Ultrasound

The main indications for US therapy include the following:

- Soft tissue shortening (e.g., contracture, scarring)
- Subacute and chronic inflammation
- Pain (such as muscle guarding, trigger points)

In human athletes, some experts consider that the most beneficial results from US are in treating tendinitis.[19] In chronic tendinitis, recommended therapy includes heating with US, followed by cross-frictional massage, to eliminate scar tissue and increase ROM. Another indication is the treatment of limited ROM associated with joint contracture, for which patients receive US before passive

ROM, stretching, or joint mobilization. A third indication is for pain relief before activity, such as in athletes with tendinitis that is mild enough to allow the person to continue training. The US treatment is administered before activity to assist in the warm-up and provide some pain relief.

In horses, US therapy has been suggested for the treatment of similar conditions. These include tendinitis, desmitis, sprains, joint lesions, lacerations, reduction of scar tissue, edema, exostosis, and myositis.[17]

Tendinitis and Bursitis

Tendinitis and bursitis are treated with US because of the ability to increase blood flow to aid healing, increase tissue temperature to reduce pain, and drive antiinflammatory drugs across the skin by means of phonophoresis. In humans lateral epicondylitis (tennis elbow), subacromial bursitis, and bicipital tendinitis are typical conditions considered for US therapy. From animal studies it appears that the stage at which US is given in the course of healing is important. Application of US in the early phase of tendon repair could be detrimental.[10]

Joint Contracture and Scar Tissue

Ultrasound is often included in protocols for treating joint contractures and scar tissue.[10] Joint contracture typically results from immobilization or trauma. The periarticular connective tissues have high collagen content, and if tissues are not mobilized, they tend to shorten to their resting, immobilized length. The principle of "heat and stretch" can be applied in this situation in an effort to increase ROM. Tissues are heated to approximately 45° C, followed by gentle passive or active stretching. There tends to be a greater residual increase in tissue length with either preheating or simultaneous heating. The effects of heat on ligament extensibility have been studied in humans.[32] Young adults underwent knee joint displacement tests before and after continuous-mode US (1 MHz, 1.5 W/cm^2 for 8 minutes). The investigators found small increases (1.3 degrees) in varus/valgus excursion at 0- and 20-degree knee flexion and in recurvatum excursion, but not in anterior-posterior drawer excursion. Their conclusion was that continuous-mode US at commonly used intensities made some knee ligaments only slightly more extensible in normal subjects. Scar tissue is denser than surrounding tissues, so it may be selectively heated by US. However, more research is needed to determine which intensities and durations are most beneficial. Figure 19-3 demonstrates US treatment over the hip before stretching in a patient who has undergone a femoral head and neck ostectomy.

Pain and Muscle Spasm

Pain threshold is usually increased after US. Diathermy and infrared therapy have a similar effect.[10] Although the physiologic mechanism underlying pain reduction remains speculative, the probable mechanism of action is the ability of all three methods to elevate tissue temperature. Heating may elevate the activation threshold of free nerve endings, produce counter-irritation, or activate large-diameter nerve fibers.[10] Peripheral nerve conduction velocities are either increased or decreased after US, partially depending on the intensity. Types A, B, and C nerve fibers have different sensitivities.[8] With intensities of 1 to 2 W/cm^2 US reduces the nerve conduction velocity of pain-carrying C fibers. This may help to reduce muscle spasm and reestablish circulation. Ultrasound also has been used to treat pain associated with neuromas, low-back dysfunction, and skeletal muscle spasm. The mechanism of action for reduction of skeletal muscle spasm may be based on thermal effects that alter the skeletal muscle contractile process, reduce muscle spindle activity, or break the pain-spasm-pain cycle.[10]



Figure 19-3 Ultrasound applied over the hip joint before stretching in a patient with a femoral head ostectomy.

Nonthermal Effects of Ultrasound to Enhance Tissue Repair

There are many situations in which US produces biologic effects and yet significant temperature change is not involved. It is not strictly true to talk about "nonthermal" mechanisms, in that the delivery and absorption of energy in the tissues will result in a small temperature rise. The term *nonthermal* in this context relates to the fact that there is no apparent thermal accumulation in the tissues, and is sometimes now referred to as a *microthermal* effect.[33] The physical mechanisms thought to be involved in producing these nonthermal effects include cavitation and acoustic streaming.[27,34]

There is evidence indicating that nonthermal mechanisms play a primary role in producing a therapeutically significant effect, and the focus of this section relates to

these effects in relation to soft tissue injuries because these are the most commonly treated conditions in practice.

Ultrasound in Soft Tissue Repair

The process of tissue repair involves a complex series of cascaded, cellular and chemically mediated events that lead to the production of scar tissue that constitutes an effective material to restore the continuity of the damaged tissue. The process is more complex than can be described in this chapter, but has been extensively reviewed elsewhere.[35]

The effect of US during the repair process varies according to the primary events that are occurring in the tissues. Immediately following injury, during the tissue hemorrhage phase, it is generally inadvisable to use US because if it has the capacity to enhance local blood flow, and there may be a clinical disadvantage of treatment while hemorrhage is occurring. After active bleeding has stopped (usually a matter of hours after injury or trauma), it is appropriate to start using US as soon as it is feasible. During the inflammatory phase, US has a stimulating effect on the mast cells, platelets, neutrophils, and macrophages.[27,36-38] For example, the application of US induces the degranulation of mast cells, causing the release of arachidonic acid, which is a precursor for the synthesis of prostaglandins and leukotrienes that act as inflammatory mediators[36,39-41] By increasing the activity of these cells the overall influence of therapeutic US is proinflammatory rather than antiinflammatory. The benefit of this mode of action is not to "increase" the inflammatory response as such (although if applied with too great an intensity at this stage, it is a possible outcome),[41] but rather to act as an "inflammatory optimizer" by speeding the process up. The inflammatory response is essential to the effective repair of tissue, and inhibition of these events serves to inhibit the repair phases that follow.[35,42-44] The more efficiently the inflammatory events can be completed, the more effectively the tissue can progress to the proliferative phase of repair. Studies that have tried to demonstrate the antiinflammatory effect of US have largely failed to do so.[45,46] It is effective at promoting the normal inflammatory events, and as such has a therapeutic value in promoting the overall repair events.[27,33,47,48]

Employed at an appropriate treatment dose with optimal treatment parameters, the benefit of US is to make the inflammatory phase as efficient as possible, and thus promote the entire healing cascade.[7,10,35,49] For tissues in which there is an inflammatory reaction, but in which there is no "repair" to be achieved, the benefit of US is to promote the normal resolution of the inflammatory events, and hence resolve the "problem." This is most effectively achieved in the tissues that preferentially absorb the energy, which are the dense collagenous tissues (ligament, tendon, fascia, joint capsule, and scar tissue). During the proliferative phase of wound healing US also has a stimulatory effect (cellular upregulation), although the primary active targets are the fibroblasts, endothelial cells, and myofibroblasts.[36,38,39,50-54] These are all cells that are normally active during scar production and US is therefore pro-proliferative in the same way that it is proinflammatory—it does not change the normal proliferative phase, but maximizes its efficiency. Harvey et al.[55] demonstrated that low-dose pulsed US increased protein synthesis, and several research groups have demonstrated enhanced fibroplasia and collagen synthesis.[50,56-60]

During the remodeling phase of repair, the somewhat generic scar that is produced in the initial stages is refined such that it adopts functional characteristics of the tissue that it is repairing.[35] This is achieved by a number of processes, but is mainly related to the reorientation of the collagen fibers in the developing scar and also to the change in collagen type, from predominantly Type III collagen to a more dominant Type I collagen.[61] The remodeling process is certainly not a short-duration phase (it can last for a year or more), yet it is an essential component of quality repair and the application of US appears to enhance the events normally associated with this phase.

Ultrasound can influence the remodeling of scar tissue in that it appears to enhance the appropriate orientation of the newly formed collagen fibers[62] and change the collagen profile from mainly type III to a more dominant type I content, thus increasing tensile strength and enhancing scar mobility.[53] Other studies have demonstrated effects of US therapy on collagen behavior in this latter repair stage.[63-66] Ultrasound applied to scar tissue may enhance its function.[53,60]

The application of US during the inflammatory, proliferative, and repair phases is of value not because it changes the normal sequence of events, but because it has the capacity to stimulate or enhance these normal events and thus increase the efficiency of the repair phases.[27,33,47] It appears that if tissue repair occurs in a compromised or inhibited fashion, the application of therapeutic US at an appropriate dose may enhance healing. If the tissue is healing "normally," the application may speed the process and thus enable the tissue to reach its end point faster than it would otherwise. The effective application of US to achieve these aims may be dose dependent,[49,64,67] although some authors have challenged this thought.[68]

Soft Tissue Wound and Fracture Healing

The use of therapeutic US in wound healing recently has been reviewed (Figure 19-4).[69] Reher et al.[70] suggested that the beneficial effects of US on wound healing, chronic ulcers, and fracture healing might be based on enhanced angiogenesis. Therapeutic US stimulates production of angiogenic factors such as interleukin-8, fibroblast growth factor, and vascular endothelial growth factor. These effects

were noted with both 1-MHz and 45-kHz exposure. More research is needed to establish the mechanism of action of US at different stages of healing and with different treatment parameters to optimize its effects.

Results may depend on variables such as the intensity and duration of treatment and the timing of application after injury. Low-intensity continuous- or pulsed-mode US has been used to treat acute and chronic wounds. Low intensities appear to enhance healing, whereas high intensities may have proinflammatory effects. Regarding the stage of healing, US therapy initiated while hemorrhage is still occurring may compromise repair, whereas US initiated after this may be beneficial.

Dyson et al.[71] reported that US enhanced growth of tissue in experimental wounds in rabbit ears. Because the temperature increases were small, the investigators suggested that the mechanism may have involved acoustic streaming. Ultrasound has also been used to treat chronic skin ulcers. Rantanen et al.[72] used a rat model of muscle contusion injury to study the effect of therapeutic US on muscle regeneration. They concluded that therapeutic US promoted the early phase of muscle regeneration, namely, the proliferation of satellite cells, but US did not seem to significantly alter the final outcome. Low-intensity US may also have a role in the healing of fractures. Numerous animal studies and controlled clinical trials have shown that low-intensity US can promote the healing of fresh fractures. Some evidence suggests efficacy in the treatment of delayed healing and nonunion fractures as well.[73-75]

Other Conditions

There was significant improvement after US treatment in people with mild to moderate idiopathic carpal tunnel syndrome.[76] Patients received 20 US sessions (1 MHz, 1 W/cm^2, pulsed mode 1:4, 15 minutes per session) applied to the area over the carpal tunnel, with the first 10 treatments performed daily and 10 additional treatments performed twice weekly for 5 weeks. The improvements were sustained 6 months after treatment. It remains to be determined if any forms of nerve compression or entrapment in dogs would benefit from a similar protocol.



Figure 19-4 Ultrasound applied to a wound on the lateral aspect of the stifle joint through a stand-off gel pad.

Ultrasound treatment may be useful in the treatment of soft tissue calcification. In a randomized, double-blind comparison of US- and sham-treated human patients with symptomatic calcific tendinitis of the shoulder, US had a positive effect in resolving calcifications and was associated with short-term clinical improvement.[77] Patients in that study received 24 sessions of pulsed US over a 6-week period. The way in which US stimulates resorption of calcium deposits has not been established. Bromiley[8] has suggested that the excitation of calcium bound to proteins promotes the fragmentation and resorption of calcified masses within soft tissue. Ebenbichler et al.[77] speculated that US may stimulate resorption of calcium deposits by (1) activating endothelial cells that subsequently release substances such as cytokines and interleukins that aid in resorption, (2) increasing intracellular calcium levels, (3) at high intensities, causing disruption of apatite-like microcrystals, and (4) causing increased temperatures that may lead to increased blood flow and metabolism, thereby facilitating disintegration of calcium deposits.

Ultrasound may also have a role in reduction of swelling. The mechanism has been attributed to acoustic streaming.[26] For this purpose pulsed US may be combined with cold therapy.

Dosage

Dosage for pulsed US is variable based on the tissue and the stage of healing. In general, 20% pulsed US with an intensity of between 0.1-1.0 W/cm^2 is used.[35,37]

Phonophoresis

The use of US to enhance the delivery of topically applied medications through intact skin is termed *phonophoresis*.[78] The target for the drug may be local (antiinflammatory or analgesic) or systemic. Some of the local conditions for which phonophoresis is used in humans and that parallel conditions in animals are tendinitis, bursitis, chronic sprains, rheumatoid arthritis, and osteoarthritis (OA).[79] Systemic conditions that may be treated by phonophoresis include motion sickness and high blood pressure. Although many studies have been conducted on humans and research animals to evaluate the penetration of various drugs through the skin with the assistance of US, few studies have been performed on companion animals with clinical conditions. Therefore more research is needed to better define the benefits and limitations of this rehabilitation modality.

When a drug is applied topically it may transfer through the skin by passive diffusion.[80,81] This may be hastened by denuding the epidermis to create minor abrasions, such as in shaving. Phonophoresis enhances transdermal drug delivery by altering the stratum corneum, which is the major barrier to drug penetration.[82,83] The mechanism of action may be that US denatures the structural keratin proteins in the stratum corneum, strips or delaminates the cornified layers of the stratum corneum, changes cell permeability, or alters the lipid-enriched intercellular structures between the cells.[78] After the stratum corneum is crossed the pharmaceutical molecules penetrate the dermis, are absorbed into the tissues and capillaries, and travel throughout the circulatory system.[80] In animals, shaving and scrubbing the skin with alcohol increase the rate of diffusion.[78] Drug penetration may occur transcellularly or intercellularly, but it occurs most readily through the hair follicles and sebaceous glands.[84] Although heating the skin before treatment dilates the hair follicles to enhance penetration, it also causes vasodilatation that enhances systemic absorption and decreases local delivery. Low-intensity, longer duration US appears to be more effective at transferring medication than high-intensity, shorter duration doses.[85] Higher concentrations of drugs applied topically also may increase tissue concentrations.[79,86]

Another variable to consider is the transmission of US energy through the drug and coupling agent. Some compounds (with such active ingredients as diethylamine salicylate, hydrocortisone, alclometasone dipropionate, and fluclorolone acetonide) are used to decrease soft tissue inflammation and did not allow transmission of US at three different frequencies (0.75, 1.5, and 3 MHz) in one study.[87] Because the sound waves were unable to penetrate the drug, they were also unable to penetrate the skin. Not only could using a drug that has poor transmission properties decrease the success of phonophoresis, it may also negate the effectiveness of the US therapy. Intensity, frequency, time, drug, and coupling agent transmission may play a role in some studies of phonophoresis that were unsuccessful.[88,89]

Several studies have been performed using steroidal antiinflammatory drugs (hydrocortisone and dexamethasone) and have shown decreased pain and increased ROM in human patients with OA, periarticular arthritis, and joint or muscle pathology.[90] In a human study using hydrocortisone phonophoresis (100 mg/g of ointment) on patients with OA, tendinitis, or bursitis, there was improvement (marked decrease in pain and a substantial gain in active ROM) in 68% of patients and partial improvement (decrease in pain but some pain remaining, and a gain in active ROM with limitations still present) in 18% of patients as compared with 27% improvement and 16% partial improvement when treated with US alone.[90] In one study using dexamethasone sodium phosphate (7 g of 0.33% dexamethasone using 1 MHz at 1.5 W/cm^2) in humans, the amount of dexamethasone delivered systemically did not alter adrenal function.[91] Another study of phonophoresis in humans showed detectable blood levels of dexamethasone sodium phosphate 30 and 60 minutes after treatment, whereas none was detected in the blood of patients that had the drug applied topically with no US.[92]

When using phonophoresis on small animals, the skin should be clipped and scrubbed. The drug may be either applied topically with a gel over it or compounded in glycerol, cream, oil, gel, or water. Many researchers believe that gels are optimal because of their ability to transmit US.[92] Pulsed US is most commonly used because continuous US increases blood flow to the area and may exacerbate inflammatory conditions. One recent human study compared ketoprofen phonophoresis (both 20% pulsed and 100% or continuous US) to placebo phonophoresis in which ketoprofen gel was rubbed over the area. Biopsies were performed on both adipose and synovial tissues of the knee approximately 1 hour after phonophoresis application. No significant differences were found between groups in the adipose tissue samples. In synovial tissues, both pulsed and continuous US produced significantly higher concentrations of ketoprofen compared with topical application ($P < .012$ and $P < .028$, respectively).[93]

One of the essential benefits of phonophoresis is that it may deliver higher drug levels to the target area, decreasing systemic side effects as compared with parenteral or oral medications.[94] Additionally, US is relaxing and may have analgesic properties.[4-6]

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20 Electrical Stimulation

David Levine and Barbara Bockstahler

Terminology Used in Electrical Stimulation

Electrical stimulation (ES) is a commonly used modality in physical therapy, which is effective for many purposes, including increasing muscle strength, muscle reeducation, increasing range of motion (ROM), correction of structural abnormalities, improving muscle tone, enhancing function, pain control, accelerating wound healing, edema reduction, muscle spasm reduction, and enhancing transdermal administration of medication (iontophoresis).[1] Terminology associated with electrical stimulation can be confusing, and, in 1990, a committee of the electrophysiologic section of the American Physical Therapy Association (APTA) developed the *Standards of Electrotherapeutic Terminology*, a document created to unify and standardize the terms and definitions used by biomedical engineers, researchers, educators, and clinicians. Historically, electrical stimulators and the terminology related to them have been referred to by their specific rehabilitative application, the use of the inventor's name, or the commercial companies that produced them. Examples include galvanic current, faradic current, diadynamic current, high voltage, low voltage, low frequency, medium frequency, transcutaneous electrical nerve stimulation (TENS), electrical muscle stimulation (EMS), functional electrical stimulation (FES), Russian stimulation, and interferential stimulation. Unfortunately the multitude of names has created confusion regarding the physiologic effects and clinical results. Transcutaneous electrical nerve stimulation has been widely used to identify stimulators that modify pain, whereas neuromuscular electrical stimulation (NMES) or electrical muscle stimulation (EMS) has been identified with muscle reeducation, prevention of muscle atrophy, and enhanced joint movement. Accurate terminology dictates that almost all electrical stimulators are TENS units, as they work transcutaneously through surface electrodes to excite nerves. In the typical scenario where the muscle is innervated by a motor nerve, NMES is the appropriate terminology, and when a muscle is denervated and requires direct muscle fiber activation through electrical stimulation, the term EMS is used.

The authors would like to acknowledge Janna Johnson for her work on the previous edition.

Neuromuscular electrical stimulation, the primary focus of this chapter, is a form of clinical electrotherapy used to treat a wide variety of physiologic disorders or injuries in humans and is recognized as a promising treatment modality for similar disorders in veterinary patients. By definition, NMES is the administration of an electrical current generated by a stimulator that travels through leads to electrodes placed on the skin to depolarize the motor nerve and produce a skeletal muscle contraction. Stimulation of motor end plates with electrical current causes nerve depolarization and subsequent activation of muscle fibers.[2,3]

This chapter describes the myriad of potential clinical uses of electrical NMES; therefore this term is used throughout. In addition to the discussion of general uses of NMES, specific attention is paid to ruptured anterior cruciate ligament (ACL)/cranial cruciate ligament (CrCL) rehabilitation because of the relatively high incidence in humans and dogs and the amount of research information that is currently available on the use of NMES for knee/stifle rehabilitation. The emerging use of electrical stimulation to treat pain in the dog is also examined.

History of Electrical Stimulation

Electricity has been used therapeutically since Scribonius Largus used an electrical ray in a footbath to treat gout. Benjamin Franklin used electrical shock to treat a frozen shoulder in 1757.[3] Electrical muscle stimulation has been specifically used in physical therapy since the mid-1700s, when electrostatic generators were used to treat patients with paralysis. In 1791 Galvani used galvanic current in in vitro experiments using neuromuscular preparations. By the early 1800s Faraday had invented the faradic current generator, which is the basis for most modern muscle stimulators. Documentation of NMES to reduce the loss of muscle weight and prevent atrophy in denervated muscles occurred in the early to mid-twentieth century. Electrotherapy has since become commonplace in physical medicine for the restoration of muscle function after injuries, before patients are capable of voluntary exercise training.[4]

Basic Concepts of Electrical Stimulation

To understand the benefits that electrical stimulation may provide in veterinary practice, a basic explanation and definition of the electrical current parameters used are summarized in Box 20-1.

Electrical Current/Waveforms

Three types of currents are commonly used: (1) continuous direct current (DC) (Figure 20-1, *A*), (2) continuous alternating current (AC) (Figure 20-1, *B*), and (3) pulsed current (either AC or DC) (Figure 20-1, *C*). Continuous direct current is a unidirectional electrical current that flows for 1 second or longer; alternating current changes the direction of flow at least once every second. Continuous direct current (also called galvanic current) has been used for wound healing, iontophoresis, and in the treatment of denervated muscle. In current clinical practice it is used only for iontophoresis. Continuous alternating current is not used therapeutically, but is our typical line current (see Figure 20-1, *B*).

Pulsed current consists of a unidirectional or bidirectional flow of charges (DC or AC) that periodically stops

Box 20-1 Typical Parameters Available in Neuromuscular Electrical Stimulation Devices

Waveform: The shape of the visual representation of pulsed current on a current/time plot or voltage/time plot. Can be symmetric, asymmetric, balanced, unbalanced, biphasic, monophasic, polyphasic, and so forth.

Amplitude: The current value in a monophasic pulse or for any single phase of a biphasic pulse.

Phase/pulse duration: The duration of a phase or a pulse, usually measured in microseconds.

Pulse rate or frequency: The rate of oscillation in cycles per second, expressed as pulses/sec (pps) or hertz (Hz). Often labeled as pulse rate or pulses/sec, or frequency on stimulators.

On/off time: The amount of time the stimulator is delivering current compared with the rest period between contractions, usually measured in seconds.

Ramp: The time in seconds from when the current begins to the peak current (example: 3-second ramp up, 6-second contraction, 2-second ramp down).

Polarity: Electrode may be either the anode (+) or cathode (–). (This is not relevant when using AC.)

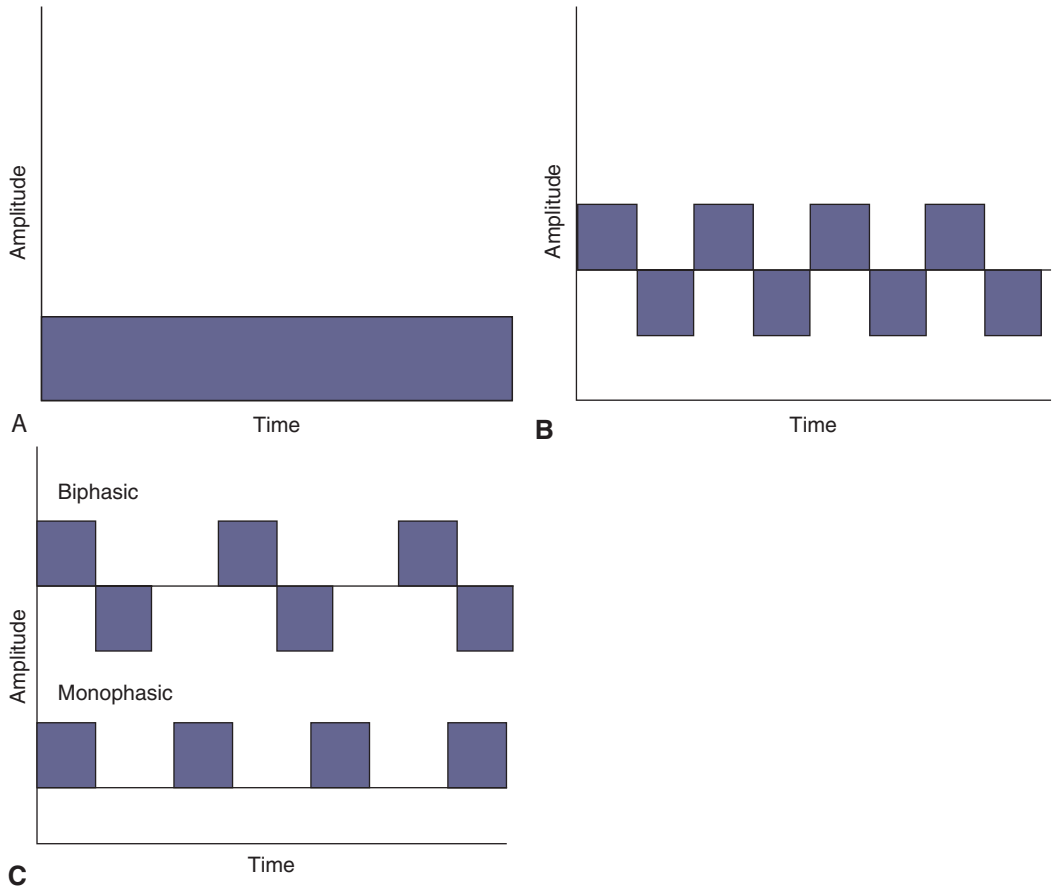


Figure 20-1 **A,** Continuous direct current. **B,** Continuous alternating current. **C,** Pulsed currents (AC and DC).

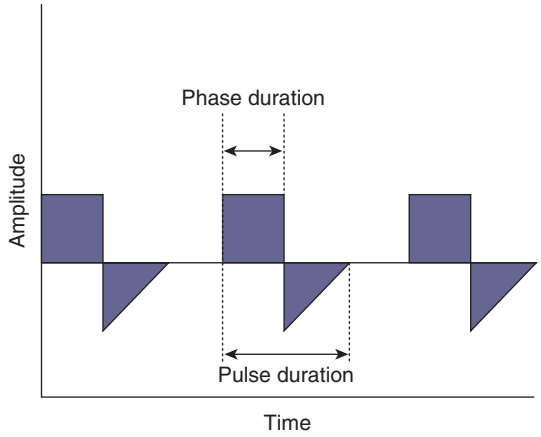


Figure 20-2 Phase and pulse duration.

for a finite time period. All NMES devices are pulsed current stimulators. Phase duration (also referred to as phase width) is defined as the time in which the current flows from the baseline in one direction and back to the baseline (Figure 20-2).

Pulse duration (also referred to as *pulse width*) is defined as the time during which charge flows in both directions (see Figure 20-2). In a monophasic current, the pulse duration and the phase duration are the same. In a biphasic current, two phases make up one pulse. Pulsed current consisting of a bidirectional flow of charge is called biphasic pulsed current.

When the flow in each direction is the same in time and amplitude-dependent features, the biphasic current pulse is considered *symmetric* (Figure 20-3, *A*). If the time- and amplitude-dependent features differ, then the biphasic current is termed *asymmetric* (Figure 20-3, *B*). Balanced asymmetric biphasic pulsed current occurs when the total charge in one phase equals the total charge of the other phase (Figure 20-4). If the total charge in each phase is unequal, it is termed *unbalanced* (Figure 20-5). Both pulsed AC and DC current forms are commonly used in portable and clinical model NMES units.

Stimulator Parameters

Current amplitude (also called magnitude or intensity) is defined as the vertical distance from the highest to the lowest peak during one electrical wave and is typically measured in milliamperes (mA) (Figure 20-6). Increasing the amplitude induces a stronger force of muscle contraction by recruiting additional muscle fibers at greater distances from the electrodes.[1,3]

Skin produces resistance to current flow by ohmic resistance and capacitive impedance. Capacitive impedance is patient dependent and cannot be modified. Clipping or shaving the hair coat and cleaning the skin with alcohol to wash away skin oils or other substances helps to lower ohmic resistance. Lowering the skin resistance diminishes the driving voltage that is necessary for current penetration

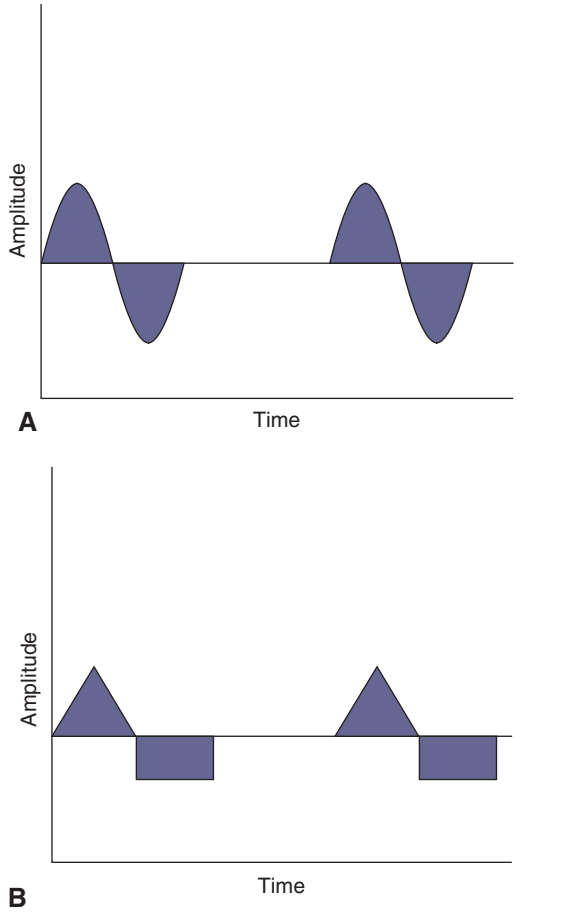


Figure 20-3 **A,** Symmetric biphasic current. **B,** Asymmetric biphasic current.

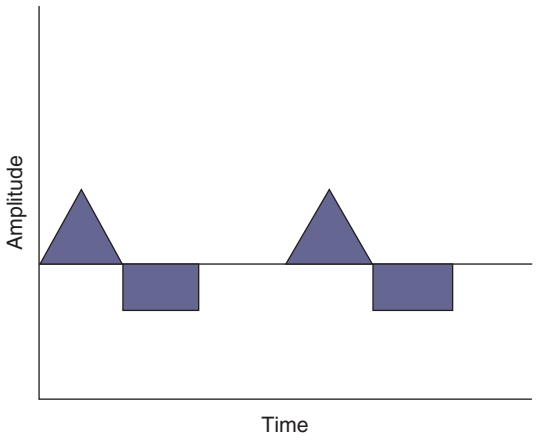


Figure 20-4 Balanced asymmetric biphasic pulsed current.

of the skin, potentially making the treatment more comfortable.[1,3]

Increased current amplitude is required to produce a given amount of muscle contraction force if the pulse or phase durations are short (<100 µsec). Portable NMES units are not always capable of producing the current

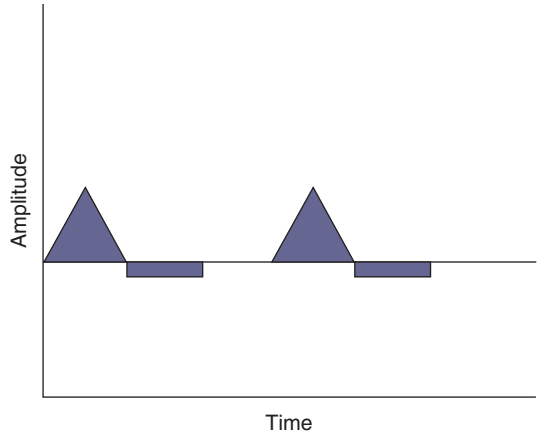


Figure 20-5 Unbalanced asymmetric biphasic pulsed current.

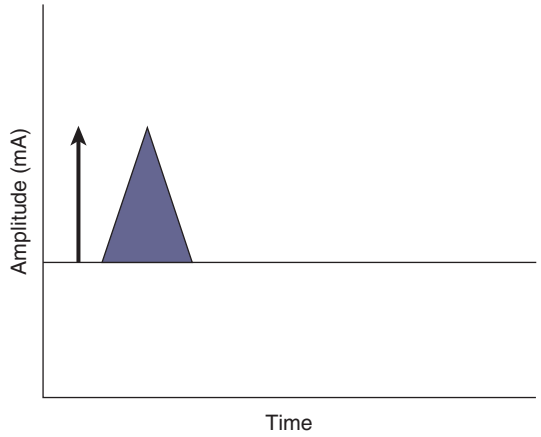


Figure 20-6 Amplitude.

output that clinical models can produce; however, most units sold for electromedical purposes are adequate. Some bargain simulators possess a very low phase charge (<10 microcoulombs) and cannot create adequate contraction of large muscle groups.

Pulse durations of 150 to 400 microseconds produce powerful contractions while minimizing the likelihood of recruiting many pain fibers.[1,3] Some stimulators that are made only for NMES do not allow control of pulse duration, but are typically set in this range. As the pulse duration increases, smaller diameter pain fibers are recruited. One study has examined pulse durations in dogs and subsequently recommended pulse durations by individual muscles (Table 20-1). This study also examined the differences in needed pulse durations between people and dogs. Although they are similar, dogs in general do not require as long a pulse duration as human motor nerves to depolarize. This can potentially increase comfort by using the shortest recommended pulse duration.[5]

Pulse rate (also called frequency, rate and pulses per second [pps]) is the number of pulses delivered per second and is measured in hertz (Hz). Tetanic muscle contractions

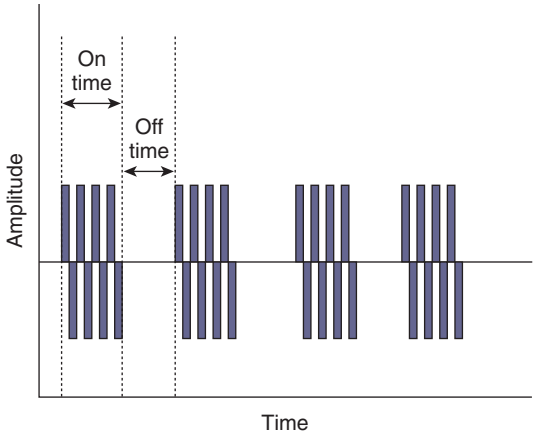


Figure 20-7 Duty cycle.

may be produced with frequencies as low as 20 Hz, but only submaximal muscle contractions are typically produced in this range. Maximal force of contraction generally occurs between 60 and 100 Hz. However, as frequency increases the rate of fatigue also increases. Lower tetanic frequencies in the range of 35 to 50 pps reduce muscle fatigue while still providing strong muscle contractions.[1,3]

Duty cycle is the ratio of on-time to total cycle time, expressed as a percentage (Figure 20-7). On-time is the period of time in which a series of pulses or bursts are delivered to the patient. Off-time is the time between on-times. A single on-time duration plus a single off-time duration constitutes the total cycle time. For example, a stimulator that causes a muscle to contract for 10 seconds followed by a 30-second rest would have a 25% duty cycle, or a 1 : 4 ratio. As on-time increases, muscle fatigue increases.[2] Optimal duty cycles vary, depending on the patient. A patient with severe atrophy may require a longer off-time to recover between contractions. Many clinicians start with duty cycle ratios between 1 : 2 and 1 : 5 and watch for signs of fatigue, which indicates the need for a longer off-time.

Ramp is a feature of NMES that helps improve patient comfort. It involves a gradual increase or decrease in current amplitude such that the number of recruited motor units gradually increases the force of muscle contraction or gradually decreases the force of contraction (Figure 20-8). The ramp time is the period of time over which the pulse is increasing or decreasing. No data to date have identified the optimal ramp time. However, a ramp up time of 2 to 4 seconds is commonly used to maximize comfort with a 1- to 2-second ramp down.[1] Suggested current parameters for muscle strengthening are listed in Box 20-2.

Electrodes

Many types of surface electrodes are available. The main criteria in choosing electrodes are as follows:

1. Flexible enough to conform to the tissue
2. May be trimmed to a specific size

Table 20-1 Recommended Pulse Durations for 11 Studied Muscles in Dogs

Muscle	Chronaxy Value (μs)	Range of Recommended Pulse Durations for Use in Dogs* (μs)	Reported Human Chronaxy Range (μs)	Range of Recommended Pulse Durations for Use in Humans (μs)
Supraspinatus	167 ± 55 (133-200)	150-190	80-160	100-300, 150-250, 250
Infraspinatus	151 ± 61 (113-188)	130-170	80-160	100-300, 150-250, 250
Deltoideus	180 ± 60 (138-221)	160-200	80-160	100-300, 150-250, 200
Triceps brachii (lateral head)	217 ± 74 (171-262)	200-240	160-320	100-300, 150-250, 200
Extensor carpi radialis	190 ± 69 (141-238)	170-210	200-360	300, 150-250, 200
Gluteus medius	179 ± 69 (136-221)	160-200	80-160	300, 250-400, 400
Biceps femoris	207 ± 63 (167-246)	180-220	440-720	300, 250-400, 400
Semitendinosus	166 ± 25 (150-181)	150-190	440-720	300, 250-400, 400
Vastus lateralis	233 ± 73 (181-282)	210-250	80-160	300, 250-400, 400
Tibialis cranialis	238 ± 111 (160-314)	210-250	200-360	300, 250-400, 400
Erector spinae	205 ± 79 (155-255)	180-220	160-320	250, 150-250, 300

*These recommended pulse durations are intervals centered over the mean values calculated in this study with an approximate range of ±10% or ±20 μs. This range facilitates the selection of a single chronaxy value when stimulating several muscles in 1 region.

From Sawaya SG, Combet D, Chanoit G, Thiébault JJ, Levine D, Marcellin-Little DJ: Assessment of impulse duration thresholds for electrical muscle stimulation (chronaxy) in dogs. *Am J Vet Res* 69:1305-1309, 2008.

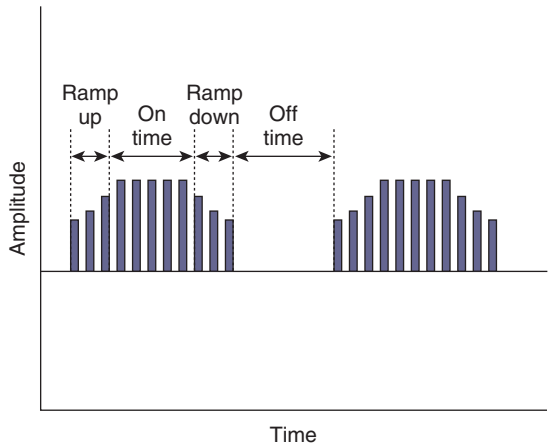


Figure 20-8 Ramp.

Box 20-2 Current Parameters for Strengthening

Frequency: Generally between 25 and 50 Hz. (These have been shown in humans to produce strong tetanic contractions while minimizing fatigue.) An alternative in dogs that are not very tolerant of electrical stimulation is to use a low frequency (<10 Hz). This will provide a twitch contraction and is better tolerated in some cases.

Waveforms: Many waveforms exist; any waveform capable of depolarizing the muscle is acceptable.

Pulse or phase duration: Between 150 and 250 microseconds.[69] Optimal pulse durations maybe found in Table 20-1.

Ramp up/down (rise and decay time): Adjust between 2 to 4 seconds up to increase comfort, 1 to 2 seconds down.

On/off time: A 1:3 or 1:4 ratio; 10 seconds on, 30 or 40 seconds off is commonly used. This may be decreased as muscle strength improves. A 1:1 or 1:2 ratio is usually used for muscle endurance training.

3. Have a low resistance
4. Are highly conductive
5. May be used many times
6. Are inexpensive

Some commercially available electrodes are effective for only a few uses, and some may be used 100 or more times (e.g., carbon-impregnated silicon rubber electrodes, metal, etc.). Conductive performance of any electrode decreases over time. Electrodes require a medium to transmit current. Commonly used media include gels, moistened sponges or paper towels; some electrodes have the media already applied. Sponges and paper towels tend to dry out, and rewetting is necessary every 30 minutes. Electrodes should be of the appropriate size to stimulate the desired muscle without stimulating unwanted muscles in close proximity. The smaller the electrode, the higher the current density that enters the muscle, and the more uncomfortable the stimulus may be.

An alternative to traditional electrodes is the use of electrodes with multiple fine wires or prongs that make contact with the skin (see Fig. 20-13). The advantage of these types of electrodes is that the hair does not need to be clipped. Rather the fine wire prongs are worked down among the hair to contact the skin. Wetting the hair before treatment helps with conductivity.

Recruitment

Neuromuscular electrical stimulators recruit Type II (fast twitch) fibers first, then Type I (slow twitch), which is the reverse of the muscle recruitment pattern in a volitional contraction.[6-8] Increasing the pulse duration increases the recruitment of smaller diameter motor units at the same depth, but increasing the pulse duration too much (800 µsec) may stimulate undesirable fibers (small-diameter pain fibers). Increasing either the amplitude or the pulse duration affects the strength of contraction because additional muscle fibers are recruited. Increasing the frequency results in the existing motor units firing at a faster rate and will increase the strength of contraction, but it also causes more rapid fatigue. Application of NMES at an optimal frequency results in an optimal physiologic response (the desired contraction) while minimizing fatigue. In a healthy uninjured individual, a maximal voluntary muscle contraction produces a greater torque (more powerful contraction) than occurs in an electrically induced contraction. However, patients with injuries or immediately following surgery may be unable or unwilling to produce a maximum voluntary muscle contraction. In these patients NMES may produce a stronger muscle contraction.

What to Look for When Buying a Neuromuscular Electrical Stimulator Unit

An acceptable NMES unit should allow flexibility in a number of parameters, including frequency (adjustable from at least 1 to 50 Hz), adjustable pulse duration (50 to 200 µsec or more), on/off times, ramp, and multiple channels (at least two to allow co-contraction or alternating contraction of two different muscle groups). Acceptable clinical units may cost $300 or more.

Indications and Contraindications for Neuromuscular Electrical Stimulation

Neuromuscular electrical stimulation is commonly used in the rehabilitation of human patients who have had orthopedic or neurologic injury. A few examples are patients recovering from fracture repair, ACL reconstruction, and meniscal debridement or repair. Patients with neurologic conditions, such as cerebrovascular accidents, closed head injuries, spinal cord injuries, or other neurologic disease involving paralysis or paresis, may also benefit from NMES. NMES has been used to increase joint mobility, decrease joint contracture, decrease edema, enhance circulation, minimize disuse atrophy, improve muscle strength, retard loss of volitional control, improve sensory awareness, decrease spasticity, diminish pain, and correct gait abnormalities.[9-12]

In human patients with low thoracic spinal cord injury, functional NMES has been used to produce knee extension with locking of the knees to allow weight bearing. Within 3 weeks of spinal cord injury, up to 50% of quadriceps muscle loss may occur. In one study NMES returned quadriceps muscle mass to near normal.[11] In a patient with an incomplete quadriplegic spinal cord injury NMES was used to strengthen a paretic hand. Two weeks of NMES produced a 33% increase in muscle force with no loss of strength 4 weeks after the treatment was discontinued.[11] Children with mild cerebral palsy treated with NMES had statistically significant improvements in gross motor, locomotor, and receipt/propulsion skills.[10] Neuromuscular electrical stimulation has also been used on the tibial muscles of children with Duchenne and Becker muscular dystrophy. Neuromuscular electrical stimulation resulted in mild, short-term increases in muscle strength, but it did not alter muscle fatigue. Whether chronic stimulation produces beneficial long-term effects has yet to be determined.[12]

In addition to the clinical use of NMES in people with neurologic and orthopedic disorders, it has been used in denervated muscle to retard atrophy and improve recovery after reinnervation in rats with surgically severed peroneal nerves. The use of NMES in improving reinnervation recovery in other species warrants further study because it may be useful to those with denervation injuries.[13]

There are a number of circumstances in which electrical stimulation should not be used (contraindications), and other circumstances in which electrical stimulation should be used only with caution (precautions). Box 20-3 outlines these contraindications and precautions.

Box 20-3 Contraindications and Precautions for the Use of Electrical Stimulation

Contraindications

High-intensity stimulation directly over the heart
In animals with pacemakers
In animals with seizure disorders
Over areas of thrombosis or thrombophlebitis
Over infected areas or neoplasms
Over the carotid sinus
Any time active motion is contraindicated
Over the trunk during pregnancy

Precautions

In areas with impaired sensation
In areas of skin irritation or damage
Near electronic sensing devices such as ECG monitors (possible interference)

Effects of Neuromuscular Electrical Stimulation in the Rehabilitation of Patients with Reconstructed Anterior Cruciate Ligaments

The specific physical effects of NMES on muscle enzymes, fibers, and perfusion has been examined experimentally in human patients with ACL injuries. The effects of NMES on dogs recovering from CrCL rupture are also beginning to be investigated.

Effect on Muscle Enzymes

In a study regarding the effects of low-frequency electrical stimulation on the metabolic profile of skeletal muscle, a portable NMES unit was used to stimulate the knee extensor muscles of sedentary men and women 6 days a week for 6 weeks.[14] No changes in creatine kinase concentrations were found in vastus lateralis muscle samples before or after NMES. There was a small decrease in phosphofructokinase, the rate-limiting enzyme of glycolysis in human muscle, which was only statistically significant in men. Hexokinase, a regulatory enzyme of skeletal muscle glucose phosphorylation, was only significantly increased in the vastus lateralis of women. Krebs' cycle and electron transfer chain marker enzyme activity of fatty acid oxidation increased significantly in both sexes, with the greatest change occurring in women. The authors concluded that NMES can significantly increase the skeletal muscle aerobic-oxidative potential of sedentary subjects. In a study by Arvidsson et al.[15] there was no difference between a postoperative ACL reconstruction–NMES-treated group and an isometric exercise group in the activity of the oxidative enzyme citrate synthase or phosphofructokinase. Research by Wigerstad-Lossing et al.[16] showed no significant reduction in the activity of the oxidative and glycolytic enzymes citrate synthase and triphosphate dehydrogenase in NMES patients following knee ligament surgery compared with untreated control knee ligament surgery patients, which had decreased enzyme activity.

Effects on Muscle Fibers

The muscle fiber atrophy that occurs with disuse following cruciate injury and surgery is significant. Neuromuscular electrical stimulation may be more effective than volitional exercise in preventing atrophy and overcoming the effects of reflex inhibition in the first few days to weeks after surgery. Electrical stimulation also recruits Type II muscle fibers first, thereby retarding their atrophy to a greater degree than Type I muscle fibers.[6,7] As Type II fiber augmentation occurs the force of contraction increases, and therefore, strength increases. With volitional contraction in humans a selective or preferential activation of fast-twitch motor units has not been demonstrated. Slow-twitch motor units with Type I skeletal muscle fibers are recruited first, followed by the fast-twitch motor units with Type II skeletal muscle fibers being recruited as the demand for force increases.[7]

Neuromuscular electrical stimulation of the triceps brachialis muscle in monkeys did not affect the distribution of muscle fiber types but enhanced oxidative capacity and caused an increase in fiber size.[17] There was a preferential recruitment of large motor axons that innervate fast-twitch fibers. However, fast-twitch motor units were not exclusively activated because there was also activation of slow-twitch motor units, but to a lesser extent. Motoneurons and muscle fibers were recruited primarily in the superficial portion of the muscle. The morphologic and biochemical adaptations that occurred with NMES were similar to those occurring after voluntary heavy-resistance exercises.

Effect on Perfusion

Neuromuscular electrical stimulation also induced capillary proliferation in monkeys in response to increased muscle blood flow. Because blood flow increased in capillaries around Type II fibers to a greater degree than around Type I fibers, there was a stimulus for proliferation of endothelial cells.[17]

Transcutaneous electrical stimulation was used on rat muscle to demonstrate that microvascular perfusion depends on evoked muscle contractions. This finding suggests that NMES should be applied at intensities that produce muscle contraction so that microvascular perfusion of stimulated skeletal muscle is enhanced.[18]

Overall Effects of Neuromuscular Electrical Stimulation

The net physical effects of NMES are an increase in muscle strength, muscle mass, and oxidative capacity.[6,14,16,17,19,20]

Another benefit of NMES is the ability to overcome the effects of reflex inhibition on the quadriceps muscles, and the potential enhanced effect of NMES on subsequent voluntary use of previously electrically activated motor units.[6] The negative effects of NMES include muscle fatigue and possibly increased pain. Muscle fatigue is the decrease in force-generating ability of a muscle because of recent activity. The rate of muscle fatigue during NMES is much greater than that which occurs during volitional exercises. The reverse recruitment order induced with NMES probably contributes to increased fatigue. However, stimulation intensity, stimulation frequency, and duty cycle may be altered to reduce muscle fatigue.[21] Increased current levels may recruit pain fibers and result in painful muscle contractions. In addition, discomfort is also associated with psychological factors in humans. Pain may be controlled by decreasing the current intensity to a level that elicits comfortable muscle contractions.[22]

Efficacy and Clinical Trials of Neuromuscular Electrical Stimulation for the Anterior Cruciate Ligament Reconstructed Knee

Currier and Kellogg[19] used NMES to treat seven patients after ACL reconstruction. Treatment successfully reduced thigh girth loss compared with three control patients during the first 6 weeks.

Snyder-Mackler et al.[6] studied the effects of clinical NMES on thigh muscle strength and gait in 10 patients undergoing ACL reconstructive surgery. Patients were randomly assigned to NMES and volitional exercise or volitional exercise alone for 4 weeks. Values obtained for cadence, walking velocity, stance time on the affected limb, flexion excursion of the knee during stance, thigh circumference, and extension and flexion torques were improved in patients receiving NMES. The knees of the patients receiving NMES were stronger in the eighth postoperative week than reported averages for non-NMES patients years after ACL reconstructive surgery. The authors concluded that NMES not only increased muscle strength but also improved functional use of the muscles at least in the immediate postoperative period. The increased muscle strength likely results in partial compensation for the loss of ACL stability.

DeLitto et al.[20] compared clinical NMES and isometric contraction with voluntary exercise and isometric contractions in patients who had undergone ACL reconstructive surgery. Each group consisted of 10 patients. Neuromuscular electrical stimulation patients received treatment for 3 weeks during the first 6 postoperative weeks. After their respective treatment period, bilateral maximal isometric measurements of gravity-corrected knee extension and flexion torque were obtained. Greater extension and flexion torque and higher individual thigh-muscle strength gains were reported when NMES was prescribed early in postoperative rehabilitation.

Lieber et al.[23] compared maximal voluntary thigh muscle contraction after 4 weeks of treatment in 20 NMES patients and 20 voluntary contraction patients who had undergone ACL reconstruction within 2 to 6 weeks of the study. They compared the two groups under conditions in which the total muscle tension in both groups was matched. When exercise intensity was matched between the two groups, the magnitude and time course of strength gains were identical. Given the variable intensity of maximal voluntary contraction that NMES patients tolerate, variability in outcomes may be expected between NMES strengthening and voluntary contraction groups. The authors concluded that when strong muscle contractions can be elicited with NMES, significant strengthening is possible.

Wigerstad-Lossing et al.[16] compared NMES using a portable unit and voluntary muscle contractions to voluntary muscle contractions alone in patients immobilized in a cast following ACL surgery. Thirteen patients were randomly assigned to the treatment group and 10 to the control group. Electrical stimulation was applied to the quadriceps muscles only, through windows in the cast, and was initiated on the second postoperative day and continued until the sixth week after surgery. Preoperative data were compared with data obtained in the sixth postoperative week, and the authors concluded that NMES in combination with simultaneously performed voluntary muscle contractions can limit muscle weakness, muscle wasting, and reduction in oxidative and glycolytic muscle enzyme activity during immobilization after knee ligament surgery.

In another study 18 male and 20 female patients undergoing ACL reconstruction were randomly assigned to one of two groups.[15] Individuals in an NMES group received a cast, isometric muscle training, and NMES with a portable unit. Individuals in the control group received a cast and isometric training alone. Transcutaneous electrical stimulation of the quadriceps was initiated during the first postoperative week and continued for a total of 5.5 weeks. Quadriceps muscle wasting was assessed with computed tomography (CT) before and 6 weeks after surgery. Percutaneous muscle biopsies were obtained from the vastus lateralis muscle before surgery, and 1 and 6 weeks after surgery. Electrical stimulation significantly reduced the amount of vastus medialis muscle wasting in female patients as measured by CT, but there was no significant difference in males. Muscle fiber area revealed that cross-sectional area of individual fibers decreased less in NMES patients than in controls, but the difference was not significant. The authors concluded that transcutaneous electrical stimulation was effective in significantly reducing vastus medialis atrophy as measured on CT, but had no significant effect on the vastus lateralis.

The results of clinical trials using NMES for postoperative rehabilitation of the ACL-deficient knee in people generally have demonstrated increased thigh girth, increased

thigh muscle strength, improved cadence, improved walking velocity and stance time, and increased oxidative and glycolytic muscle enzyme activity.

Rehabilitation Programs for Treatment of Anterior Cruciate Ligament Deficiency

In human surgery, rehabilitation of patients after ACL reconstruction is necessary for an optimal outcome.[24-27] Controversy exists regarding what constitutes the ideal postoperative rehabilitation program for human patients. Current trends in volitional exercise regimens are geared toward accelerated rehabilitation programs. Typical accelerated programs involve ROM exercises and cryotherapy started immediately postoperatively, and crutch use for 2 to 10 days. Closed kinetic chain exercises in the form of exercises in which the foot is fixed and motion at the knee joint is accompanied by motion at the hip and ankle joint (such as squats) are initiated as early as 7 days postoperatively. Closed kinetic chain exercises appear to decrease knee joint compressive forces, thereby decreasing the anteroposterior translation of the tibia, as compared with open-chain knee extension exercises.[28] Neuromuscular electrical stimulation is commonly used during the first 3 to 6 weeks of rehabilitation, depending on the ability of the patient to volitionally contract the quadriceps. At approximately week 10 light jogging, Stairmaster, and agility drills may generally be attempted. From weeks 12 to 24 strengthening programs, proprioceptive training, and increased running and agility programs are added as tolerated. Advancement from one level of activity to another is based on physical examination, knee laxity testing, and isokinetic and functional testing.[16,25,28-30]

Postoperative rehabilitation and the role of the quadriceps and hamstring muscles in improving stifle function have been largely ignored in veterinary medicine. Postoperative care of dogs, regardless of the surgical technique used, traditionally has included several days to weeks of external support to reduce postoperative swelling and provide joint immobilization. This is done to protect the surgical repair until pericapsular fibrosis occurs in the case of extracapsular repair, provide protection of an intracapsular graft while it remodels, protect the site of fixation of the graft, and permit healing of the osteotomy and repair site with a tibial plateau leveling osteotomy (TPLO).[31-42] However, immobilization exacerbates muscle atrophy and weakness and produces deleterious effects on cartilage, bone, and ligaments.[4,15,16,24,26]

Veterinary surgeons should not make the error of using all aspects of the accelerated volitional rehabilitation protocols designed for human patients in their veterinary patients post-CrCL stabilization. The majority of human ACL surgical repairs involve using the bone–patellar tendon–bone technique, which is strongest the day of surgery and loses strength as tendon remodeling occurs. Many canine CrCL-deficient stifles are stabilized using the extracapsular lateral fabella suture technique, which relies on collagen formation and scar tissue as the primary stabilizer of the joints.[43] This technique results in immediate strength following surgery because of the stabilization sutures, and the periarticular structures gain strength as tissue healing and collagen maturation occur over time. Intracapsular techniques typically are not as strong, and the graft weakens over several weeks before regaining some strength. Therefore rehabilitation protocols for canine patients must be based on the surgical stabilization technique used. However, NMES may be useful in the rehabilitation of the stifle without compromising the stability of the repair.

The negative effects of quadriceps reflex inhibition leading to quadriceps atrophy following ACL injury and the positive protective effects of hamstring muscle strengthening to enhance stability and constraint of the stifle following CrCL injury have not been adequately addressed in veterinary medicine. Clinicians may wish to consider adapting ACL-protective rehabilitation procedures to the dog, to protect the surgical repair. Improved postoperative functional outcome following ACL reconstruction in humans is positively correlated with return of quadriceps strength.[6,19,26] Although the hamstring muscles also atrophy following ACL injury and surgery, the atrophy is generally less than that found in the quadriceps muscles, probably because the hamstring muscles cross both the knee and hip joints, which results in continued function and muscle contraction, thereby diminishing the degree of atrophy.[26]

Neuromuscular Electrical Stimulation Use and Clinical Recommendations in the Dog

Neuromuscular Electrical Stimulation Use in Veterinary Medicine

In establishing a rehabilitation program, the cost-benefit ratio must be assessed. For a program to be cost and labor effective there must be earlier or more protected return to function, increased joint ROM, increased muscle strength, and an overall improved outcome.[6] Although cost is a major concern in veterinary medicine, of equal importance is how a rehabilitation program is applied to the dog. Obviously the use of crutches or other limb assistive devices are not directly applicable to the dog. However, external coaptation with soft padded bandages and cage rest, when needed, appear to be practical alternatives. Bicycling, weightlifting, proprioceptive training, and jumping rope, which are the mainstays of rehabilitation in people, are generally either not possible in dogs, or are performed with modifications. A practical rehabilitation alternative to enhance operative outcome is needed for dogs. Electrical stimulation provides a cost- and labor-effective means of establishing early, protected return to function, as well as

the possibility of decreased degenerative joint disease (DJD) and increased muscle mass.[44,45]

Neuromuscular electrical stimulation is an effective means of rehabilitation of the ACL-deficient knee in people and is considered by some to be more effective than volitional exercise, especially during the early rehabilitation period. Because the CrCL-deficient dog has been used extensively as a model for ACL deficiency in humans, it logically follows that post-ACL reconstruction rehabilitation methods in humans could be applied to the dog. Rehabilitation of the stabilized knee determines the success of surgical outcome, especially in the physically active human patient. Appropriate rehabilitation seems especially important to enhancing surgical outcome in large working dogs used for hunting, obedience trials, service, tracking, or police work. Loss of function in the working dog not only affects the quality of life of the patient, but may also lead to economic losses associated with an animal's inability to work. Surgical stabilization of the stifle is the treatment of choice in large-breed dogs, but all affected stifles appear to develop DJD and joint laxity regardless of the surgical intervention.

To date only a few studies have reported the use of NMES in the rehabilitation of dogs. In a study by Johnson et al.[44] NMES was used following CrCL surgery in experimental dogs, and the portable units used in this study proved quite feasible for use in dogs. The affected thigh was clipped and scrubbed with isopropyl alcohol. A limb-positioning device was constructed to maintain the limb in a normal stance position during stimulation. The rationale for maintaining the limb in a normal stance position was that stimulating the muscles in this position would be more functional. Adhesive tape was used to fix the limb-positioning device to the lateral surface of the affected pelvic limb during the 30-minute treatment session.

Carbon electrodes (4.5 × 4.6 cm) were placed over the distal portion of the biceps femoris muscle, and the proximal portion of the semitendinosus muscle. Additional carbon electrodes (5.1 × 10.2 cm) were also positioned over the distal portion of the vastus medialis muscle and the proximal portion of the vastus lateralis muscle. Water-soluble electrode gel was applied as a coupling agent, and the electrodes were fixed in position with paper tape. Channel 1 was connected to the hamstring muscle electrodes, and channel 2 was connected to the quadriceps electrodes. The parameters for the program were as follows:

Channel 1: Ramp up—3 seconds; ON: 12 seconds; ramp down—2 seconds; OFF: 25 seconds

Channel 2: Ramp up—2 seconds; ON: 12 seconds; ramp down—1 second; OFF: 25 seconds

Channel 2 started 3 seconds after channel 1, automatically eliciting cyclic hamstring and quadriceps co-contraction. A symmetric biphasic waveform of 35 pulses per second was used. Amperage was increased to the tolerance level of the animal and was reduced if gross movement was noted at the stifle joint or the animal displayed any signs of distress, including turning its head in recognition of the stimulus or becoming agitated. Amperage was reduced to a level below that which produced these signs. Each treatment session lasted 30 minutes once daily, five times per week, for 4 weeks. Outcome measures were assessed at weeks 0, 2, 5, 7, 9, 13, and 19. Outcome measures included radiographs, force plate analysis, subjective lameness scores, drawer sign, thigh circumference, ROM, palpable crepitation during ROM, and postmortem evaluation of stifle joints.

Radiographic results demonstrated that the NMES group had significantly fewer bony changes associated with osteoarthritis over time. Thigh circumference was significantly greater in the NMES group at weeks 9 and 13, and subjective lameness scores were also improved over time in the NMES group ($P < .001$). The postmortem evaluation revealed that the NMES group had less cartilage damage ($P < .07$), less palpable crepitation during ROM, and less osteophyte formation. Range of motion, force plate analysis, and joint laxity were not significantly different between the groups. Meniscal damage was significantly greater in the NMES group, however.[44]

In a study by Millis et al.[45] a clinical trial was designed to compare the outcomes of postoperative physical rehabilitation to traditional postoperative care in dogs surgically treated for cranial cruciate rupture. Ten dogs receiving treatment for cranial cruciate rupture were randomly assigned to either a treatment group for postoperative physical rehabilitation, or a control group. Lateral fabellar-tibial stabilization was performed in each dog by the same surgeon. Dogs in the control group were cage rested and walked on a leash for 20 minutes twice daily for 2 weeks. Dogs in the treatment group received passive ROM exercises, NMES, and walking exercises for the same 2-week period. Thigh girth measurements and maximum angles of flexion and extension were taken every other day during the 2-week period and 4, 8, and 12 weeks after surgery. The influence of other variables, such as meniscal injury, and the degree of preexisting osteoarthritis on outcome were also assessed.

By the end of the 2-week period thigh girth was significantly larger in the rehabilitation group and remained greater throughout the rest of the study, but the difference late in the study was not statistically significant. Maximal extension of the stifle was significantly greater in the rehabilitation group beginning halfway through the initial 2-week period and remained greater throughout the rest of the study. Maximal flexion was not significantly different between the two groups. The presence of moderate to severe osteoarthritis at the time of surgery retarded recovery in both groups. Interestingly meniscal injury at the time

of surgery had no effects on any of the measured parameters in the short term. In this study, muscle mass and maximum range of stifle motion were improved with postoperative physical rehabilitation. The increased muscle mass in the dogs receiving rehabilitation may have been due, in part, to NMES.

Neuromuscular electrical stimulation has been used clinically on dogs for many purposes, including to diminish joint contractures and decrease muscle atrophy associated with a variety of disorders such as inherited Labrador myopathy, postoperative atrophy, and nerve injury. Other uses have included improving limb function, decreasing pain, decreasing muscle spasm (commonly associated with intervertebral disk disease), and decreasing edema.

Neuromuscular electrical stimulation may be valuable as part of the treatment for postoperative rehabilitation of dogs undergoing femoral head and neck excision. It may also help to improve the outcome of surgically repaired chronic hip luxations, or any other surgical procedure in which quick return of muscle mass is beneficial. Neuromuscular electrical stimulation may also be used for the conservative treatment of shoulder instability[46] or iliopsoas muscle trauma.[47]

Animal Reaction and Safety

Precautions should be taken to avoid injury to the handler and animal. A muzzle should be applied and the animal placed in lateral recumbency during the initial treatment. In some cases, tranquilization may be necessary if the animal is anxious. We recommend that treatment only be given under the supervision of trained personnel.

Preparation and Electrode Placement

The hair over the area to which electrical stimulation will be applied should be clipped to lower impedance (Figure 20-9). The skin should be cleaned with alcohol before treatment. It is best to locate the motor point (the area in which the motor nerve enters the muscle) so that an adequate contraction is obtained with as low a current as possible to minimize discomfort to the patient. Usually the motor point is very predictably found in the middle portion of the muscle belly. Electrodes may be placed solely on one muscle to cause a contraction and motion at the joint the muscle acts upon (Figure 20-10), or they may be placed on opposing muscle groups to cause a co-contraction (Figure 20-11) that may simulate an isometric contraction and result in little or no joint movement, or alternating stimulation of antagonist muscles to cause contractions of the muscles and movement of the joint.

Motor point location is performed by first applying gel to the skin, and then placing the electrode over the general area of the motor point, or the point at which the nerve enters the muscle (typically in the middle of the muscle belly). The other electrode is placed further distally over the muscle. With the unit on, the proximal electrode may



Figure 20-9 Preparing the skin for treatment.

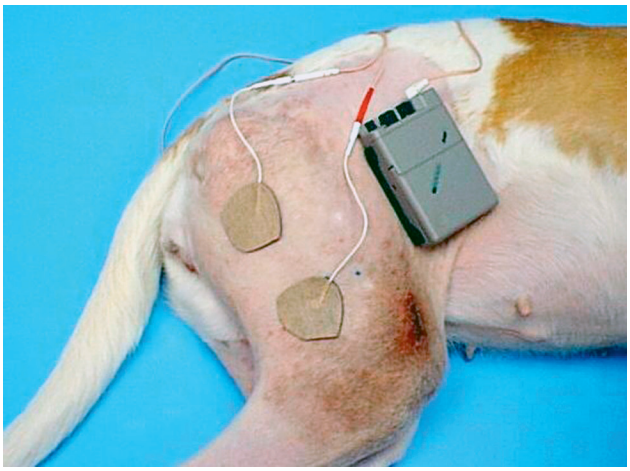


Figure 20-10 Application of electrical stimulation for contraction of the caudal thigh muscles. (Courtesy D. Millis.)

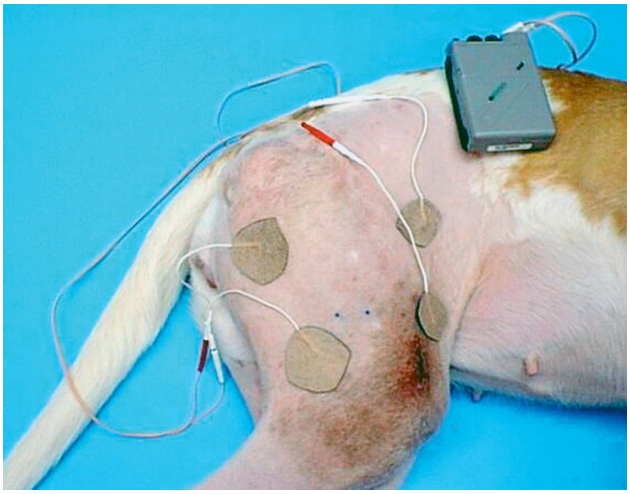


Figure 20-11 Application of electrical stimulation for cocontraction of the cranial and caudal thigh muscles. (Courtesy D. Millis.)

then be moved around until a good contraction is achieved. Setting the frequency at 1 Hz helps in motor point determination because the twitch contraction is more obvious and becomes stronger as the electrode moves closer to the motor point. An indelible marking pen may be used to draw a circle around the electrode. This allows the electrode to be placed in the same area during subsequent treatments, without having to repeat the process of motor point location. Remember to adjust the frequency to the desired setting (25-50 Hz) before beginning the actual treatment.

Treatment Time and Frequency of Treatment

Although the optimum time and frequency of treatment are unknown, most clinicians believe that electrical stimulation should be applied to the desired area(s) for 15 to 20 minutes, three to seven times per week. Occasionally a patient may experience muscle soreness early in the treatment program if electrical stimulation is used too frequently, for treatment periods that are too long, or with application of current that is too great (muscle contraction is too strong). In these cases, skipping treatment for a day or two and resuming treatment with reduced treatment time, frequency, or strength of contraction is usually adequate to resolve the problem. Co-contraction of opposing muscle groups should be considered if no joint motion is desired.

Electrical Stimulation for Pain Control

The use of electrical currents has a long tradition of pain control in humans and has been commonly used in medical practice since the 1970s. There are a variety of commonly used currents for pain control, but in all of them, surface electrodes are placed over defined locations and peripheral nerves are stimulated via the skin.

Pain-Relieving Mechanisms of Electrical Stimulation

The pain relief mechanisms of TENS are explained by multiple theories. In 1965, Melzack and Wall published their gate control theory of pain control. This theory describes how pain may be relieved by electrical stimulation. Pain signals are transmitted by small cutaneous A-δ and C fibers. If a TENS current is applied, the large cutaneous (A-β) fibers are stimulated. The signals from the A-β fibers activate inhibitory neurons in the substantia gelatinosa of the spinal cord dorsal horn and block the transmission of pain impulses from the periphery to the brain.[48] The A-β fibers can be stimulated by low current intensities and high frequencies; therefore, an electrical current at a high frequency (50 Hz or greater) is appropriate to activate this pain control mechanism.[49] Further mechanisms involve the release of endogenous opiates.[50-52] These endorphins are released from the pituitary in response to low frequency (<10 Hz), high pulse duration (>100 µsec) stimulation, and they produce analgesia when released. Interneurons are located in the substantia gelatinosa that produce enkephalins to inhibit the C fiber cells in the region. Collaterals of the A-δ fibers connect with these interneurons and stimulate them. If A-δ fibers are stimulated by electrical impulses, the C fiber system-type pain is decreased.[49] This is also one of the suspected mechanisms of action of acupuncture. Therefore, low-frequency, high-intensity TENS might be sufficient to stimulate the same pain control system that may occur with acupuncture. In an animal model, it was shown that high-frequency TENS increased the concentration of γ-aminobutyric acid in the spinal cord. This caused a reduction of central sensation and primary hyperalgesia.[53]

Types of Electrical Stimulators

Many different waveforms, amplitudes, and frequencies have been recommended for pain control, and the literature describes many applications. The high-frequency, short-pulse duration (conventional) TENS (50-150 Hz, 2-50 µsec pulse duration, low intensity) is one of the most frequently used current parameters. The intensity is increased until the dog shows some initial response, such as moving the limb, or turning the head toward the area, which we assume is a tingling sensation described by people. No muscle contraction should occur. As described the treatment is presumed to stimulate large, low-threshold A-β fibers to activate the gate control mechanism.

Acupuncture-like TENS (low-frequency, high-pulse duration, high-intensity, 1-10 Hz, 100-400 µsec pulse duration, high-intensity) is another mode of electrical stimulation for pain control. Regarding the low frequency stimulation, some authors differentiate between the acupuncture-like TENS with frequencies between 1 and 4 Hz, and frequencies between 5 and 10 Hz. The acupuncture-like TENS is administered at an intensity that evokes visible muscle contractions. This stimulates the high-threshold A-δ fibers and C fibers that may result in the release of endogenous opioids. This form of TENS is often applied directly to acupuncture points and also to motor points in the segmentally related myotome.

Burst mode TENS is a series of pulses at 1 to 4 Hz and a high internal or carrier frequency (40 to 150 Hz), 100 to 250 µsec pulse duration and a high intensity. The implied benefit is that it combines the benefits of both conventional and acupuncture-like TENS.

Modulated TENS offers the possibility to vary pulse length, frequency, and amplitudes automatically. This cyclic variation is believed to prevent the nervous system from becoming adapted or conditioned to the currents.

Interferential (IFC) and premodulated interferential are additional currents commonly used for pain control. These currents are very comfortable and have been commonly used in canine rehabilitation. They are delivered at a high



Figure 20-12 Application of electrodes for lumbar pain. (Courtesy D. Millis.)

carrier frequency (approximately 4000 Hz). The parameters recommended for acute pain are 80 to 150 Hz (frequency modulated), and for chronic pain are 1 to 10 Hz (frequency modulated). If the frequency modulation is not used, a fixed frequency of 50 to 100 Hz for acute pain and 2 to 4 for chronic pain is supported by the literature.[54] When using interferential current, four electrodes from two separate channels are used; the electrodes from one channel are crossed at a 90-degree angle to the electrodes from the other channel (Figure 20-12). Premodulated interferential uses just 1 channel and 2 electrodes for small areas. While it is not true interferential current, it feels almost identical and is very comfortable.

Research in Human Medicine

Transcutaneous electrical nerve stimulation has been the topic of numerous clinical trials in human medicine regarding its effectiveness for pain control in various disorders. Unfortunately the results are inconsistent because of a variety of reasons, such as weak study design, missing control/placebo groups, and small numbers of participants. The use of TENS was evaluated in several systematic metaanalyses and reviews in humans. Again the results were inconclusive, showing clear benefits of electrical stimulation for pain control in some studies,[55-57] whereas others showed no effect.[58,59] Nevertheless there is reasonable evidence in human medicine that TENS is an effective method for pain relief, especially in cases of chronic musculoskeletal pain. In one metaanalysis, the authors concluded that high frequency TENS, as well as TENS with frequencies lower than 10 Hz, produced a significant effect and acupuncture-like TENS fell just short of significance.[54] Another metaanalysis focused on the treatment of knee OA using different types of TENS currents.[55] When the combined efficacies of conventional TENS and acupuncture-like TENS were compared with placebo, pain improved significantly in the treatment group. In addition, knee stiffness was improved in the combined treatment group. The significant pain-relieving effect was also detectable if conventional and acupuncture-like TENS were evaluated separately. Interestingly this metaanalysis found that pain relief by acupuncture-like TENS was approximately two times better than with high-frequency TENS.

Transcutaneous electrical nerve stimulation treatment is also an effective method in cases of postoperative pain. In humans who underwent lumbar canal stenosis surgery, pain and the doses of analgesics administered were evaluated in two groups. One group received pain medication and TENS treatment (80 Hz during the first 30 minutes of the therapy, switching to 5 Hz in the second half), whereas the other group received the same medication, but no TENS. In the TENS-treated group, pain and the total doses of analgesic medications were significantly reduced compared with the control group.[60]

Research in Veterinary Medicine

Electrical stimulation for pain control may be used in numerous orthopedic and neurologic disorders. Studies in humans have shown benefits in acute[60] or chronic[55,54] pain caused by osteoarthritis, spondylarthrosis, and after orthopedic and neurologic surgery. Before starting the treatment, special awareness should be given to the contraindications and precautions listed in Box 20-3. A few studies have been conducted in dogs, including the effects of TENS on osteoarthritic pain in the stifle of dogs.[61] Five dogs that had chronic mild OA that was originally induced by CrCL transection and stifle stabilization were treated. Pretreatment ground reaction forces were determined using a force plate before electrical stimulation to assess the functional use of the affected limb. Immediately following these measurements, premodulated electrical stimulation (70 Hz) was applied to the involved extremity around the stifle. After treatment ground reaction forces were determined at 30-minute intervals over a 4-hour period. Each dog was also reevaluated 24 hours after treatment. Significant improvement in ground reaction forces was found 30 minutes after treatment. These differences persisted for 210 minutes after TENS application and were statistically significant 30, 60, 120, 150, and 180 minutes after treatment. The greatest improvement was found immediately after treatment. Each of the dogs was reevaluated following a 4-day rest period and still exhibited a mild increase in weight bearing on the affected extremity, but these differences were not significant. In this preliminary study, positive benefits of TENS application were apparent in dogs with osteoarthritic stifle joints.

In another study, 29 overweight dogs with lameness caused by OA of the hip, elbow, stifle, or shoulder were included in a weight loss program.[62] The dogs were randomly divided into two groups and the diet was combined with physiotherapy. Owners of dogs in both groups were instructed to perform massage, passive range of motion, and gradually increase levels of controlled exercise. Group 1 dogs were additionally treated with TENS in a physiotherapy clinic twice weekly. Body weight, subjective lameness, and pain scores were evaluated monthly. A force-plate analysis was performed every second month. Asymmetric weight distribution between limbs affected and unaffected by OA was demonstrated using symmetry indices, calculated using peak vertical force (PFz) and vertical impulse (IFz). During the first 2 months of the study the animals in both groups lost about the same amount of body weight, but at day 90 the TENS-treated group had lost significantly more weight than group 2. This difference was also noted at the 6-month point. During the course of the study the lameness scores decreased significantly, beginning at day 30 in group 1 and at day 60 for group 2. Pain scores also decreased, with the difference reaching significance sooner in group 1 (at 60 vs. 90 days). Force plate analysis showed that symmetry indices for PFz and IFz in group 1 were significantly improved at each evaluation. In group 2, only the symmetry index for PFz improved, and only at day 120. At day 60, there was no difference in body weight between groups, but the symmetry indices were only significantly improved in group 1. This suggests that TENS could have a significant influence on lameness. The authors concluded that a combination of dietary management and intensive physiotherapy including TENS treatments may effectively reduce disability in dogs that are overweight and have osteoarthritis. Further study of TENS application in dogs, including different parameters and altered physical activity, may reveal additional benefits.

Preparation and Electrode Placement

The hair (unless very short) should be clipped before the treatment if rubber electrodes or gel pads are used. If electrodes are not coated with gel, suitable contact medium, such as ultrasound or electrocardiogram gel, should be applied in an even layer to ensure uniform transmission of the current to the skin. Clipping should be performed carefully to avoid skin damage. Another possibility is the use of pads with brushlike needle pads. In this case, clipping the hair is not necessary, but the skin should be wetted with water or alcohol. For each treatment session the patient should be placed in a relaxed position (e.g., lateral recumbence) on a soft and comfortable surface.[63] To achieve a beneficial effect, the electrodes should be placed in the proper location. Several possible locations may be chosen, and the choice should be adapted based on the effectiveness of the treatment. Sometimes it is necessary to try

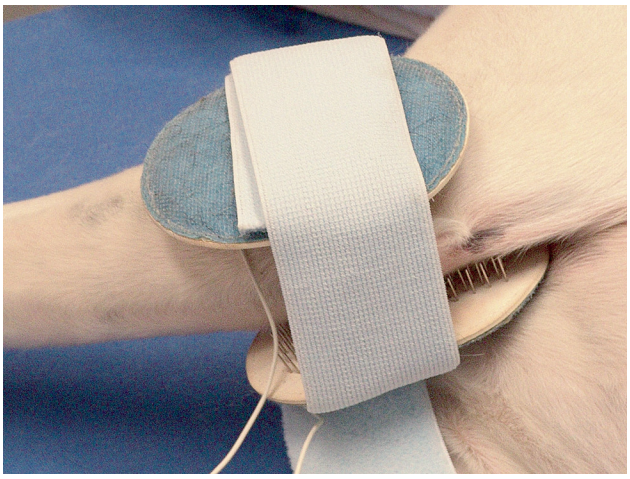


Figure 20-13 Application electrodes for chronic pain using flexible E-pads because of elbow dysplasia. (Courtesy B. Bockstaler.)

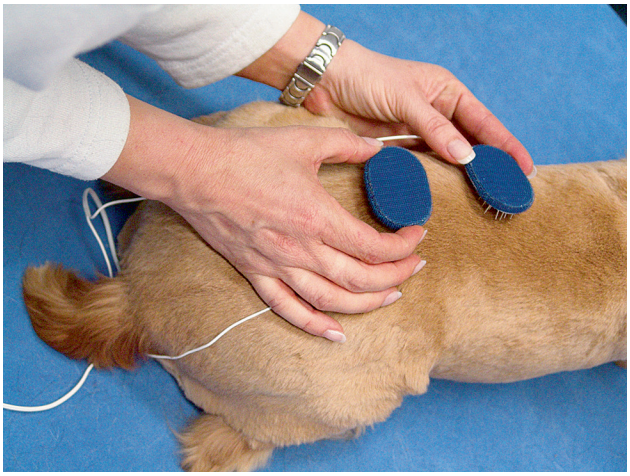


Figure 20-14 Application of electrodes for lumbar pain utilizing flexible E-pads. (Courtesy B. Bockstaler.)

several locations to achieve the best pain-relieving effect. In a study of human long-term users, most patients applied the electrodes over or directly proximal to the painful area.[64,65] In a dog one example would be placement over the medial and lateral aspects of an arthritic elbow (Figure 20-13) or stifle joint, or along the edges of the painful area (e.g., cranial and caudal region of painful back muscles) (Figure 20-14). This so-called "local placement" is mainly used in chronic conditions.

In cases of acute disease (e.g., an acute stage of OA) if placing the electrodes over the area is painful, or aggravates the symptoms, segmental stimulation (stimulation within a dermatome) may be applied with the electrodes placed to the left and right of the vertebral column near the nerve origins of the target region.[63] To place the electrodes correctly an in-depth knowledge of canine neuroanatomy is necessary. For example, to treat hip joint pain the pads could be placed over the L4-S1 nerve roots on the side of

the painful hip. Other possibilities include placement of electrodes over acupuncture or trigger points, or along the peripheral superficial nerve tracts.

Intensity, Treatment Time, and Frequency of Treatment

The intensity must always be adapted to the needs of the individual patient and their response to the treatment. To evaluate treatment success the pain level of the animal should be evaluated initially and during the course of treatment. This is necessary to monitor the effectiveness of the selected parameters, such as electrode position, intensity, and frequency, and the recovery of the animal. The intensity can be chosen according to the subjective evaluation of the patient, but it also depends on the desired pain-relieving mechanism and the type of TENS.

To make the animal comfortable with TENS the treatment is often started with an intensity of the current just below the sensory response threshold. In dogs this can be achieved by carefully increasing the intensity until the animal shows a reaction, such as glancing at the electrodes or tensing the limb muscles. The intensity is then decreased slightly just to the point that the animal stops reacting. Especially when treating animals it is always mandatory to start the treatment carefully and with patience. Each patient should have time to adapt to the treatment and relax. With increasing intensity the patient likely will feel a tingling sensation. This should be neither painful nor should it cause muscle contractions. A higher intensity is often not recommended and not tolerated by the animal. Accordingly a distinction is drawn between intensities below the motor threshold (no visible muscle contractions), at motor threshold (visible twitch contraction), and above it (wavelike muscle movements and contractions).

There are different recommendations for treatment duration. For example, for treatment of OA using conventional TENS, treatment times vary between 15-,[62] 30-,[61,66,67] and up to 60-minute[68] sessions. Also, the timing of the treatment varies from three sessions daily for 3 weeks,[68] to twice daily for 6 weeks, to twice weekly for 4 weeks.[69]

In general, the following recommendations may be applied.[63] In acute stages, a lower-intensity, short treatment duration, and short intervals are suitable. Therefore, in an acute case, it is possible to treat the animal one to two times daily, with a 15-minute treatment duration. In chronic conditions, higher intensities with longer treatment durations and intervals between the treatments are recommended; for example, treatment two to three times per week, for 5 to 6 weeks, with each session lasting 30 minutes.

Future Areas for Study

Continued clinical trials investigating electrical stimulation protocols for muscle strengthening and pain control need to be conducted. Given the similarities in neurologic and orthopedic conditions between dogs and people, it seems that the indications for electrical stimulation are similar. Iontophoresis is another area that should be studied in dogs to investigate its possible clinical application.

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21 Laser Therapy in Canine Rehabilitation

Darryl L. Millis and Debbie Gross Saunders

Healing properties have been attributed to light for thousands of years. Light provides electromagnetic radiation in the form of photons. A number of forms of light have been used for therapeutic purposes, including sunlight, incandescent light, infrared light, ultraviolet light, light-emitting diodes (LEDs), and, recently, therapeutic lasers. This chapter focuses on therapeutic lasers rather than surgical lasers. Laser therapy has been increasingly incorporated into rehabilitation programs for a variety of conditions, including skin wounds; muscle, tendon, and ligament injuries; neurologic conditions; arthritis; and pain. Many studies have indicated encouraging results with laser therapy. The principles of lasers, research pertaining to the use of laser therapies in rehabilitation, and the basics of laser therapy are addressed.

Laser Therapy

The term *laser* is an acronym for *light amplification by stimulated emission of radiation.* The concept of the use of light for therapeutic purposes, called *phototherapy,* originated from the belief that sun and other sources of light, such as infrared and ultraviolet light, have therapeutic benefit. Many different types of lasers are available for medical and industrial purposes. Low-power laser devices, a form of artificial light, were first used as a form of therapy more than 30 years ago. Today, a variety of lasers are in use for various purposes.

The initial types of lasers used for rehabilitation purposes, commonly known as low-level laser therapy (LLLT), are also called *cold lasers.* In contrast, surgical lasers are high power and capable of thermal destruction of cells and tissues. Recently another form of laser, known as a *therapeutic laser,* has been introduced for rehabilitation purposes and delivers more power than low-level lasers, but less power than surgical lasers. Therapeutic lasers have become increasingly popular in both small and large animal rehabilitation for a variety of conditions.

The lasers used in rehabilitation help to modulate cellular functions. This process is known as *photobiostimulation* and is defined as nonthermal interaction of monochromatic radiation with a target site.[1] Although the physiologic interaction of this type of energy application on tissue is still not completely understood, low-energy lasers have been reported to modulate various biologic processes, such as mitochondrial respiration and adenosine triphosphate (ATP) synthesis, to accelerate wound and joint healing, and to promote muscle regeneration.[2,3] In addition, acute and chronic pain control has been reported using this type of low-energy photon therapy.[4] Treatment of chronic and acute edema, neurologic conditions, and postoperative care are some other popular conditions treated with laser therapy.

Properties of Lasers

Basic light sources emit electromagnetic radiation that is visible to the normal eye. Although natural light sources, such as sunlight, are forms of electromagnetic radiation, lasers are artificial sources that emit radiation in the form of a flow of photons (Figure 21-1). The process of light emission begins with activation of electrons in the laser unit, generally either helium-neon (HeNe), gallium-arsenide (GaAs), or gallium-aluminum-arsenide (GaAlAs) to an excited state.[5] When the electrons drop from their excited state to their ground state, photons are emitted. Although some photons are absorbed by the laser chamber wall, others stimulate the emission of other photons, and together they travel in the chamber, amplifying this stimulated emission, which results in a chain reaction. Some of these photons are released through a semireflective mirror to form a beam of light.

The major difference between laser light and light generated by normal sources is that laser light is monochromatic, coherent, and collimated. *Monochromatic* means that all light produced by the laser is of one wavelength, and therefore a single color. Sunlight, or white light, may be broken into several different colors of different wavelengths by a prism (Figure 21-2). Laser light has electromagnetic radiation of only one wavelength. Commercial lasers occasionally have two or more different wavelengths within a single unit to achieve different effects, but each component within the unit has a single wavelength and is monochromatic. The coherent properties of light mean that the photons travel in the same phase and direction (Figure 21-3). Laser light is also collimated, which means that

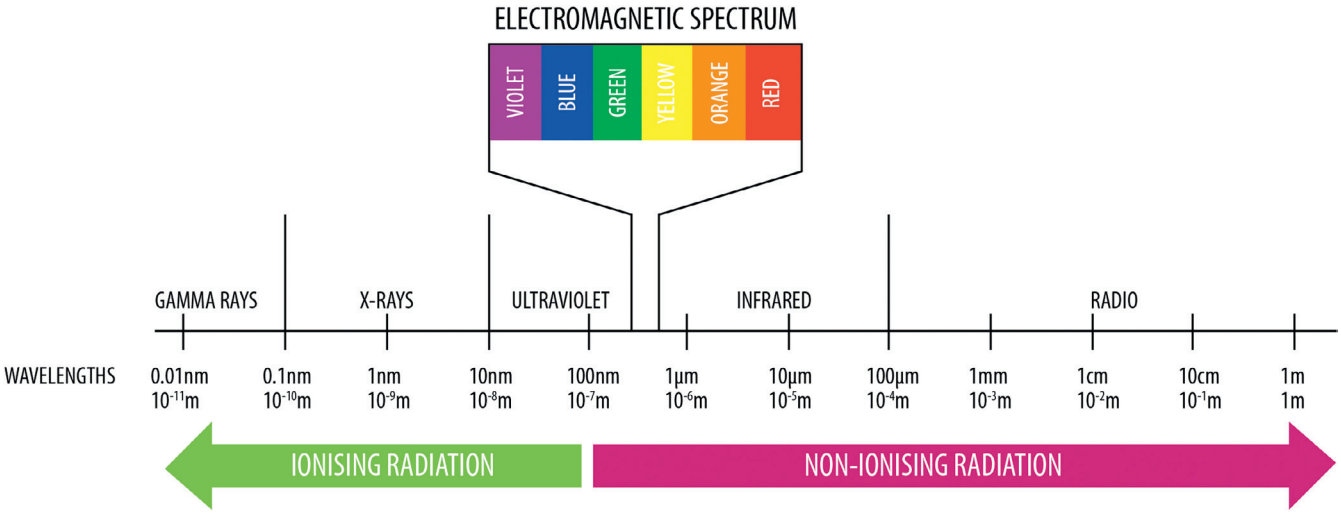


Figure 21-1 Electromagnetic spectrum. Laser light typically falls within the infrared or near red portion of the electromagnetic spectrum. (Photo Courtesy Companion Laser, LiteCure LLC, Newark, Delaware.)

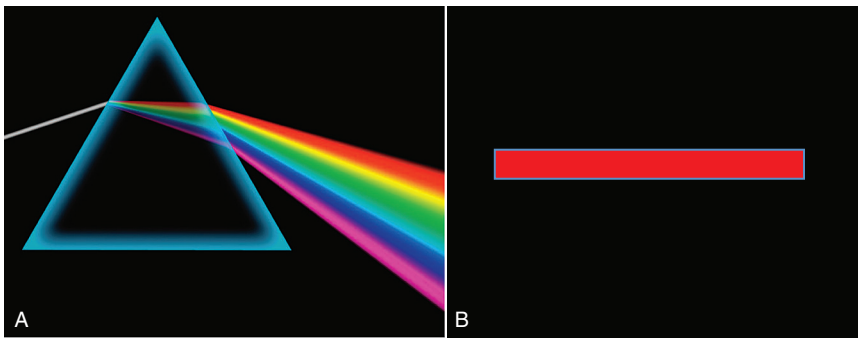


Figure 21-2 **A,** Sunlight, or white light, may be broken into several component colors with different wavelengths. **B,** Laser light is monochromatic and is produced by a single wavelength.

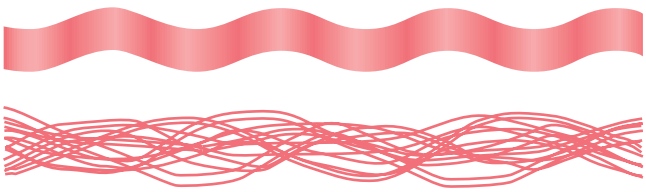


Figure 21-3 Laser light is coherent, with the photons traveling in the same phase and direction, whereas normal light is noncoherent.

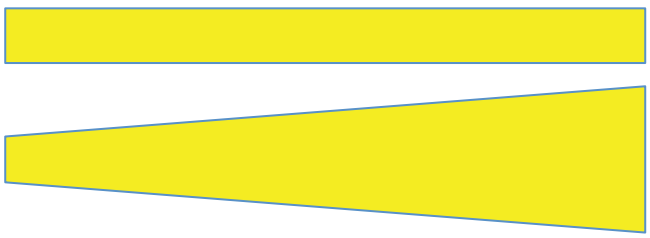


Figure 21-4 Laser light is collimated, which means that there is minimal divergence in the laser beam over a distance. Normal light is not collimated, and the beam diverges over a distance.

there is minimal divergence in the laser beam over a distance (Figure 21-4). Using a monochromatic light source allows the absorption of the light to be targeted to specific wavelength-dependent chromophores, or photon acceptors.[5] The properties of coherence and collimation allow the light to be focused precisely on small areas of the body. These properties allow low-level laser light to penetrate the surface of the skin with no heating effect, no damage to the skin, and few or no side effects when properly used.

Laser light interacts with tissues in various ways. Light may be reflected, scattered, transmitted, or absorbed. Reflected photons have no clinical effect and may be dangerous to tissues that encounter reflected photons, such as the eyes. In addition to surfaces such as watches, tables,

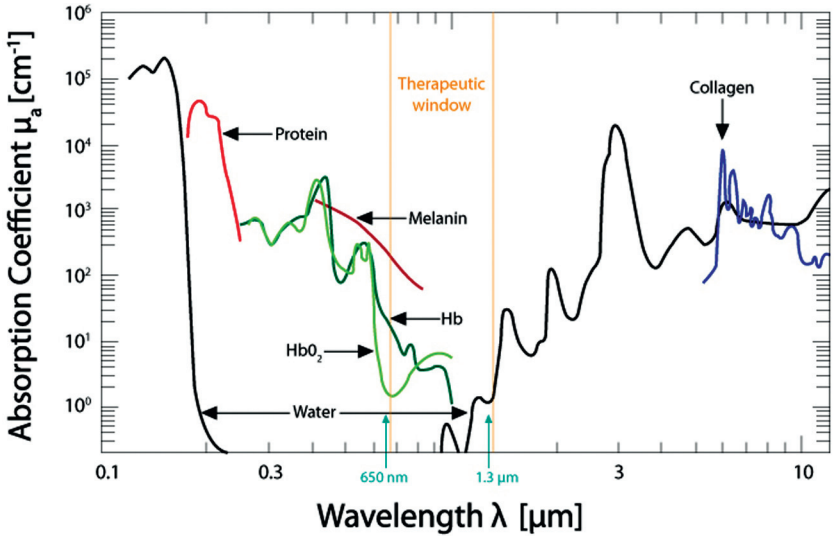


Figure 21-5 The wavelength of the photons is important in laser therapy. Various substances preferentially absorb laser light at different wavelengths. (Photo Courtesy Companion Laser, LiteCure LLC, Newark, Delaware.)

and instruments, the epidermis is responsible for reflecting most of the photons from the skin. To reduce reflection of photons, the laser beam should be directed as nearly to 90 degrees as possible to the skin surface. As photons pass through the tissues, some are scattered. Each photon that is scattered when striking an object reduces the amount of energy that can be directed at the target tissue. Scattering decreases as the wavelength increases because longer wavelengths penetrate deeper into tissues. Transmitted photons pass completely through the tissue without being absorbed. This is rarely a problem in rehabilitation because the tissues are generally thick enough to prevent complete transmission. Finally, photons may be absorbed. Photons are absorbed by chromophores (molecules that absorb certain wavelengths of light) and it is in this manner that laser may affect tissues. The most common chromophores are water, hemoglobin, melanin, cytochrome C system in mitochondria, proteins, and amino acids.[6] The relative amounts of chromophores vary among tissues, and the absorption of photons by chromophores varies, in part, on the wavelength.

The wavelength of the photons is important in laser therapy. Wavelengths are measured in nanometers (nm) and determine, in part, the biologic effect on tissues. For example, ultraviolet light (100-400 nm) is absorbed primarily by melanin, proteins, and nucleic acid; visible light (400-760 nm) is scattered and absorbed, with the absorption primarily by melanin, hemoglobin, and myoglobin; with near infrared light (760-1400 nm), photons are mainly scattered, but a variety of chromophores absorb these photons, although somewhat weakly; in the far infrared zone (1400-10,000 nm), absorption is almost entirely by water (Figure 21-5).[6] Therefore the optimal window to have tissue penetration with less scattering and surface absorption is likely to be in the 600-1200 nm range.

In addition to wavelength, the power of the laser is also a characteristic that is important. Power is a unit of time and is expressed in watts (W) or milliwatts (mW).

$$1 \text{ watt} = 1 \text{ joule/second}$$

The spot size of the unit indicates the surface area the laser covers while held in a stationary position. This is also known as the *power density,* or *intensity,* and indicates the power per surface area unit. It is usually indicated by watts/cm^2. Larger spot areas result in a more homogeneous passage of laser light through tissues with less photon scattering and light dispersion.

The energy is the power emitted over time, and is usually measured in joules. This is also frequently used to report the dosage of laser light.

$$1 \text{ joule} = 1 \text{ watt} \times 1 \text{ second}$$

For example, a 50-mW laser delivers 1 J of energy in 20 seconds of treatment time. A 500 mW laser takes 2 seconds of treatment time to deliver 1 J of energy. A 1-W laser takes only 1 second, and a 10-W laser takes $\frac{1}{10}$ of a second. From a treatment standpoint, a higher watt laser delivers the treatment in a shorter time. If an arthritic stifle is being treated, it may require a total dose of 100 to 500 J depending on the size of the dog and treatment area. So the treatment time may vary greatly depending on the unit used.

The energy density, or radiant exposure, is the energy per surface unit and is typically indicated by joules per cm^2, or J/cm^2. This term is also clinically called the *dose of laser energy* and is important in determining and describing treatment protocols. To date, only the dose of laser energy

Box 21-1 Biologic Effects of Laser Therapy

Activation of respiratory chain enzymes, especially cytochrome C oxidase
Oxygen production
Formation of proton gradients across cell and mitochondrial membranes
Adenosine triphosphate production
DNA production
Cell proliferation
Reduced cyclooxygenase and prostaglandin E_2 production

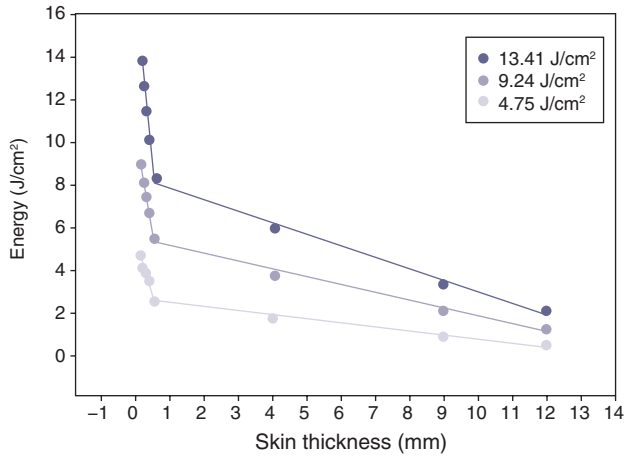


Figure 21-6 Three different doses of laser light and penetration through skin. Greater doses of laser energy penetrate deeper into tissues. (From Topping A, Gault D, Grobbelaar A et al: Does Low Penetration of Human Skin by the Normal Mode Ruby Laser Account for Poor Permanent Depilatory Success Rates? *Lasers Med Sci* 16:224-229, 2001.)

has been shown to have biologic effects as compared with the rate of administering laser energy, or the power of the laser (Box 21-1). Greater doses of laser energy penetrate deeper into tissues (Figure 21-6). The Lambert-Beer law indicates that for homogenous tissues with a constant coefficient of absorption, more photons will reach a given tissue depth with a greater dosage, and the intensity of light passing through tissues decreases exponentially with increasing tissue depth. There is a great deal of difference among lasers related to the power, with those delivering energy at a greater rate generally costing more. Although there is not thought to be an advantage in efficacy regarding low- versus high-power lasers, the dosage (total joules) can be administered in a much shorter time with a high-power laser, therefore reducing labor costs. In addition, high-power lasers are generally administered in a sweeping fashion, whereas lower power lasers are usually held in place until the administration of the dose for a particular spot treatment is complete. The sweeping motion may result in more thorough coverage of the treatment area as compared with the spot treatment of lower power lasers.

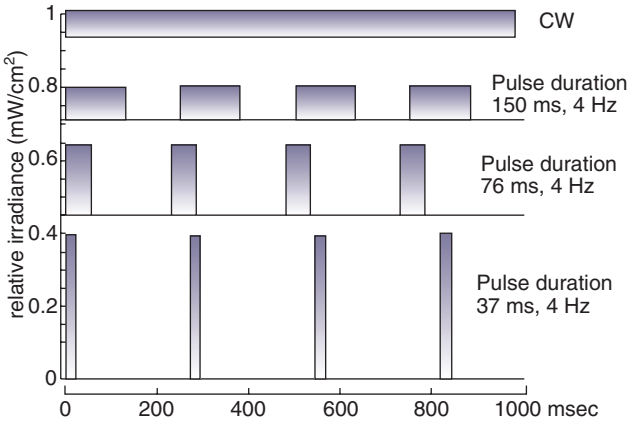


Figure 21-7 Diagram of laser light delivered as continuous wave or pulsed laser light of various pulse durations. (Based on a diagram from Hashmi JT et al: Effect of pulsing in low-level light therapy. *Lasers Surg Med* 42:450-466, 2010.)

The sweeping motion also allows the clinician to cover other surrounding areas that may be causing secondary or tertiary pain. For example, when treating canine hip dysplasia to address pain and inflammation, the coxofemoral joint should be treated from all areas—medial, lateral, caudal, and cranial. With the sweeping motions covering the four areas, the laser may affect the surrounding soft tissues that are often painful in clinical canine hip dysplasia.

Lasers can emit photons continuously or in a pulsed fashion (Figure 21-7). With continuous wave laser, radiation is emitted at a constant power over the entire treatment time. With pulsed wave laser treatment, impulses may be emitted at varied rates, with an on time when energy is emitted (pulse width or pulse duration [PD]) and an off time when there is no energy emitted. The duty cycle (DC) is the percentage of time that radiation is emitted in relation to the total on-off time. The pulse rate, or frequency (F), is measured in Hertz (Hz). The relationships between these measures may be expressed as:

$$DC = F \times PD$$

One report reviewed the literature regarding pulsed versus continuous laser application.[7] Although the authors concluded that there is some evidence that pulsed laser has different effects than continuous laser, further work is necessary to define optimal treatments for various conditions, and to determine the optimal pulse structures. One theory behind the use of pulsed laser is that with relatively short pulses, the laser could be excited to higher levels as compared with continuous wave laser, in which thermal damage to the tissues might limit the maximum amount of energy that can be applied. With short pulses, the thermal effects on tissues might be reduced, limiting tissue damage. Pulsed GaAs and indium-gallium-arsenide (InGaAs) lasers may allow for deeper tissue penetration without thermal damage, as well as allowing for shorter treatment times. In addition to decreasing the thermal effects on tissues, pulsing of the

laser may also have some resonance with certain functions, such as brain waves and opening of cellular ion channels. Pulsing may also have effects on photodissociation of substances, such as nitric oxide (NO) from protein binding sites, allowing multiple episodes of dissociation. Although the actual depth of penetration is probably not that different in pulsed versus continuous wave lasers, for many conditions, depth of penetration is a less important issue, such as with treatment of elbow or stifle arthritis. One author concluded that pulsed lasers (904 nm) were not significantly more effective than continuous-wave lasers (810-830 nm) for treatment of tennis elbow in people.[8] Both types were equally effective, but half the energy was needed with pulsed lasers. However, a review of literature comparing continuous wave laser with pulsed laser identified six of nine studies that found pulsed wave to be more effective.[7] A clinical study of wound healing and experimental studies of pain management and stroke recovery have suggested that pulsed laser has better results than continuous laser. Another study comparing continuous and pulsed laser found both to be equally effective. Only two of the nine studies reported better results with continuous than pulsed laser, although both treatment methods achieved better results than placebo treatments. Some limitations of the studies in this review were that the same parameters of laser wavelength were not used in some instances, making direct comparisons difficult. One study found a combination of pulsed and continuous laser to be more effective than either alone for stimulating nerve regeneration in a median nerve transection and repair study,[9] whereas most studies have shown continuous laser to be more effective than pulsed laser for nerve conduction and regeneration.[7] Studies comparing various rates of pulsed laser treatment show no consistent results, with some demonstrating better results with lower frequencies and some with higher frequencies.[7]

Classification of Lasers

The American National Standards Institute in the United States and the International Electrotechnical Commission have defined classes of lasers based on their ability to damage tissues. In general it is the thermal damage that injures tissues, especially the eyes, and in some cases, skin. Even small amounts of laser light can cause permanent damage to the retina.[10]

Current classifications of lasers are related to the wavelength and the maximum output power (in watts) or energy (in joules). These classifications allow the placement of various lasers into categories based on their ability to cause tissue damage, especially to the eyes. For example, Class 1 lasers are very mild are safe under all conditions of normal use, and include supermarket scanners and post office readers. Class 1M lasers are safe for all uses except when passed through a lens, such as a microscope or telescope. These types of lasers have large-diameter beams or divergent beams and cannot achieve the power to cause damage unless the laser is focused with a lens. The power is less than that of a Class 3B laser, but cannot damage the retina unless the light is focused. Class 2 lasers are in the visible-light spectrum (400-700 nm) and include items such as laser pointers, and occasionally therapy lasers. Because these lasers are visible light, the blink reflex generally limits exposure to less than 0.25 seconds. Class 2 lasers are limited to 1 mW continuous wave, or more if the emission time is less than 0.25 seconds or if the light is not spatially coherent. Class 2M lasers, similar to Class 1M lasers, have a large-diameter light beam or the beam is divergent and is safe unless the light is viewed through an optical instrument.



Figure 21-8 Class IV laser.

Class 3B lasers are either continuous light in the 315 nm to far infrared ranges limited to 500 mW, or pulsed lasers 400-700 nm wavelength limited to 30 mW. When using Class 3B lasers, protective eyewear is required if direct viewing of the light may occur. Class 3R lasers are considered to be safe with restricted exposure and are visible-light continuous lasers with power limited to 5 mW.

Class 4 lasers have the greatest potential to cause tissue damage and include all lasers with power greater than that of Class 3B lasers (Figure 21-8). Surgical, industrial cutting lasers, and some therapeutic lasers are Class 4 lasers. Surgical lasers typically have power between 30 to 100 W, whereas therapeutic lasers may be 1 to 15 W. Class 4 lasers may burn the skin or cause permanent eye damage as a result of direct, diffuse, or indirect beam viewing, such as might occur with reflection of the beam, even from matte surfaces. Therefore, the therapist must use great care to control the beam path and protective eyewear must be worn by all in the immediate area. The U.S. Food and Drug

Administration (FDA) requires all Class 3B and Class 4 lasers in the United States to have safety features, including a key switch, a safety interlock dongle, a power indicator, an aperture shutter, and an emission delay (normally 2-3 seconds). In addition, the U.S. Occupational Safety and Health Administration requires the use of adequate eye protection when eye exposure may occur while operating lasers in these classes.

Lasers Used in Physical Rehabilitation

Most lasers used in physical rehabilitation are Class 3 or 4 lasers and have a finite lifespan that generally varies form 5000 to 20,000 hours. LLLT devices are typically Class 3 lasers and are low power, often less than 100 mW, and they do not heat tissues. The lack of heat generation is significant. If heat is generated by a Class 3 laser, the unit may not be functioning properly or the device may be improperly used. LLLT devices typically have small treatment beam diameters, up to 1 cm. The basic types of lasers used for LLLT are gaseous HeNe and GaAs or GaAlAs semiconductor or diode lasers. HeNe lasers have a visible red light with a wavelength of 632.8 nm, whereas GaAs and GaAlAs have invisible light near the infrared band with a wavelength of 820-904 nm.

The wavelength of the laser is important because it determines, in part, the laser's effect. Longer wavelengths are more resistant to scattering than shorter ones. Therefore, GaAs and GaAlAs lasers penetrate more effectively (direct effect up to 2 cm, indirect effect up to 5 cm) than HeNe lasers (direct effect up to 0.5 cm, indirect effect up to 1 cm) because there is less absorption or scattering in the epidermis and dermis. Light waves in the near infrared ranges penetrate the deepest of all light waves in the visible spectrum (Table 21-1). Although this spectrum of light is not visible, commercial lasers should have an LED that allows the therapist to see where the laser light is aimed.

Lasers can only have an effect if they stimulate cells, and the depth of laser penetration is important to determine if cells at a particular depth may be stimulated. Very few studies have measured the depth of laser penetration through skin, and these studies have been done primarily on human skin samples. Readers should use caution in interpreting the results of these studies and extrapolating them to dogs because of inherent differences in skin thickness, skin composition, cutaneous blood supply, skin color, and thicker hair coats. Lasers with shorter wavelengths are most effective for surface-level conditions because they do not penetrate to deep tissues or large joint capsules. Wound care and wound healing should benefit from lasers with shorter wavelengths. Light that is not absorbed by water, hemoglobin, or melanin is gradually attenuated as it passes through tissues. The level of scattering and absorption is such that HeNe (632.8 nm) laser light loses approximately one third of its intensity during the first 0.5-1 mm of tissue depth.[11] The depth (in centimeters) at which the energy of a laser beam is 36% of its original values is termed the *first depth of penetration.*[12] This attenuation of energy is derived by dividing the original value by a constant, 2.78. Subsequent depths of penetration may be determined by dividing by 2.78 again, so that the level of energy at the second depth of penetration is 13%. However, because biologic effects may be noted with relatively low energy (0.01 J/cm^2), lasers that typically deliver 1 to 4 J/cm^2 may penetrate up to 0.5 to 2 cm before the energy level is so low that they have no effect. These factors apply to human skin, and, although there are many similarities between human and animal skin, there are significant differences caused by hair, pigmentation, and tissues.

One study evaluated the penetration of ruby laser light (694 nm) in human white skin. Doses of 4.75 J/cm^2, 9.24 J/cm^2, and 13.41 J/cm^2 were administered to the skin.[13] (see Figure 21-6). The most likely explanation for the tremendous drop in energy in the first mm is scattering of laser light, which supports the hypothesis that the greatest hindrance to a photon energy beam traversing the skin is from the stratum spinosum of the epidermis, collagen in the dermis, or both. Beyond the depth of the dermis and at the levels of the subcutaneous fat, the energy drop per distance traveled was not as great. The mean maximum depth of penetration was 14.8 mm which appeared to be a function of wavelength and not dosage. The natural chromophore for this wavelength of laser is melanin. Therefore the depth of penetration might be expected to be less in dark-skinned dogs. After passing through the skin, penetration through the muscle becomes easier because muscle has a smaller scattering coefficient.[14] In fact, transmission of laser light between 600 and 800 nm is approximately fourfold greater in skeletal muscle than skin.[15]

Another study with laser of similar wavelengths had similar results. An HeNe laser (632.8 nm, 50 mW power) and a semiconductor laser (675 nm, 21 mW power) were used to measure transmission in human skin and skin with granulation tissue from leg wounds with ulcers.[16] In the thickest sample (epidermis, dermis, and subcutaneous tissue, 2 cm thickness), approximately 0.3% of HeNe laser and 2.1% of semiconductor laser light penetrated through

Table 21-1 Wavelengths of Various Components of the Electromagnetic Spectrum

AM Radio	10,000 cm
TV and FM	100 cm
Microwave	10 cm
Infrared	700 nm
Ultraviolet	10 nm
X-rays	1 nm

all layers. Penetration of laser light was greatly attenuated at 1 to 3 mm of tissue depth. Transmission in granulation tissue was approximately 2.5 times higher than that in normal skin.

In another study of depth of penetration, a GaAlAr laser (wavelength 850 nm, 100 mW power) was applied to human abdominal skin samples.[17] The intensity of laser radiation was reduced by 66% after being transmitted through a 0.784 mm sample of human abdominal tissue. In this study, most laser radiation was absorbed within the first 1 mm of skin. Additional research is needed in small animals to determine the depth of penetration with different hair coats and skin color.

Biologic Effects of Laser Therapy

Most studies of laser use in rehabilitation have centered on wound healing and pain management. However, information regarding their efficacy in reducing pain or promoting tissue repair is incomplete.[18] Recently, interest has been generated in the United States regarding their use in treating people, and a natural extension has been an interest in treating animals. In evaluating the potential usefulness of laser therapy in rehabilitation, the reader is encouraged to be critical of studies that have been performed, and to evaluate these studies in light of recent advances in laser technology and the application of new information. Until recently, low-energy and therapeutic lasers were not approved for medical treatment in this country. However, as more evidence becomes available, they will likely be increasingly used.

Most of the potential responses of cells and tissues to laser energy have been studied in in vitro models. Photons delivered to the cells and tissues trigger biologic changes within the body. Photons are absorbed by chromophores and respiratory chain enzymes (especially cytochrome C oxidase, the terminal enzyme of the mitochondrial respiratory chain) within the mitochondria and at the cell membrane. Copper components of cytochrome C oxidase are photoacceptors.[19] Cellular signaling causes a cascade of cellular reactions resulting in things such as NO dissociation from cytochrome C oxidase.[19] This, in turn, results in further changes down the respiratory chain. Oxygen production and the formation of proton gradients across the cell and mitochondrial membranes may also occur. The enzyme flavomononucleotide is activated and initiates the production of ATP. Even small changes in ATP levels can change cell metabolism. The role of ATP in cellular energetics is well known, but ATP may also act as a signaling molecule to enhance cell to cell communication.[19] ATP may bind with the cell receptor P2X, which opens a channel to allow sodium and calcium to enter cells, resulting in a cascade of intracellular interactions. Increased intracellular calcium positively affects mitochondrial function. ATP may act as a neurotransmitter when released by nerve cells, but may also have other effects when released by other cells, such as bone production and cell proliferation.[20] The changes in ATP may, through its effects as a neurotransmitter, explain some of the effects of laser therapy on pain modulation and the effect of acupuncture.

The effect of laser treatment on various cells in vitro has been evaluated in several studies.[21] HeNe laser (632 nm) with a power of 10 mW and a dose of 0.43 J/cm^2 resulted in greater proliferation and differentiation of human osteoblasts in vitro.[22]

DNA production may also be stimulated. Photons also appear to affect tissues by activating enzymes, which trigger biochemical reactions in the body. Because cellular metabolism and growth are stimulated, lasers have the potential to accelerate tissue repair and cell growth of structures, such as tendons, ligaments, and muscles. Although lower dosages of laser energy appear to stimulate tissues, higher dosages may actually inhibit responses such as tissue healing. The enzyme kinetics may differ between high enzyme/substrate, and low enzyme/substrate conditions.[23]

A great deal remains unknown regarding the mechanisms of laser light in biologic systems. For example, NO and carbon monoxide inhibit cytochrome C oxidase. The effects of near infrared laser radiation in conjunction with NO on cytochrome C oxidase are unknown.[24] Further work is also needed regarding the effects of temperature, pH, exposure times, and frequency of photobiomodulation treatment on cytochrome C oxidase.

Laser therapy may also stimulate stem cell proliferation.[19] In addition to the direct effects of laser at a particular depth of tissue, indirect effects may also be seen. These cellular and tissue effects are decreased in the deeper tissues, and are catalyzed by the energy absorption in the more superficial tissues.

Laser therapy may also have antiinflammatory effects, similar to nonsteroidal antiinflammatory drugs (NSAIDs) and steroids. Studies of laser therapy in cell culture have indicated that inflammation can be reduced by a decrease in prostaglandin E_2 (PGE_2) and cyclooxygenase-2 (COX-2) concentrations.[25,26] This effect has also been demonstrated in an animal model,[27] as well as people with Achilles tendinitis.[28] Frequently, owners seek alternatives to NSAIDs for their animals, and laser therapy may offer alternative care. Animals that are unable to consume NSAIDs may also benefit from laser therapy as an alternative for inflammation.

Research Regarding Laser Therapy

Wound Healing

Laser light stimulates fibroblast development and may affect collagen production to repair tissues. Laser light may also accelerate angiogenesis and increase the formation of new capillaries in damaged tissues, possibly improving the rate of wound healing (Box 21-2). Therefore laser therapy may aid healing of open wounds and burns. There is an increased growth factor response within cells and tissues, which may

Box 21-2 Laser Therapy Research

Wound Healing
- Fibroblast stimulation
- Capillary formation and angiogenesis
- Collagen formation
- Enhanced adenosine triphosphate, protein, and growth factor production
- Vasodilation
- Lymphatic drainage
- Potential inhibition of wound healing at high doses

Bone and Cartilage
- Enhanced early bone repair
- Increased collagen deposition and bone trabeculae
- Adjunct to treatment of osteomyelitis
- Fibrous healing of cartilaginous defects
- Improved maintenance of cartilage in immobilized joints

Arthritis
- Inhibition of inflammation
- Inhibition of cyclooxygenase-2 enzyme and prostaglandins
- Reduced pain
- Possible reduction of morning stiffness in rheumatoid arthritis

Ligament and Tendon Conditions
- Pain reduction in acute tendonitis
- Improvement with lateral epicondylitis
- Reduced pain and inflammation in Achilles tendonitis
- Improved collagen organization
- Improved biomechanical properties

Analgesia
- Reduced pain in postoperative incisions

Inhibits Nociceptors
- Potential reduction in transmission of pain signals to pain centers in the brain
- Increased release of endorphins and enkephalins
- Stimulation of trigger points and acupuncture points
- Slowing of nerve conduction velocity
- Reduced action potential
- Suppressed substance P
- Disruption of axonal flow

Peripheral Nerves and Spinal Cord
- Promotion of nerve recovery after injury
- Increased axonal sprouting and growth
- Increased myelinization
- Reduced degeneration of neurons
- Increased growth-associated protein-43 and calcitonin gene–related peptide

be related to increased ATP and protein synthesis. Laser light therapy causes vasodilation and also may improve lymphatic drainage. This may result in decreased edema and swelling caused by bruising or inflammation.

In cell culture, doses of 0.5 to 16 J/cm^2 were evaluated in vitro as a single exposure on two consecutive days.[29] A single dose of 5 J/cm^2 resulted in increased proliferation and viability of injured fibroblasts. Higher doses (10 and 16 J/cm^2) had reduced cell proliferation and viability, with damage to the cell membrane and DNA. The results of this in vitro study do not necessarily imply that similar findings would be present in vivo, but do suggest that there may not be a linear dose-response effect.

A review of experimental studies related to laser treatment of wounds in 8 mouse and 39 rat model studies has been reported.[30] Wound healing in rodent models differs from wound healing in people because of the loosely attached skin in rodents as compared with people, making wound contraction a significant factor in healing in rodents. The loosely attached skin of rodents may be similar to dogs and cats, and therefore mice and rats may be a useful model of wound healing to evaluate treatments for dogs and cats. Many studies continue to inadequately report laser parameters used in treatment protocols. In spite of these shortcomings, the results of these studies suggest that laser or monochromatic light is effective as a treatment for wounds when applied at appropriate doses. Coherence of light may not be an important factor when treating surface wounds. LEDs with monochromatic light appear to have effects on topical wounds, similar to those obtained with laser light. Wavelengths in both the visible red (630-685 nm) and infrared (700-1000 nm) typically stimulated wound healing in a variety of models. Some studies suggested that the best results were obtained when using 660-nm laser light in the early stages of wound healing, whereas 780 nm produced positive results throughout all stages of wound healing.[31,32] In general, there was no clear association between outcomes and power levels used in the studies evaluated in this review.[30] However, lack of standard experimental design, including total dose administered, made it difficult to draw conclusions. In those studies that reported energy density or when calculation was possible, there appeared to be a dose-response relationship.[30] However, there may be a biphasic response, with positive effects seen with lower dosages, and inhibitory effects at higher dosages.[30] Treatment with a total dose of 20 J/cm^2 divided between 638 and 830 nm light produced better outcomes than using either wavelength alone, suggesting a possible effect of both superficial and deep treatments.[33] However, two studies with infrared laser treatment using higher dosages had reduced stimulation[34] or inhibitory effects.[35] In one study of rats with circular wounds receiving a single 830-nm laser treatment with 1.3 J/cm^2, more rapid epithelialization and wound contraction resulted compared with 3 J/cm^2, although both treatments resulted in stimulation of healing compared with controls, and there was no difference between the treated groups by day 14.[34] In a mouse wound model, 18 J/cm^2 of 980-nm laser treatment had a beneficial effect on wound healing, whereas 36 J/cm^2 was too aggressive and resulted in decreased healing.[35]

Laser therapy may also be beneficial for difficult wounds in metabolically compromised patients. Laser

photostimulation accelerated wound healing in diabetic rats in one study.[1] Diabetes was induced in male rats by streptozotocin injection and two 6-mm diameter circular wounds were created on either side of the spine. The left wound of each animal was treated with a 632.8 nm HeNe laser at a dose of 1 J/cm^2 5 days per week until the wounds closed (3 weeks). There was a marginal increase of biomechanical properties of the laser-treated wounds, including an increase in maximum load (16%), stress (16%), strain (27%), energy absorption (47%), and toughness (84%) compared with control wounds. The amount of total collagen was significantly increased in laser-treated wounds. It was concluded that laser photostimulation promoted tissue repair by accelerating collagen production and promoting overall connective tissue stability in healing wounds of diabetic rats. HeNe lasers also improved wound healing by increasing collagen synthesis when corticosteroids or nonsteroidal antiinflammatory agents were administered to rats with surgical abdominal wounds.[36] Laser treatment significantly increased collagen synthesis.

One study reported 100 clinical cases with healing wounds treated with LLLT.[37] There was a marked increase in collagen formation, increased vasodilation, and accelerated DNA synthesis. This researcher recommended 1 J/cm^2 of laser treatment.

A statistical metaanalysis was performed to determine the overall treatment effects of laser phototherapy on tissue repair.[18] After performing a literature search, the effectiveness of laser treatment was calculated from each study using standard procedures. Thirty-four peer-reviewed papers met the inclusion criteria for tissue repair. There was a positive effect of laser phototherapy on tissue repair. Collagen formation, rate of healing, tensile stress and strength, time needed for wound closure, number and rate of degranulation of mast cells, and flap survival were improved with laser therapy. Laser treatment with a wavelength of 632.8 nm had the greatest effect, and 780 nm had the least. This metaanalysis concluded that laser therapy was an effective treatment for tissue repair.

Another study reviewed the literature regarding the in vitro and in vivo effects of low-intensity laser therapy on the wound healing process, especially in diabetic patients.[38] Although many of the in vivo studies lacked specific information on dosimetric data and appropriate controls, the data from appropriately designed studies indicated that LLLT should be considered as an adjunctive adjuvant therapy for refractory wound-healing disorders, including those experienced by diabetic patients.

However, results from other controlled and blinded studies have been less clear regarding the efficacy of LLLT for the treatment of wounds.[11] Studies in laboratory animals have suggested that LLLT may improve healing during the early stages of wound healing, but the effect may not result in improved total healing time.[39,40] Another large review paper did not find unequivocal evidence that LLLT was beneficial for the treatment of wound healing.[41]

Bone and Cartilage Effects

Bone and cartilage may also be affected by laser treatment. In one study of bone healing, rats received a defect in a femur.[42] Rats were treated for either 12 sessions (4.8 J/cm^2 per session, 28-day follow up) or three sessions (4.8 J/cm^2 per session, 7-day follow up) with 40 mW 830 nm laser light. Treatments were applied three times per week, and two other groups served as untreated controls. Rats were sacrificed on either day 7 or 28 after surgery. Although there were significant differences between treated and control animals regarding the area of mineralized bone at 7 days, there were no differences at 28 days. The authors suggested that LLLT may have some effect on early bone repair.

Another study of bone healing in rats compared ultrasound with laser.[43] A GaAlAs laser (780 nm) was applied using 30-mW power and a dose of 112.5 J/cm^2. The ultrasound group was treated with 1.5 MHz, at 30 mW/cm^2. Both groups received 12 total treatments, with 5 treatments administered per week. Bones were harvested on day 20. Maximum load at failure was greatest in the laser-treated group. Ultrasound resulted in promotion of bone resorption at the osteotomy site, whereas laser caused bone formation in comparison with control osteotomies.

The use of GaAlAs laser was compared with bone morphogenetic protein (BMP) and bovine organic bone graft in a rat femoral bone healing model.[44] Rats were assigned to four groups: control; laser treatment; BMPs plus organic bovine bone graft; and BMPs plus organic bovine bone graft plus laser treatment. The laser-irradiated groups received treatments every 48 hours for seven treatments, beginning immediately after the surgical procedure. The laser therapy (830 nm, 40 mW) consisted of 16 J/cm^2 per session divided equally over four points (4 J/cm^2 each) around the defect. The subjects were sacrificed after 15, 21, and 30 days. There was increased deposition of collagen at 15 and 21 days, as well as increased bone trabeculae at the end of the experimental period in the irradiated animals versus the nonirradiated controls. The greatest healing was seen in the group treated with organic bovine bone graft, BMP, and laser treatment.

LLLT has also been investigated as an adjunctive treatment for infections of the musculoskeletal system. In one study, the effect of various doses of 808 nm (100 mW continuous laser, at doses of 7.64 J/cm^2, 15.29 J/cm^2, and 22.93 J/cm^2) laser therapy was evaluated in induced chronic osteomyelitis of rat tibias, created with methicillin-resistant *Staphylococcus aureus*.[45] Rats had either surgical debridement, surgical debridement plus laser therapy, or no treatment. Infection levels decreased by 37%, 67%, 81%, and 93% in groups treated by debridement only, or debridement plus laser at 7.64 J/cm^2, 15.29 J/cm^2, or 22.93 J/cm^2, respectively, compared with the nontreated control group. The authors

concluded that laser therapy may be an adjunctive treatment for the management of osteomyelitis. Laser has also been used to create a laser shockwave to clear biofilm from medical devices in vitro.[46] Bacteria surrounding medical devices, such as stents and orthopedic implants, may make a biofilm coating that prevents penetration of antibiotics. In this study, the biofilm was disrupted by a pulsed Nd:YAG laser that produced laser-generated shockwaves, making the bacteria susceptible to conventional treatment.

A more complete review of the effects of laser therapy for bone repair has been published.[47] Most of the research regarding bone healing has been performed in cell culture or in rodent models. However, the authors acknowledge that more study on the laser properties, wavelength, and energy dosage is needed, along with improved study design.

The safety of laser therapy on epiphyseal cartilage has been evaluated in rats.[48] Young rats had either no laser irradiation, 5 J/cm^2, or 15 J/cm^2 of 830 nm GaAlAs laser application to the proximal tibial epiphyseal cartilage every other day for a total of 10 sessions. Laser irradiation increased epiphyseal cartilage thickness and the number of chondrocytes, but these effects were not great enough to increase bone length.

Another study evaluated osteochondral lesions of the knee treated intraoperatively with laser therapy in rabbits.[49] Bilateral osteochondral lesions were created in the femoral medial condyles. All of the left lesions underwent immediate stimulation using a GaAlAs laser (780 nm), and the right knees were left untreated as a control group. After 24 weeks, the condyles were examined histomorphometrically. The condyle treated with laser had better cell morphologic findings and repair of osteocartilaginous tissue.

The effect of laser therapy on cartilage was further investigated in another study that evaluated whether intraoperative laser biostimulation can enhance healing of cartilaginous lesions of the knee in rabbits.[50] Bilateral chondral lesions were created in the medial femoral condyles. The lesion in the left knee of each animal was treated intraoperatively using a diode GaAlAs 780-nm laser (300 J/cm^2, 1 W, 300 Hz, 10 minutes), and the right knee was left untreated. Cartilage was then examined 2, 6, or 12 weeks after surgery. The rabbits receiving laser therapy had progressive filling with fibrous tissue of the cartilaginous lesion, whereas no changes were apparent in the untreated group.

Although laser therapy may have some benefit to treat cartilage injury, it may also have benefit to help maintain the health of cartilage during periods of disuse and immobilization. The influence of laser therapy (632.8 nm, HeNe, 13 J/cm^2, three times a week) on the articular cartilage of rabbit stifles immobilized for 13 weeks was examined in one study.[51] The number of chondrocytes and depth of articular cartilage of the treated rabbits were significantly higher than those of the sham-treated group. The cartilage surface of the sham-treated group was rough and fibrillated, whereas the surface of the experimental group was intermediate between a nonimmobilized control group and the sham-treated group. The authors of this study concluded that low-power HeNe laser irradiation reduced the adverse effects on the articular cartilage of rabbits immobilized for 13 weeks. Another study evaluated the use of 810 nm laser therapy on bone and cartilage during joint immobilization of rat knees.[52] Three groups of rats received either 3.9 W/cm^2, 5.8 W/cm^2, or sham treatment. After 6 treatments over a 2-week period, tissues were harvested for testing. Results indicated that cartilage stiffness, assessed by indentation testing, was preserved in both laser groups.

Treatment of Arthritis with Low-Level Laser Therapy

There is much interest in treating various forms of arthritis with laser therapy, including osteoarthritis (OA) and various forms of inflammatory arthritis, such as rheumatoid arthritis. Models of various forms of arthritis have also been studied in animal models. Zymosan was injected into knee joints of rats to create inflammatory arthritis in one study.[53] LLLT (830 and 635 nm, 20 mW) or LED (628 nm) was applied to animals immediately and 1 and 2 hours after injection, using the same dose of 2.5 J/cm^2. A positive control group was pretreated with a dexamethasone injection into the knee 1 hour before zymosan injection. Laser treatment significantly reduced edema by 23%, vascular permeability by 24%, and pain by 59%, whereas LED treatment had no effect on any of the outcome parameters. Laser treatment may inhibit inflammation by reducing PGE_2 levels by inhibiting the COX-2 enzyme. Another inflammatory joint model also suggested that laser treatment may reduce inflammatory changes.[54] Hydroxyapatite and calcium pyrophosphate crystals injected into rat knee joints resulted in inflammatory changes in the non–laser-treated control animals, but HeNe laser (633 nm, 5 mW, 8 J/cm^2 daily for 5 days) reduced the intensity of the inflammatory changes.

Laser therapy has been used for the treatment of OA in people. The effect of laser therapy in OA of the knee was investigated in one double-blind study.[55] One treated group that received infrared laser (GaAlAs) and the other that received HeNe laser treatment were compared with a group that received placebo treatment. Patients were treated for 15 minutes twice daily for 10 days. Total dose for each session was 10.3 J for HeNe and 11.1 J for GaAlAs. The laser-treated groups were significantly less painful compared with the placebo groups, but there was no difference between the HeNe and the GaAlAs groups. The Disability Index Questionnaire also revealed an improvement in the laser groups. Patients receiving laser treatment had less pain for 2 months to 1 year after treatment.

Laser treatment was also performed on 20 human patients with OA of the knee, ranging from 42 to 60 years of age.[3] All patients had previously received conservative treatment with poor results. The laser device used for this

treatment was a pulsed infrared diode laser, 810 nm wavelength, once per day for 5 consecutive days, followed by a 2-day rest interval. The total number of applications was 12 sessions. Laser treatment was performed on five periarticular tender points, for 2 minutes each. Pain relief and functional ability were assessed using a numerical rating scale, self-assessment by the patient, index of severity for OA of the knee, and analgesic requirements for comfort. There was significant improvement in pain relief and quality of life in 70% of patients, compared with their previous status, but there was no significant changes in range of motion (ROM) of the knee. Although the authors of this study indicated that laser treatment was beneficial, there was not an untreated control group. Therefore the results should be interpreted with caution.

A double-blind randomized study was conducted in 90 patients with OA of the knee to evaluate a GaAs laser in combination with 30 minutes of exercise.[56] One group received 5 minutes of laser therapy, with 3 J delivered. Another group was treated for 3 minutes and received 2 J, and a third group received placebo laser and exercise. Patients received a total of 10 treatments and were studied for 14 weeks. Patients receiving laser treatment had significantly improved pain, function, and quality of life measures after treatment and had improved scores as compared with the placebo laser group.

The analgesic and microcirculatory effects of LLLT were evaluated in a recent study of people with knee OA.[57] Laser therapy (830 nm, continuous wave, 50 mW, 6 W per treatment site) or placebo treatments were administered twice weekly for 4 weeks. Pain, circumference, pressure sensitivity, and flexion were all improved in the laser treatment group, whereas the placebo group had no improvement in flexion or pain. Thermographic measurements showed a 0.5° C increase in temperature in the laser group, suggesting an increase in circulation.

A similar randomized, placebo-controlled study of 60 patients with knee OA indicated no significant improvement with 50 mW 830 nm GaAlAs LLLT at 3 weeks or 6 months.[58] In this study, patients received 3 or 1.5 J per painful joint, or placebo laser treatment, five times per week, with 10 total treatments.

Finally, a metaanalysis review was conducted on the efficacy of laser therapy on OA in people.[59] Seven trials were included, with 184 patients randomized to laser, and 161 patients to placebo groups, using a variety of lasers and treatment protocols. Treatment duration ranged from 4 to 12 weeks. Pain was assessed in four trials. The pooled estimate of three trials showed no effect on pain measured using a scale, and two demonstrated very beneficial effects with laser. In another trial, with no scale-based pain outcome, significantly more patients reported pain relief (yes/no) with laser. One study found knee ROM was significantly increased. Other outcomes of joint tenderness and strength were not significant. Lower dosages of laser were found to be as effective as higher dosages for reducing pain and improving knee ROM. The authors concluded that for OA, the results are conflicting in different studies and may depend on the method of application and other features of laser application, including wavelength, treatment duration, dosage, and site of application over nerves instead of joints.

The efficacy of laser therapy for the treatment of rheumatoid arthritis in people has also been reviewed.[60] Five placebo-controlled trials evaluating 222 patients met inclusion criteria of the review. Treatments were typically administered two to three times per week for 4 weeks. Compared with a control group, laser therapy reduced pain by 1.10 points on a 10-point visual analogue scale (VAS) relative to placebo, reduced morning stiffness duration by 27.5 minutes, and increased tip to palm flexibility by 1.3 cm. Other outcomes such as functional assessment, ROM, and local swelling did not differ between groups. There were no differences based on laser therapy dosage, wavelength, site of application or treatment length. The authors concluded that laser therapy could be considered for short-term treatment for relief of pain and morning stiffness for patients with rheumatoid arthritis. Further evaluation of wavelength, treatment duration, dosage, and site of application over nerves instead of joints is needed. A recent study, however, generally showed no effect of laser therapy (GaAlAr laser, 785 nm, 3 J/cm^2, and mean power of 70 mW) on the hands of patients with rheumatoid arthritis.[61]

Treatment of Tendon and Ligament Conditions with Laser Therapy

Low-level and therapeutic laser has been recommended for the treatment of various tendon and ligament conditions, but the clinical efficacy of this treatment remains controversial. Laser therapy has been used in people for tendinopathies of the Achilles tendon, patella tendon, medial and lateral elbow tendons, and shoulder rotator cuff tendons.

The effect of GaAs laser therapy (904 nm, 4000 Hz, 0.5 to 1 J/cm^2) for tendinitis was evaluated in 74 treated people and 68 people receiving placebo treatment in a randomized, blinded fashion.[62] All patients received six treatments during a period of 3-4 weeks. Pain was estimated objectively using a pain threshold meter, and subjectively with a VAS before treatment, at the end of treatment, and 4 weeks after the end of treatment. Laser therapy generally had a significant, positive effect compared with placebo measured from the first assessment to 4 weeks after the end of treatment. Laser treatment appeared to be most effective for acute tendinitis.

Another study evaluated the effect of laser therapy in seven people with bilateral Achilles tendinitis to see if laser treatment has an antiinflammatory effect.[63] Laser therapy (904 nm, 5.4 J per point, 20 mW/cm^2) or placebo was applied to either Achilles tendon in random fashion. PGE_2 was reduced 75 to 105 minutes after laser therapy, and pain

pressure threshold values increased after laser therapy. The authors concluded that laser therapy reduces pain and inflammation in people with acute Achilles tendinitis.

A rat model of partial calcaneal tendon lesions also evaluated the effects of laser therapy.[64] Sixty rats were assigned to five groups: standard control (no lesion induced and no laser therapy); control (lesion induced, but no laser therapy); and groups 3, 4, and 5, which suffered the lesion and underwent laser therapy for 3, 5, and 7 days, respectively. Laser therapy was delivered with a GaAsAl laser (830 nm), power of 40 mW, power density of 1.4 W/cm^2 with continuous waveform, with a dose of 4 J/cm^2, and total energy of 0.12 J every day during treatment, always in the same period, for 3, 5, or 7 days starting on the day the lesion was produced. Control lesions had significantly less collagen fiber organization compared with the standard controls. There were no differences in collagen organization between group 4 and standard control. Groups 3 and 5 were similar to each other and had greater collagen fiber organization than the animals with control lesions. Laser therapy was effective in the improvement of collagen fibers organization of the calcaneal tendon after undergoing a partial lesion in this study, and early treatment for 5 days appeared to give the optimal response in this study.

Lateral epicondylitis can be a challenging condition to treat in people. A GaAs laser (904 nm, 12 mW, 70 Hz, 0.36 J/point with four or five points treated over the most painful area, two to three treatments per week for a total of 10 treatments) or placebo treatment was applied to people with lateral epicondylitis.[65] Laser treatment resulted in significant improvement in some objective parameters, but not subjective parameters, perhaps because the subjective scales were not sensitive enough to detect small differences.

One recent review and metaanalysis evaluated the use of low-level laser for tendinopathies in people.[66] The results of 25 studies were conflicting, with roughly half showing a positive effect and half being inconclusive or showing no effect. Positive results from studies of lateral epicondylitis and Achilles tendinopathy appear to be related to the dosage of laser applied. Studies suggesting efficacy of laser treatment used doses similar to those recommended by the World Association of Laser Therapy, which suggests doses of 1-8 J and power less than 100 mW/cm^2 for superficial tendons. For deeper tendons, such as the rotator cuff, power up to 600 mW/cm^2 and total dosages of 3-9 J have been recommended.

Laser therapy may also be useful for other forms of soft-tissue injury, such as ligament healing. In one study, 24 rats received surgical transection to their right medial collateral ligament and eight received sham operation.[67] After surgery, 16 received a single dose of GaAlAs laser to their transected ligament for 7.5 minutes or 15 minutes, and eight served as control with placebo laser, whereas the sham group did not receive any treatment. The ligaments were biomechanically tested either 3 or 6 weeks following surgery. The ultimate tensile strength and stiffness of laser and sham groups were larger than controls. The laser and sham groups had improved stiffness from 3 to 6 weeks. It was concluded that a single dose of laser therapy improved the biomechanical properties of healing medial collateral ligaments 3 and 6 weeks after injury. However, a randomized clinical trial of laser therapy for the treatment of ankle sprains in people indicated that laser treatment was not effective.[68]

Analgesia and Pain Management

Interest in the use of laser therapy to treat acute and chronic pain has increased in recent years. In fact, the World Health Organization's Committee of the Decade of the Bone and Joint has recently incorporated laser therapy into guidelines for the treatment of neck pain in people.[69] Knowledge of the effects of laser irradiation on pain has increased in recent years.[69] Nociceptors, which are Aδ and C peripheral nerve fibers, lie just beneath the surface of the skin. Because these nerve fibers are relatively superficial, they are within the depths of laser stimulation. In addition, neurons that supply blood vessels and contribute to vasoconstriction and vasodilatation associated with inflammation are also within the depth of laser penetration. Therefore laser has the potential to influence pain perception by direct or indirect actions on nociceptors, as well as modulation of inflammation, which may contribute to pain. In fact, one study showed a reduction in postoperative pain and drug intake in people after immediate postoperative laser treatment to a surgical incision.[70] Laser therapy may suppress central sensitization and result in long-term depression of persistent pain with repeated application. The exact mechanisms by which laser therapy results in analgesia are unknown, but are the focus of active research. Although both nociceptors and motor nerves may be affected by laser irradiation, sensory nerves appear to be preferentially affected. It is likely that application of laser results in mainly inhibitory effects on aspects of nerve function, especially pain receptors.

The results of studies regarding pain management with the use of laser have been somewhat controversial. However, studies performed have resulted in the approval by the FDA of 635 nm low-level lasers for the management of chronic, minor pain, such as OA and muscle spasms. Laser therapy may have some analgesic effects by blocking pain transmission to the brain. Some studies have shown changes in the conduction latencies of the radial and median nerve after laser therapy,[71,72] but others have shown no effect.[73] Laser treatment may also increase the release of endorphins and enkephalins, which may further provide analgesic benefits. Laser therapy has been used to stimulate muscle trigger points and acupuncture points, which may provide pain relief.[74]

Although the precise mechanism by which laser therapy may provide analgesia is unknown, several studies have

investigated other possible mechanisms. One study evaluated the effects of diode laser irradiation of peripheral nerves.[75] The response was evaluated by monitoring neuronal discharges from the L5 dorsal nerve roots elicited by application of various stimuli to the hindpaw of rats, including brush, pinch, cold, heat stimulation, and chemical stimulation by injection of turpentine. Diode laser irradiation (830 nm, 40 mW, 3 min, continuous wave) of the saphenous nerve significantly inhibited neuronal discharges elicited by pinch, cold, heat, and chemical stimulation, but not discharges induced by brush stimulation. These data suggest that laser irradiation may selectively inhibit nociceptive neuronal activities.

Another study evaluated the effect of laser therapy on the head of rats.[76] Rats received various combinations of laser (0, 6.4, and 12 J/cm^2) and naloxone (0, 5, and 10 mg/kg) prior to a hot plate test. Laser therapy (820 nm, pulsing) was applied to the rats' skulls. Hindpaw lick latencies (in seconds) in response to the hot plate test were recorded immediately, 30 minutes, and 24 hours after the administration of treatment. When animals were tested immediately following laser irradiation at 12 J/cm^2, significant analgesia resulted. Treatment with naloxone at either dose antagonized this effect, but naloxone produced no significant hyperalgesia when given alone. This suggests that opioid peptide mechanisms may mediate the analgesic action of laser therapy of the cranium.

Recently, the effects of laser irradiation on peripheral nerves and their potential effects on analgesia have been reviewed.[77] A total of 44 studies were included in this review. In 13 of 18 human studies, pulsed- or continuous-wave visible and continuous-wave infrared laser irradiation slowed nerve conduction velocity and reduced the amplitude of compound action potentials. In 26 animal studies, infrared laser irradiation suppressed evoked action potentials, pain-related neurotransmitters, such as substance P, and proinflammatory mediators, such as bradykinin. Disruption of axonal flow and microtubule arrays may be responsible for neural inhibition. The varicosities that form as a result of laser therapy inhibit axonal flow and block the transport of ATP, which is necessary to generate action potentials. The authors concluded that a range of inhibitory effects on peripheral nerves has been demonstrated, which may reduce acute pain by inhibiting peripheral nociceptors. In chronic pain, spinal cord changes induced by laser irradiation may result in depression of pain. Laser therapy seems to be most effective in reducing nerve conduction velocity when it is applied to several points over the nerve, causing an additive effect as compared with irradiation of a single point. In addition, continuous-wave laser therapy appears to be more effective than pulsed-wave application.

Laser acupuncture is another potential method of achieving pain control that has recently generated interest.[74] Laser acupuncture is the stimulation of traditional acupuncture points with low-level laser. Most animal models of laser acupuncture studies have shown no benefit over placebo treatment, although the power used in those studies was generally very low. The results of many studies of laser acupuncture in people are hampered by inappropriate or poor description of laser application. Many of these studies have shown no positive benefits, but they used very low power. One study of laser-mediated acupuncture anesthesia (2.8-6 mW, 632.8 nm) for tooth extraction or minor facial surgery reported a 95% success rate. However, the characteristics determining the success rate were not defined.

A randomized, double-blind, placebo-controlled study of laser therapy in 90 subjects with chronic neck pain was conducted to determine the efficacy of 300 mW, 830 nm laser.[4] Subjects were randomized to receive 14 treatments over 7 weeks with either active or sham laser to painful regions of the neck. Assessments were made at baseline, week 7, and week 12. Mean VAS pain scores improved by 2.7 in the treated group and worsened by 0.3 in the control group. However, the treated group began the study with higher mean VAS scores, despite randomization. Significant improvements were also seen in the laser therapy group compared with placebo for other total outcome measures, but some component portions of assessments did not differ significantly between the two groups. Laser therapy, at the parameters used in this study, was efficacious in providing pain relief for patients with chronic neck pain over a period of 3 months.

The results of laser therapy on acute and chronic back pain may differ from those obtained from treatment of neck pain. A randomized, double-blind, placebo-controlled study compared the effectiveness of laser therapy on pain and functional capacity in people with acute and chronic low back pain caused by lumbar disk herniation (LDH).[78] Patients were randomly allocated into four groups. Group 1 (acute LDH, n = 20) received hot pack plus laser therapy; group 2 (chronic LDH, n = 20) received hot pack plus laser therapy; group 3 (acute LDH, n = 20) received hot pack plus placebo laser therapy, and group 4 (chronic LDH, n = 20) received hot pack plus placebo laser therapy, for 15 sessions over 3 weeks. Pain, ROM, and disability were evaluated using standard techniques, such as VAS and global assessment scores, before and after 3 weeks of treatment. After treatment, there were significant improvements in pain severity, patients' and physicians' global assessment, ROM, and disability scores in all groups. However, no significant differences were detected between the four treatment groups with respect to all outcome parameters. There were no differences between laser and placebo laser treatments on pain severity and functional capacity in patients with acute and chronic low back pain caused by LDH.

A randomized, double-blind study of 100 patients with neck and shoulder pain indicated that 90% of the patients in the treated group had at least a 30% improvement in the degree of pain relief, as compared with only 14% of the patients in the placebo group.[79] Most patients had reduction of their pain immediately after treatment, and the improvement was typically maintained for 24 hours. A follow-up

study of another 100 patients indicated that 65% of the treated patients had improvement in their pain, but only 12% of untreated patients improved.[79]

A metaanalysis evaluated the effect of laser therapy on pain relief.[18] Nine papers met the inclusion criteria for pain control. The overall treatment effect for pain control was positive. Another review of laser therapy with location-specific doses for pain from chronic joint disorders suggested that some benefit may be derived from the use of lasers.[80] A literature search identified 88 randomized, controlled trials, of which 20 trials included patients with chronic joint disease. Laser therapy was applied within the suggested dose-range to the knee or temporomandibular joint capsule to reduce pain in chronic joint disorders. The results showed a mean difference in change of pain using a VAS by 45.6% in favor of laser therapy. Global status was also improved 33.4% more in patients in the laser therapy group. Although laser therapy appeared to reduce pain in patients with chronic joint diseases, the heterogeneity in patient samples, treatment procedures, and trial design calls for cautious interpretation of the results.

Peripheral Nerves and Spinal Cord

Peripheral nerve and spinal cord injuries can be devastating to patients of all species. Studies have been recently conducted which, along with the few known side effects of laser therapy, make this an exciting potential treatment for nerve and spinal cord injuries.

Initial studies evaluated the use of laser therapy on peripheral nerve regeneration in a crushed sciatic nerve model in rats. Most of the studies indicated that laser therapy was effective in promoting nerve recovery.[81-83] Laser therapy was applied directly to the nerve or transcutaneously. With direct stimulation, wavelengths of 540, 633, and 780 nm were found to be effective. Subsequent studies indicated that laser therapy applied to the nerve injury and the corresponding segments of the spinal cord with 633 nm laser at a dose of 10 J/cm^2 seems to improve the outcome. Although it appears that laser therapy is most effective when applied during the early posttraumatic period,[84] laser therapy may also be beneficial for chronic nerve conditions. A study of human patients with incomplete peripheral nerve or brachial plexus injuries present for 6 months to several years indicated that laser therapy resulted in progressive improvement of peripheral nerve function.[85]

Studies of rat sciatic nerve injuries indicated that there was increased functional activity, decreased scar tissue formation, decreased degeneration of motor neurons, and increased axonal growth and myelinization with laser therapy applied to the spinal cord immediately after wounding, and for 30 minutes daily for 21 days using 16 mW 632 nm HeNe laser.[86] This study suggested that laser therapy applied directly to the spinal cord may improve the recovery of corresponding peripheral nerve injuries. Additional studies on laser therapy applied to surgically repaired peripheral nerves have also shown promise in studies of rats.[87-89]

Laser therapy appears to enhance axonal sprouting, resulting in accelerated healing. Laser therapy applied to the corresponding segments of the spinal cord also helps to reduce the degenerative changes in the neurons and helps induce proliferation of astrocytes and oligodendrocytes, which may result in an increased ability to produce myelin.[89] In addition, laser therapy may enhance the production of proteins and growth factors associated with axonal sprouting, such as growth-associated protein-43 and calcitonin gene-related peptide, and may stimulate the proliferation of Schwann cells.

In an initial study, laminectomy and transection of the spinal cord at T12-L1 were performed in 17 dogs.[85] An autograft of the sciatic nerve was implanted in the injured area. Ten dogs received laser therapy for 20 days, and the others did not. The 7 that did not receive laser therapy were paralyzed, whereas the 10 treated dogs stood between 7 and 9 weeks, and walked between 9 and 12 weeks. Treated dogs did not have prominent scar tissue, and there were new axons and blood vessels originating in the spinal tissue and extending into the graft. However, the study did not report the results of neurologic testing to know whether or not the dogs were spinal walking or had functional walking ability.

More recently, a prospective study was performed to determine if low-level laser therapy and surgery would result in earlier ambulation than surgery alone for intervertebral disk herniation.[90] Thirty-six dogs with acute paralysis or nonambulatory paraparesis were randomly assigned to a surgery or surgery plus laser group. A 5 × 200-mW 810-nm cluster array laser was used to deliver 25 W/cm^2 to the skin over the spinal segment associated with the hemilaminectomy and the two adjacent ones (one cranial and one caudal). However, the dose in joules per cm^2 was not clear. The laser array was applied to each area for 5 days or until dogs became ambulatory with paraparesis or ataxia. The time to achieve this level of recovery was significantly lower in the low-level laser therapy group (median 3-5 days) than the control group (median 14 days). The authors concluded that low-level laser therapy in combination with surgery decreases the time to ambulation in dogs with T3-L3 myelopathy secondary to intervertebral disk herniation.

The use of transcranial laser or LED therapy has recently been reviewed for the treatment of stroke, traumatic brain injury, and neurodegenerative diseases in people.[91] There is some apparent transmission of near infrared light through the scalp and skull to stimulate the cortical surface of the brain. Because cortical neurons are rich in mitochondria, these cells may be responsive to light therapy. There may be upregulation of cytoprotective antioxidant enzymes, heat shock proteins, and antiapoptotic

proteins with light therapy, perhaps helping to preserve neurons afflicted with hypoxic or traumatic events.[91] In addition, light therapy may increase neurogenesis, which has been demonstrated in rat models of stroke.[92,93] In particular, there may be newly formed neuronal cells in the area ipsilateral to the lesion; however, there may be no new cells in the area of the lesion. Transcranial laser therapy may increase ATP in cortical neurons.[91] In human stroke patients, transcranial laser therapy may improve outcome when applied approximately 18 hours after moderate stroke over the entire head.[94] Transcranial laser treatment has also been investigated in mild traumatic brain injury in mice and has beneficial effects. There are also cases of treatment of people with reduced cognitive function after brain injury in which cognition was improved after laser treatment.[91]

Application of Laser Therapy to Dogs

Before applying laser therapy to a patient, there are two fundamental attributes that must be established. First, the type of laser must be known, as well as the wavelength. The wavelengths of most lasers used for laser treatment are typically in the infrared or near-infrared range of 600-1000 nm. Wavelengths of low-power lasers commonly used are 632.8 nm (HeNe, gas) in the visible light range, 810 nm (GaAlAs, diode), and 904 nm (GaAs, diode) in the infrared region of the light spectrum. The wavelength is the prime determinant of tissue penetration. Lasers that do not penetrate as deeply (630 to 740 nm) are suitable for acupuncture point stimulation and wound healing but have not proved their clinical effectiveness with deep-seated musculoskeletal conditions. Infrared lasers (750 to 1500 nm) penetrate more deeply and are used to treat trigger points, ligaments, joint capsules, and intraarticular structures. The output power (watts or milliwatts) must also be known. Based on this, and the condition to be treated, the dose of laser light (J/cm^2) is determined. For most conditions, a dose of 1 to 8 J/cm^2 is typically applied. The time the laser must be applied to an area to deliver the dose must be calculated. For example, if a 904-nm laser with a maximum output power of 250 mW is used, it will take 4 seconds to deliver 1 J.

$$0.250\ \text{W} = 1\ \text{J}/\text{X seconds}$$

$$(0.250\ \text{W})(\text{X seconds}) = 1\ \text{J}$$

$$\text{X seconds} = 1\ \text{J}/0.250\ \text{W}$$

$$\text{X} = 4\ \text{seconds}$$

With this particular laser, it will be necessary to hold the laser on one point for 4 seconds to deliver 1 J of energy. With a 1-W laser, this takes 1 second. It is crucial to always understand what the particular laser unit possesses with regard to power and wavelengths, and the dose, in J/cm^2, a condition requires. From there, it should be easy to determine how much time is needed for laser application. Some laser units compute this with preprogrammed software, whereas others require the therapist to calculate how many joules are needed and how many joules are emitted per second. Some units may take 1 second or less to deliver 1 J, whereas other units may take up to 20 seconds to deliver 1 J.

The greater the power density and longer the wavelength, the deeper the penetration is through tissues. Unfortunately, the optimal wavelengths, intensities, and dosages have not been adequately studied in dogs, and information in people is difficult to interpret because of different conditions and treatment regimens. More laser dosage is not necessarily better, because overdosing may retard the desired effect. A common mistake in using laser therapy, especially in wound healing, is to experience a positive result and automatically assume more will be better. The healing process can only progress at a certain rate.

Laser therapy is generally administered with a handheld probe, with a small beam area that is useful to treat small surfaces; other lasers have several beam areas in the same unit to treat larger areas (Figure 21-9). Laser energy may be applied with the laser probe in contact with the skin, which eliminates reflection from the skin and minimizes beam divergence, or with the probe not held in contact. With the noncontact method, it is necessary to hold the probe perpendicular to the treatment area to minimize wave reflection and beam divergence. Noncontact application is recommended for wound treatment. The appropriate dosage may be applied to larger areas by administering the calculated dose to each individual site in a grid fashion, or by slowly moving the probe over the entire surface, being certain to evenly distribute the energy to each site. In any case, the probe should be held perpendicular to the skin. A coupling medium is not necessary, as in ultrasound, because the laser beam is not attenuated by air.

To maximize laser application, the hair should be clipped because 50% to 99% of the light may be absorbed by hair. Little is known about the transmission of laser light to deeper tissues in darker dogs, but HeNe laser energy is likely to be absorbed because of the pigment. It has been recommended that the dose be increased by 25% in treatment areas with dark pigmentation. Any iodine or povidone iodine should be washed off the area. Any topical medications, especially corticosteroids and other photosensitizing agents, should be removed. The therapist should wear protective eye wear, because damage may occur to the retina if the laser shines into the eyes, and the patient's eyes should also be protected (Box 21-3).



Figure 21-9 Laser therapy is generally administered with a handheld probe, with a small beam area that is useful to treat small surfaces.

Box 21-3 Key Points for Laser Usage in Dogs

1. Clip hair.
2. Measure area to be treated (a playing card is 57 cm^2).
3. Determine treatment dose.
4. Increase dose for dogs with dark skin by 25%.
5. Determine the number of J/cm^2, total joules, and the length of treatment time for laser application. If treating an area the size of a playing card with 10 J/cm^2, the total treatment is 600 J. If using a 10-W laser, the treatment time is 60 seconds.
6. Place safety goggles on all in the immediate area.
7. Hold laser perpendicular to the skin (direct contact minimizes laser light reflection; noncontact application is recommended for wounds).
8. Apply laser treatment using an overlapping grid technique to be certain that the treatment is applied equally to all areas.

Application for Various Conditions in Dogs

Laser therapy has been used for the treatment of wounds; pain; inflammation; OA; and muscle, ligament, and tendon injuries. In general, Class IV laser energy is applied in an overlapping grid or fanning fashion, always moving the laser to avoid heating the area. Other lasers may be applied using a point-to-point application of a dose per unit area. One can imagine the area covered by a grid with square centimeters, and the laser treatment is given in the squares. The following doses have been suggested for treating various conditions. However, there is currently little or no research available regarding dose or frequency of treatment in dogs for different conditions; the information is extrapolated from research in other species and anecdotal information and should be used with caution (Box 21-4).

Open Wounds

Clinically, laser therapy has been applied to both traumatic and surgical wounds, lick granulomas, abrasions, and skin conditions associated with allergies. The causes and sources of lick granulomas should be treated appropriately to complement the laser treatments. Laser therapy assists with the reduction of inflammation and wound healing, as indicated previously. Pain should also be considered in wound management as well, and laser therapy may be helpful to provide some pain control. Traumatic wounds also need to be treated appropriately with regard to infections, wound dressings, and other related issues. Debriding and cleaning should be performed before laser therapy is performed. When treating wounds, it is beneficial to document the size of the wound with photographs and measurements. The wound should be measured at each treatment session to document progress or lack of progress. Corresponding medications and bandage changes should be documented as well.

Traumatic wounds, such as degloving injuries, are understandably very painful and the animal may not tolerate direct contact. The laser head should be held above the wound to avoid contamination of the wound. The head should be cleaned thoroughly with rubbing alcohol prior to and after treatment. Most laser heads have a small lip or surface on them, preventing direct contact with the lens and the wound. Laser treatment is applied directly to the wound at a treatment dosage of 2 to 8 J/cm^2. Acute wounds may be treated with a lower dosage daily for the first 7 to 10 days. The entire wound area should be treated, including the periphery of the wound.

Postsurgical Wounds

Postsurgical wounds may be treated immediately after the surgical procedure to assist with healing. The postsurgical wounds appropriate for laser are not limited to orthopedic and neurologic cases. General postsurgical wounds, including spays, other abdominal surgeries, dewclaw removals, benign soft tissue removals, and other soft tissue surgeries, may respond to laser therapy. Laser treatments assist with cell proliferation, antiinflammation, and DNA synthesis. Because of this, surgical wounds following removal of neoplasia should not be treated with laser.

Box 21-4 Dose Guidelines for Various Uses in Dogs

Analgesic Effect

- Muscle pain: For acute pain apply 2 to 4 J/cm^2; for chronic pain, apply 4 to 8 J/cm^2.
- Joint pain: Apply 4 to 6 J/cm^2 for acute pain, and 4 to 8 J/cm^2 for chronic pain.

Antiinflammatory Effect

- Acute and subacute: Apply 1 to 6 J/cm^2.
- Chronic: Apply 4 to 8 J/cm^2.

Open Wounds

- Acute wounds: Apply 2-6 J/cm^2 sid for 7-10 days.
- Chronic wounds: Apply 2-8 J/cm^2 sid.
- Laser head should not be applied directly to the wound.
- Clean laser head before and after treatment.
- Do not apply laser to wounds following removal of neoplasia.

Postsurgical Wounds

- A daily dose of 1-3 J/cm^2 is recommended for the first 7 to 10 days if possible, followed by a 1- to 2-day break, continued until the wound is healed. If daily treatment is not possible, then treatment three times per week may be performed.

Lick Granulomas

- Administer 1-3 J/cm^2, directly over the entire granuloma and at least 1 cm from the periphery. Depending on the size of the granuloma, the wound should be treated as frequently as possible; daily to a few times a week is beneficial. The granuloma should be treated until the wound is healed and hair growth has resumed.

Osteoarthritis

- Administer 8-10 J/cm^2.
- Treat along the joint lines and surrounding area.
- Hip: Start treatment at the greater trochanter, then direct around the cranial, medial, and caudal surfaces of the hip in a circumferential pattern.
- Stifle: Start at the patella. The joint line may then be followed either medially or laterally to complete the full circumference.
- Hock and digits: Start below the point of the hock and circle around the hock while applying treatment. It is important that the anatomy of the tarsus be considered, with its distal extent at the tarsometatarsal junction, to be certain that the appropriate areas are treated. The calcaneal tendon may also be treated. Individual toes may be treated but it is recommended that a small laser apparatus be used.
- Shoulder: Start at the greater tubercle, continuing circumferentially around the joint. Because of the tissue depth in this area, a longer wavelength laser source is necessary. If the biceps tendon or other tendons are involved, the treatment should include the length of the tendon.
- Elbow: Start below the olecranon process and continue around the entire joint in a circumferential manner. Treating distally and proximally from the elbow also benefits the surrounding soft tissue.
- Carpus and digits: Start at the accessory carpal pad and then treat in a circumferential manner. Sweeping motions may be performed distally and proximally to cover the distal and proximal aspects of the joint as well as the surrounding soft tissue. The digits may be approached the same way, although it is sometimes difficult to move in a circumferential manner. The dorsal and palmar aspects of the digits may be treated.
- Cervical spine: Treat the entire cervical spine from the suboccipital area down to the upper thoracic region. The laser may be applied directly over the dorsal cervical spine, and then moved over the epaxial musculature on both sides. Do not apply the laser for a prolonged period over the carotid arteries.
- Thoracic and lumbar spine: Treat directly over the area as well as the epaxial and surrounding musculature.
- Tendon conditions (biceps tenosynovitis, supraspinatus tendonitis, patellar tendonitis, and other tendon inflammatory conditions): The length of the superficial aspect of the tendon should be treated along with the surrounding soft tissues.

sid, Once daily.

The laser apparatus should be held directly over the wound and either moved in a sweeping fashion or held directly over the wound. The recommended treatment schedule is 1-3 J/cm^2 daily for the first 7-10 days, with a 1- to 2-day break, then continuing laser treatment until the wound is healed. Realistically, if the animal is not in the hospital, it may be difficult to laser the wound daily. Therefore as frequent treatments as possible should be pursued, with a minimum of three times per week. Laser therapy should continue a few times per week until the wound has closed and healthy tissue has formed. Clinically, steps should be taken to avoid self-mutilation by the dog, such as application of an Elizabethan collar.

Lick Granulomas

Lick granulomas are a common problem seen in small animal practice and may be treated with laser therapy. The source of the lick granuloma must be initially identified and treated appropriately, and then the wound may be addressed. There are often underlying causes of the lick granuloma. The lick granuloma should be treated with 1-3 J/cm^2, directly over the granuloma and at least 1 cm from the periphery. Laser treatment contributes to wound healing, decreases inflammation, and also contributes antihistamine effects. Depending on the size of the granuloma, the wound should be treated frequently. Daily to a few

times a week is beneficial. The granuloma should be treated until the wound is healed and hair growth has resumed. However, the underlying cause must be determined and treated, whether it is anxiety, neurologic, orthopedic, or the result of other causes.

Osteoarthritis

As in human medicine, OA is very common in dogs. The initial step when addressing the arthritic patient is a thorough assessment of ROM, weight-bearing status, girth measurements, palpation, overall function and owner perception of problems with home activities. A pain scale may be used to determine the level of pain. Weight-bearing status may be determined through dynamic or static forceplates, or scales. The overall function and goals of the owner should be determined in the evaluation, and progress using the various assessment tools should be monitored.

After the problem list and the goals have been determined, treatment with laser therapy may be implemented as part of a multimodal treatment program. The specific goals of laser therapy often include decreased pain, inflammation, and improved circulation. Arthritic joints should be thoroughly treated along the joint lines and surrounding area (Figure 21-10). Recommendations for arthritic joints have ranged from 4 J/cm^2 up to 30 J/cm^2, but more appropriate doses may be 8-10 J/cm^2. The apparatus may either be held directly over the area for the designated number of joules, or used in a sweeping fashion depending on the unit.

Hip

Canine hip dysplasia and resultant OA are very common. The coxofemoral joint should be completely treated. A good landmark to start treatment is the greater trochanter. The treatment should then be directed around the cranial, medial, and caudal surfaces of the hip. The treatment is directed in a circumferential pattern. Referred pain or secondary pain should also be addressed. The lumbosacral area and the musculature of the hip are often painful because of compensations and may also be treated.



Figure 21-10 Treatment of an arthritic joint with a therapeutic laser.

Stifle

Arthritic stifles are easily treated with therapeutic laser. A nice starting point is the patella. The joint line may then be followed either medially or laterally to complete the full circumference. The patella ligament may be treated proximally and distally. Cranial cruciate repairs or deficient stifles often have soreness in the distal hamstring muscles and patients may benefit from treating this area as well.

Hock and Digits

Hock and digit joints are often difficult to treat with modalities such as cryotherapy or moist heat because of their size and irregular contour. Laser therapy is an easy treatment for these areas. Arthritic conditions of the hock may be treated by beginning below the point of the hock and circling around the hock while applying treatment. It is important that the anatomy of the tarsus be considered, with its distal extent at the tarsometatarsal junction, to be certain that the appropriate areas are treated. The calcaneal tendon may also be treated. Individual toes may be treated, but it is recommended that a small laser apparatus be used. If only a large unit is available, it may be used in a sweeping motion over the affected digits.

Shoulder

The shoulder and affected areas are easily treated. A good starting point is the greater tubercle, with the laser continuing circumferentially around the joint. Because of the tissue depth in this area, a longer wavelength laser source is necessary. If the biceps tendon or other tendons are involved, the treatment should include the length of the tendon. The cervical region and the cervical strap muscles may benefit from treatment because of possible secondary compensations that may occur from shoulder issues and lameness.

Elbow

The elbow is a very common area for arthritic changes and may benefit from laser therapy. The laser should be directed around the entire joint in a circumferential manner. A good starting point is below the olecranon process; treatment should then proceed around the area. Treating distally and proximally from the elbow also benefits the surrounding soft tissue, in particular the triceps muscles. Secondary and tertiary soreness and pain may be noted in the shoulder and the carpal region, and they may benefit from laser treatment.

Carpus and Digits

The carpal joint usually responds well to laser therapy to help reduce the inflammation and pain associated with

arthritic changes. Arthritic digits may also be aided by therapeutic effects of laser, in addition to sesamoiditis. The carpal joint should be treated in a circumferential manner. The accessory carpal pad is a good starting point; the laser should then be directed around the joint. Sweeping motions may be performed distally and proximally to cover the distal and proximal aspects of the joint as well as the surrounding soft tissue. The digits may be approached in the same way, although it is sometimes difficult to move in a circumferential manner. The dorsal and palmar aspects of the digits may be treated, however.

Cervical Spine

The entire cervical spine should be treated from the suboccipital area down to the upper thoracic region. The laser may be applied directly over the dorsal cervical spine, and then moved over the epaxial musculature on both sides. Caution should be exercised to be certain that the laser is not applied for a prolonged period over the carotid arteries. The cervical musculature is thick and arthritic conditions of the cervical spine may benefit from the effects of laser.

Thoracic Spine

The lower thoracic spine is especially affected with arthritic changes and compensations. Treating directly over the area as well as the epaxial and surrounding musculature may be very beneficial.

Lumbar Spine

The lumbar spine, surrounding areas, and musculature may be treated the same as the thoracic spine.

Tendon Conditions

Biceps tenosynovitis, supraspinatus tendonitis, patellar tendonitis, and other inflammatory conditions involving tendons may be treated with laser therapy. The length of the superficial aspect of the tendon should be treated along with the surrounding soft tissues. The biceps tendon communicates with the shoulder joint capsule; therefore the shoulder joint capsule should be treated as well.

Precautions Regarding the Use of Laser Therapy

In general, the thermal effects of laser may injure tissues, especially the eyes, and in some cases, skin. Even small amounts of laser light can cause permanent damage to the retina.[10] Because lasers have coherent light, even a small amount of laser light may be focused on the retina through the eye, and as little as 10° C increase in temperature may cause permanent damage to the retinal photoreceptor cells. The visible and near-infrared regions of the electromagnetic spectrum may penetrate into the eye, but because there is visible light, the blink reflex helps protect against damage. Infrared laser light is not visible, and therefore a blink reflex does not occur as with bright visible light. A person exposed to laser light in an eye may not experience any pain or discomfort until a pop is heard as a result of explosive boiling and damage to the retina.[10] Caution must be used to select appropriate eyewear to be certain that the particular wavelength will be blocked and that an appropriate optical density is used to attenuate the beam power (Figure 21-11).



Figure 21-11 Protective eyewear should always be used when using a laser therapy.

Box 21-5 Precautions Regarding Laser Therapy

- Use protective eye wear.
- Do not direct laser treatment to the eye.
- Use caution with laser beam reflection from metal surfaces.
- Use caution with treatment in the presence of the following conditions:
  - Pregnancy
  - Open fontanels
  - Growth plates of immature animals
  - Malignancies
  - Photosensitive areas of the skin
- Dark colored skin, tattoos, and hair can absorb laser light and undergo excessive heating, resulting in discomfort.

Other contraindications and precautions to laser therapy include pregnancy, treatment over open fontanels or growth plates of immature animals, over malignancies, and over photosensitive areas of the skin.[5] Dark-colored skin, tattoos, and hair can absorb laser light and undergo excessive heating when a Class IV laser is applied, resulting in discomfort (Box 21-5).

Summary of Laser Therapy

Lasers are a potentially useful tool in veterinary rehabilitation. Although their use remains controversial, several studies have demonstrated benefit to using laser therapy.

Especially promising for their use in veterinary rehabilitation are studies showing preservation of cartilage properties with treatment, improvement in peripheral nerve injuries, and as a possible adjunct to managing pain, such as in patients with OA. Laser therapy also appears to have benefit in early wound healing. Laser therapy is noninvasive and there are no reported side effects when used properly.

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22 Applications of Extracorporeal Shockwave in Small Animal Rehabilitation

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Extracorporeal shockwave therapy (ESWT) has been in use since the 1970s, when its original application was to provide a noninvasive means to fragment uroliths, allowing for subsequent passage of the small fragments through the urinary tract.[1-3] Extracorporeal shockwaves have also been applied to the fragmentation of sialoliths, choledocholiths, and pancreatoliths.[4] Applications to enhance bone healing began after observations of the effects of shockwaves on the pelvis during animal lithotripsy studies.[5-7] These studies showed that a significant osteogenic response, specifically callous formation and bony remodeling, was elicited after exposure to extracorporeal shockwaves.

The use of ESWT has also been investigated for the treatment of human musculoskeletal diseases such as pain associated with plantar fasciitis, lateral epicondylitis, calcifying tendinitis, and femoral head necrosis, as well as for the treatment of delayed union and nonunion fractures.[1,8,9] Integration into treatment of soft tissue disorders, such as treatment of pain and erectile dysfunction associated with Peyronie's disease or hypertonia secondary to stroke, has also gained favor.[10,11]

The use of ESWT in equine musculoskeletal disorders includes insertion desmopathies, bone spavin, tendon and ligamentous calcification, navicular disease, exostoses, fractures and microfractures, back pain, and osteoarthritis (OA) of tarsometatarsal and distal intertarsal joints. Extracorporeal shockwaves have been used in dogs as a part of the treatment regimen for hypertrophic nonunions, tendonitis, spondylosis, and OA[4-6,12] (Figure 22-1).

How Shockwaves Are Generated

Extracorporeal shockwaves are acoustic waves of high pressure and velocity produced outside of the body. These waves are characterized by high-amplitude acoustic pressures (20-100 megapascals [MPa]) with a short build-up time of approximately 5-10 nanoseconds, an exponential decay to baseline to a negative deflection of approximately 10 MPa, and a wave cycle time of 300 nanoseconds (Figure 22-2).[2,6,13,14] These pressure waves differ from ultrasound waves because of their lower frequency, minimal tissue absorption, and lack of a thermal effect.[2]

Focused generators produce pressure waves in a fluid medium when electrical energy is converted to mechanical energy. These waves may be focused by reflection of the pressure waves within the generator to the focal point, which is at the treatment site (Figure 22-3).[6,13] The focused waves behave as sound waves in the tissues, releasing energy when tissue density changes are encountered (i.e., ligament to bone interface).[3,6] At the interface of density change, part of the wave is transmitted while the other part is reflected.[7] The greater the change in tissue density, the greater the energy released.

Three types of focused shockwave generators exist: electrohydraulic, electromagnetic, and piezoelectric. Piezoelectric generators use crystals that expand and deform when stimulated by high-voltage electricity, initiating a pressure wave.[2,7,13] The electromagnetic system consists of coils arranged in such a way as to generate opposite magnetic fields when high voltage electricity is applied.[2,7] When a high current pulse is released through the coil, a strong magnetic field is generated, creating a high current in the opposing metal membrane. The membranes are forced away from each other as a result of the opposing currents, causing compression of the surrounding fluid medium, resulting in a shockwave.[2,7,13] Electrohydraulic generators use a high-voltage spark gap, causing plasma bubble generation. Expansion of the bubble results in sonic pulse propagation and subsequent collapse results in a reverse pulse, manifesting a shockwave (Figure 22-4).[2,7,13]

Delivery of Energy

Delivery of energy to tissue requires the use of coupling gel in conjunction with a percussive handheld applicator. Energy can be focused via spherical arrangement of crystals, spherical generator, or acoustic lenses. Focusing energy results in greater tissue depth penetration, up to 110 cm, with a rapid decline of energy at the periphery, allowing an intense amount of energy delivery to a relatively small area.[6,13] Eighty percent of the maximum shockwave pressure resides in the focal area. Hence, its maximum value is in the focus with a decrease in pressure at the periphery of the focal area.[4] Piezoelectric generators have a small focal volume with high-energy flux values and low overall energy



Figure 22-1 A focused extracorporeal shockwave unit.

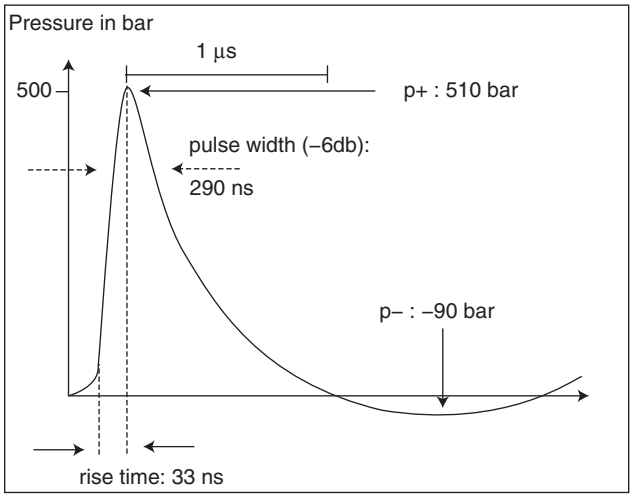


Figure 22-2 Pressure profile of an extracorporeal shock wave. Note the rapid increase to a relatively high pressure, followed by a rapid decline. (Courtesy of Pulsevet, Inc.)

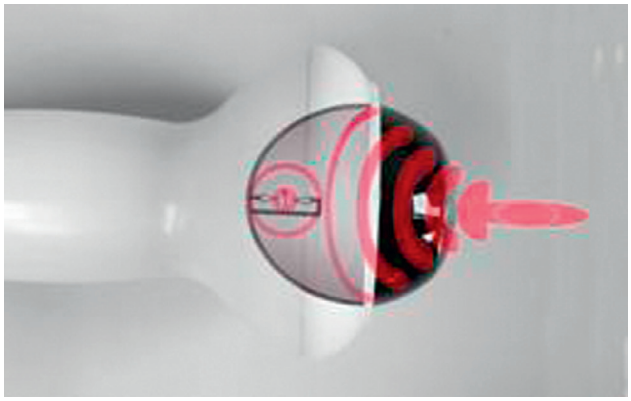


Figure 22-3 A trode used to apply extracorporeal shockwaves to a focused tissue depth. (Courtesy of Pulsevet, Inc.)

transfer. Electromagnetic generators also have a small focal volume and high-energy flux, but tend to be less concentrated than the piezoelectric system. Electrohydraulic generators produce the largest focal volumes with lower energy flux density and overall high energy transfer.[13]

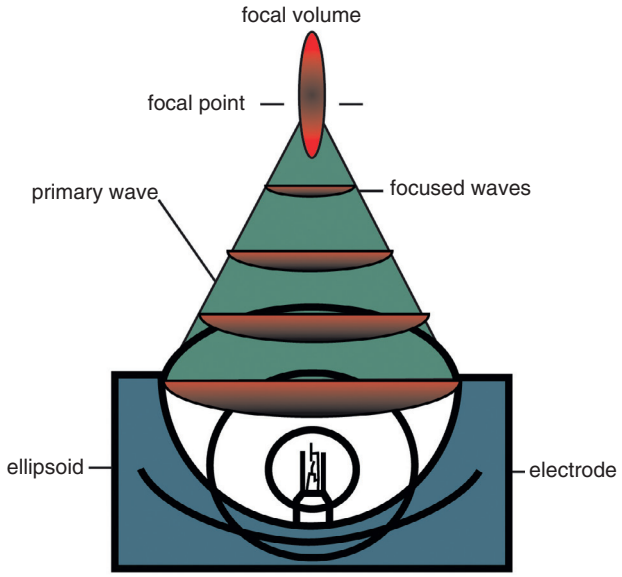


Figure 22-4 A spark gap focused shockwave generator. The shape of the parabola determines the depth of penetration of focused extracorporeal shockwaves. (Courtesy of Pulsevet, Inc.)

An alternative to focused extracorporeal shockwaves exists as a radial extracorporeal shockwave device, or a ballistic generator. Shockwaves from this device are generated pneumatically (mechanical concussion) and are propagated through tissues in a radial pattern. Radial pressure waves have lower energies, characterized by a slower rise time, and a negative component that is the same magnitude as the positive component.[15] The energy applied by this device is greatest at the applicator-skin interface and decreases proportionally to the square of the distance from the applicator head, applying energy to a wider circumference with decreased tissue penetration.[12,13,15-17] The rapid release of energy occurs over a relatively wide area with a rapid decrease in energy delivered to deeper tissues.[5,6,12]

Research Regarding Extracorporeal Shockwave Therapy

General Biologic Effects

The primary effects of shockwaves are produced by the generation of mechanical forces. Secondary effects occur by indirect generation of mechanical forces through cavitation. Compression and tension generation occurs as the shockwave travels through the tissue. Varying amounts of reflection and transmission of energy occur at varying interfaces. In bone, for example, the amount of energy attenuation depends on the amount of cancellous bone, because attenuation of the shockwave increases exponentially with increased bone thickness.[9] This is largely due to the various tissue interfaces; hence most of the energy from the shockwave is deposited in cancellous bone.[2,6]

Cavitation bubbles are made during the tensile phase of the shockwave, and as size increases, a large amount of energy is delivered to the bubble. Cavitation bubbles formed secondary to shockwave-generated compression and tension forces have the potential for deposition of large amounts of energy after subsequent collapse, releasing high-energy water jets and high-temperature production.[3,7] Cavitation at the surface interface is the mechanism by which fragmentation of uroliths occurs.[3] Cavitation also leads to the production and release of free radicals, ultimately creating chemical reactions within the tissue. ESWT may also increase cell membrane permeability. The growth and collapse of cavitation bubbles, which may lead to surface deformation and damage is one potential mechanism of action for increasing membrane permeability.[18]

A dose-dependent cytotoxicity or decrease in cell viability is present with increased numbers of waves and energy density. Different cell populations differ in their sensitivity to shockwave energy. Bone marrow, for example, has a decreased sensitivity compared with malignant cell lines.[19] Human fibroblasts have an increased sensitivity to higher numbers of shockwaves rather than the higher energy of shockwaves, decreasing the growth potential of fibroblasts while in suspension.[20] The reepithelialization rate of skin wounds in pigs is also affected by ESWT, and the effects depend on the energy level used.[4,21]

Analgesia

The mechanism of action for induction of analgesia and tissue healing is poorly understood. Human patients tend to exhibit a bimodal analgesic effect, in which an initial decrease in pain 3-4 days after initial treatment has been attributed to the direct effects of shockwaves on nociceptors and impaired synthesis of substance P.[22] After the initial analgesic effects, a gradual recurrence of pain is noted with a subsequent gradual resolution of pain noted over 3-4 weeks that is attributed to angiogenesis and tissue matrix remodeling.[22] Analgesic effects in humans appear to be more consistent in chronically affected patients rather than acutely affected patients.[23] Horses have also exhibited bimodal analgesia; however, the mechanism remains unknown.[13]

It has been hypothesized that analgesia is induced by hyperstimulation of nociceptors resulting in an intense sensory input to the periaqueductal gray area of the midbrain. This sensory input promotes descending inhibition and modulation of sensory stimulus transmission through the dorsal horns of the spinal cord, known as the "Gate Control Theory."[15,24,25] A second theory is that induction of cell damage from shockwave application prevents appropriate membrane potentials required for transmission of signals.[15] It has been shown that shockwaves induce production of cytokines and growth factors, endothelial nitric oxide synthase (eNOS) and up-regulation of bone morphogenetic protein (BMP) expression.[26-28] As a result of cytokine and growth factor release, a decline in inflammation and swelling, with short-term analgesia, is noted.

Decreases in calcitonin gene–related peptide (CGRP) in free nerve endings and dorsal root ganglia (DRG) of rats after application of ESWT to the plantar skin of the hindlimb support the theory that altered nerve function of the locally treated area may be the source of analgesic effects.[29,30] Substance P gained interest as a mode of analgesia, as an increase in basal concentration was noted 24 hours after ESWT application to the femur of a rabbit, followed by a gradual decrease in basal secretion over 6 weeks. This decrease was attributed to a potential degeneration of nerve endings in the treated area.[31] Later studies by Haake and colleagues,[32-34] did not find evidence of ESWT influence on spinal antinociceptive opioid mechanisms or on nonopioid mechanisms (substance P or CGRP). ESWT did not appear to activate peripheral nerves to induce analgesia, as evidenced by a lack of increase in c-Fos protein or its gene expression.[32] Local desensitization and subsequent reinnervation has been theorized by Murata and colleagues,[35] in which an increase in activating transcription factor 3 and growth-associated phosphoprotein 43 was measured in rat DRG neurons after treatment with ESWT. Although a definitive mechanism of action and therefore treatment protocol for analgesia still remains to be elucidated, Takahashi and colleagues[36] described a cumulative effect of shockwaves on nerve fibers on the skin of rats, suggesting that multiple applications of low-energy shockwaves may provide longer-lasting antinociceptive effects.

McClure and colleagues[25] and Abed and colleagues[37] investigated analgesia induced by focused or radial shockwaves. Twenty-four horse limbs were assigned to receive control (sham treatment), ESWT, or radial pressure wave treatment (RPWT). Analysis of cutaneous analgesia revealed a substantial increase in cutaneous threshold to electrical stimuli for ESWT and RPWT at day 3. Analysis of neuropeptide concentrations, substance P, and CGRP did not reveal a significant difference between the groups.[25,37] Sensory nerve conduction velocity was found to be statistically lower in nonfocused radial pressure waves when compared with control sensory nerve conduction. Disruption of the myelin sheath in medium- to large-diameter axons was noted under transmission electron microscopy, potentially creating a prolonged conduction velocity in radial pressure wave–treated nerves.[15]

There appear to be a number of potential mechanisms whereby ESWT exerts analgesic effects. The method of application, area of application, energy level, and frequency of application may have a role in determining whether the effect is directly related to pain pathway modification or stimulation of tissue healing.

Cartilage and Chondrocytes

There is tremendous interest in evaluating ESWT as a treatment for joint conditions, such as OA. Mueller and colleagues[8] reported on the analgesic benefit of radial ESWT for hip OA in dogs, characterized by improved

ground reaction force difference between limbs 4 weeks following shockwave therapy and a potential long-term benefit of at least 3 months following treatment. Results of analgesia were similar to a study of canine stifle OA, in which three shockwave treatments (200 shockwaves were applied to each of four sites using a 20-mm focused depth applicator, followed by 175 shocks to each of the same four sites using a 5-mm focused depth applicator, at an energy flux density of 0.14 mJ/mm^2, for a total of 1500 shocks) were administered 3 weeks apart. An improvement in peak vertical force and vertical impulse was evident at 21 days and continued to improve to the end of the study, at 98 days. Despite these improvements, the changes were not statistically significant from control groups perhaps because of small numbers of animals in each group.[1] In contrast, Francis and colleagues[38] reported significant improvement in ground reaction forces of dogs with elbow or hip OA when compared with control groups. A follow-up study of another group of dogs with elbow OA confirmed these findings and suggested that the amount of improvement with ESWT is similar to what would be expected when treating with an average nonsteroidal antiinflammatory drug (NSAID), despite the fact that the dogs were already receiving treatment for their arthritis, including NSAIDs.[39]

Several studies have investigated how ESWT may affect the processes involved in OA. One study evaluated the effects of ESWT on the metabolism of human OA chondrocytes in vitro, especially the expression of β_1 integrin, interleukin (IL)-10, and tumor necrosis factor alpha (TNF-α).[40] Human articular cartilage was obtained from patients with knee OA undergoing total joint replacement. After isolation, chondrocytes underwent ESWT with different parameters of impulses, energy levels, and energy flux density. Chondrocyte intracellular IL-10 and TNF-α levels were evaluated. At baseline, osteoarthritic chondrocytes expressed significantly higher levels and IL-10 and TNF-α levels. Following ESWT, there was a significant decrease of IL-10 and TNF-α intracellular levels. IL-10 levels decreased with any number of impulses and energy levels, whereas TNF-α decreased at middle energies. ESWT significantly reduced the nitric oxide level in the synovial cavity of knee joints in a rabbit anterior cruciate ligament (ACL) transection model as compared with untreated rabbits with cruciate ligament transection.[41] Chondrocyte apoptosis and the severity of cartilage lesions were also less in rabbits with OA treated with ESWT. The authors concluded that ESWT may be a useful treatment for knee OA because of the reduced progression of OA in rabbits. Although promising, further studies are necessary to determine if ESWT may modulate the changes seen with OA.

Another study evaluated the effects of ESWT on the development of OA in a rat ACL transection OA model.[42] OA of the knee was detected radiographically 12 weeks after study initiation in the ACL-transected group, but very subtle changes were noticed in the control non-transected knees and the ACL-transected plus ESWT groups (single treatment with 800 shocks, 0.18 mJ/mm^2). The articular cartilage of ACL-transected rats showed significant increases in cartilage degradation and chondrocyte apoptosis and significant decreases in subchondral bone remodeling compared with the control and ACL-transected plus ESWT rats. In addition, the ACL-transected plus ESWT rats had significantly decreased cartilage degradation and increased chondrocyte activity comparable to non-transected controls. The authors concluded that ESWT may have chondroprotective effects and improved subchondral bone remodeling in this rat OA knee model. In fact, ESWT of the subchondral bone may result in greatly reduced progression of OA.[43] Another study by the same group also showed that ESWT reduced cartilage changes in a rat OA model, with the most beneficial effects seen 4 weeks after ESWT. The group receiving ESWT had significantly lower histologic changes, increased collagen type II in the articular cartilage, and increased vascular endothelial growth factor (VEGF), BMP-2, and osteocalcin in the subchondral bone compared with the untreated rats.[44] Studies in a horse OA model, however, indicated no effect on subchondral bone, although increases in the serum biochemical markers osteocalcin and C-terminal telopeptide indicated bone remodeling.[45]

Clinical function and the pain of OA may also be modulated by ESWT. In a rat OA model, walking duration on a treadmill was significantly extended 4, 7, and 14 days after ESWT in rats with knee OA, but began decreasing by day 21.[46] Although OA resulted in increased expression of CGRP in DRG neurons innervating the knee as compared with normal rats, ESWT reduced CGRP-positive DRG neurons, suggesting that ESWT is a useful treatment for knee OA.

Effects of ESWT on articular cartilage are of some concern, however, because if disruption of the structure of cartilage occurs secondary to ESWT application, joint health may be compromised. Gross disruption of cartilage integrity was absent after radial shockwave application to equine third metacarpal or metatarsal bone.[16] A dose-dependent increase in lactate dehydrogenase after radial shockwave application supported the thought of increased chondrocyte membrane permeability.[16] Focused shockwave effects on articular cartilage have also been investigated and these studies indicated a lack of radiographic, histologic, and macroscopic changes within joint cartilage of growing rabbits.[47] In vitro investigation of effects of focused shockwaves on articular chondrocytes revealed a dose-dependent increase in cytotoxicity, as well as decreased proliferative potential at higher energy doses.[48] Another study evaluated a single bout of ESWT (1500 shockwaves of 0.5 mJ/mm^2) on the structural integrity of articular cartilage of rat femoral heads in vivo.[49] Although there were no changes in histologic scores after ESWT,

positive immunostaining for tenascin-C and chitinase 3–like protein 1 was found up to 10 weeks after ESWT in treated cartilage and collagen type II mRNA was increased 1 and 4 weeks after ESWT. Other changes included expansion of the rough-surfaced endoplasmic reticulum, detachment of the cell membrane, and necrotic chondrocytes. Because all of these changes have been associated with early OA, there is some concern that high-energy ESWT directly applied to cartilage might cause degenerative changes in hyaline cartilage.

Tendon and Ligaments

In collagenase-induced models of suspensory ligament desmitis, destruction of abnormal metaplastic tissue and increased numbers of collagen fibrils was noted on histologic examination, suggesting possible promotion of healing of desmitis with ESWT.[2,50] Evaluation of the bone-tendon interface in dogs and rabbits has revealed increased neovascularization after treatment, as indicated by an increase in neovessels and angiogenic markers, such as vascular endothelial growth factor, eNOS, and proliferating cell nuclear antigen expression.[51,52] In a collagenase-induced patellar tendinopathy model in rabbits, increased tensile strength, hydroxyproline concentration, and tenocyte numbers, in addition to increased neovascularization, were reported.[53]

In a rat model of collagenase-induced Achilles tendonitis, low-energy ESWT induced repair coinciding with an increase in transforming growth factor beta$_1$ (TGF-β_1) and insulin-like growth factor I (IGF-I) concentrations with tenocyte proliferation, neovascularization and progressive tissue regeneration, whereas higher energies impaired DNA, extracellular matrix synthesis, and biomechanical characteristics of tendons.[26] Increases in TGF-β_1 expression were noted in early tendon repair stages and IGF-I expression was increased throughout the healing process, indicating that TGF-β_1 and IGF-I are important mediators in ESWT-stimulated cell proliferation and tissue regeneration within a healing tendon.[26,50] An increase in metachromasia was also noted and may be an indication of increased extracellular matrix production by cells.[50,54] Increases in glycosaminoglycan and protein synthesis have been seen as early as 3 hours after ESWT application, with an overall tendency toward anabolic metabolism. Acceleration of healing may therefore be attributed to an initiation of ESWT-induced stimulation to the tissue with a subsequent decrease in cellular metabolism over 6 weeks.[55]

Increased edema and inflammatory cells in the paratenon of rabbits has been described and could indicate that although ESWT has the potential to treat various tendinopathies via induction of tissue regeneration, there is also the potential for harm of tendinous tissue with application of ESWT.[55-57] Documented changes appear to be dose dependent, either by the number of impulses or higher energy flux densities, and may decrease the mechanical strength, making these structures susceptible to tearing.[58] The amount of energy and number of pulses delivered should be taken into consideration, because tendons and ligaments appear to be more susceptible to damage from excess energy.[54-57]

Radial pressure wave application has been investigated for the treatment of various tendinopathies, including insertional tendinopathies, with treatment successes reported in up to 80% of treated patients.[59,60] In a study evaluating shockwave therapy for the treatment of supraspinatus calcifying tendinopathy, one dog exhibited subjective and objective improvement in lameness 21 days after shockwave therapy.[61] Another dog in the same study was bilaterally affected and improvement was noted at 28 and 49 days after treatment in the right limb but not in the left limb. In both cases, disruption of the calcified material was not apparent after treatment.

ESWT has also been evaluated for the treatment of patellar ligament desmitis in dogs after undergoing tibial plateau leveling osteotomy (TPLO).[62] Dogs that had TPLO surgery were evaluated by radiographs and ultrasonography preoperatively and 4, 6, and 8 weeks after TPLO. At 4 and 6 weeks, treated dogs received 600 shocks with an energy level of 0.15 mJ/mm^2, with a focused depth of application of 5 mm on the patellar ligament. There was a significant difference in distal patellar ligament thickness between groups at 6 and 8 weeks postoperatively. No significant ultrasonographic differences were found.

Soft Tissue and Wound Healing

Soft tissue healing may be promoted as a result of neovascularization and an increase in vascularity of the area. Neovascularization enhances healing and decreases pain associated with injury and can be seen as early as 4 weeks after treatment.[54] In a dorsal skin random flap model of rodents, there was a significant reduction in the distal necrotic area of flaps and a significant increase in tissue perfusion with a single treatment of ESWT when compared with control skin flaps. These findings indicate that a single dose of ESWT may enhance flap survival by increasing flap tissue perfusion. Increased expression of VEGF and proliferating cell nuclear antigen expression within the flap ischemic area indicated induction of neovascularization and cellular proliferation, a proposed mechanism for enhanced flap survival. A reduction in leukocyte infiltration and suppressed TNF-α suggested reduction of the inflammatory response; alternatively, it may result from the decrease in necrotic tissue within the ischemic zone.[63]

Schaden and colleagues[64] studied the effects of radial shockwaves for the treatment of soft tissue wounds in humans. Response to treatment, defined as 100% wound epithelialization, was seen in 75% of wounds, in which causal factors or site specificity of the wound did not influence success of treatment. Acute wounds were more likely to heal completely than chronic wounds and large chronic

wounds demonstrated suboptimal healing. Overall, the use of unfocused, low-energy ESWT on patients with acute and chronic soft tissue wounds was associated with complete closure of 75% of wounds.

Haupt and colleagues[21] described the effects of ESWT on partial thickness wound healing in piglets. Wounds treated with low doses of shockwaves had significantly enhanced rates of epithelialization. On histopathologic examination, wounds treated with low-dose shockwaves had a larger number of dilated vessels and macrophages in the perivascular space. The newly formed epithelial layer was twice the thickness as that of control wounds and wounds treated with high-dose shockwaves. In irradiated wounds, the rate of healing was increased by 50% when low-dose shockwave treatment was used.

The reepithelialization rate is inhibited with high-dose shockwaves, with intermediate energy having no effect on reepithelialization. Low energies appear to have a stimulatory effect on reepithelialization rate.[4]

Bone Healing

Bone healing is thought to be enhanced by extracorporeal shockwaves,[65-67] possibly as a result of induction of microfractures within the trabecular bone, resulting in release of osteoinductive growth factors and the formation of a fracture hematoma. Bone marrow stromal growth and differentiation of osteoprogenitor cells have been correlated with induction of TGF-β_1 and VEGF, indicating that recruitment of bone-marrow mesenchymal cells may have a primary role in ESWT promoted bone repair.[68-71] Increases in BMP-2, BMP-3, BMP-4, and BMP-7 were documented as early as 4 weeks following ESWT treatment in a segmental defect model in rats, suggesting a vital role of BMP in ESWT-induced cell proliferation and bone regeneration.[69-71] Investigation into transformation of the physical stimulus of ESWT into an intracellular mitogenic response suggests that ESWT induces mitogen-activated protein kinases, extracellular signal-regulated kinase, and p38 kinase, contributing to bone regeneration in a rat segmental bone defect.[72] Growth factors in turn induce neovascularization, and recruit inducible osteoprogenitor cells, resulting in osteoblast proliferation and bone formation.[24,70] Endothelial NO synthase and an increase in BMP expression have been associated with osteosynthesis in treated areas. In a rabbit model, treated femurs were characterized by increases in periosteal bone formation secondary to an earlier increase in periosteal and trabecular bone disruption.[27]

Energy levels that are too great may have deleterious effects on bone, however. Studies conducted on rabbits reported soft tissue and medullary edema, decreased blood flow and bone metabolism, periosteal detachment, and even fracture of cortical bone, with an increase in frequency and severity as the energy density increased.[73,74] Other findings of microdamage of cortical bone were noted in racehorses in an ex vivo model. More specifically, focused shockwaves were associated with an increase in microcrack density and radial shockwaves were associated with an increase in microcrack length.[75] In a study evaluating the acute effects of ESWT on the third metacarpal and metatarsal bone, however, McClure and colleagues[2] discovered that evidence of cortical microfractures and soft tissue damage was absent. Scintigraphic, thermographic, and histologic assessment of ESWT effects on the fourth metatarsal bone in normal horses did not reveal evidence of damage in the treated area.[76,77] Absence of microfractures is not an indication of normal mechanical properties, as accumulation of microscopic damage was not detected in canine femurs until 15% of the modulus of elasticity was lost, indicating that accumulation is threshold dependent.[12] McClure and colleagues[2] postulated that the mechanisms for stimulation of osteogenesis included bone marrow hypoxia, subperiosteal hemorrhage, increased regional blood flow and activation of osteogenic factors rather than a result of cortical microfractures.[2] Evaluation of osteons in this study revealed the potential of ESWT to provide full cortical thickness stimulation of osteonal remodeling.

In humans, treatment of nonunion fractures with noninvasive ESWT resulted in a 76% success rate, compared with a 79% success rate with surgical therapy. The success rate improved when there was greater biologic activity in the nonunion, with a success rate of 100% for hypertrophic nonunions and 36.4% success rate in atrophic nonunion fractures. Current theories for enhancement of fracture healing include increases in bone metabolism secondary to increases in bone growth factors such as TGF-β_1, basic fibroblast growth factor, VEGF, and osteocalcin measured in a rabbit atrophic nonunion model.[28] In a radial nonunion model performed in dogs, 80% of the dogs exhibited a bridging callous 6 weeks after shockwave treatment; by 9 weeks there was narrowing of the fracture gap and an increase in the bridging callous, and finally complete bony union at 12 weeks following treatment.[4] Persistent radiographic nonunion was present in four of five of the control dogs and an incomplete bone union was evident in the last control dog. Wang and colleagues[78] described the effects of ESWT on an acute fracture model on 16 tibiae of eight laboratory dogs. In this study, radiographic callous formation was not significantly increased from control specimens until 12 weeks following treatment, although measurements were consistently greater in the treated group by 4 weeks. Cortical bone at 12 weeks was histologically more dense, thicker, and heavier than control callous, signifying the potential advantage of treating acute fractures that have the potential of becoming nonunions. In addition, fractured bone treated with high-energy ESWT has increased bone mineral density, higher ash and calcium content, and higher peak load, peak stress, and elastic modulus than in untreated fractures.[67,71,79,80] Therefore it can be assumed that ESWT treatment improves the quality

and mechanical qualities of bone during fracture repair. Shockwave therapy is associated with decreased incidence of disturbed fracture healing, and may be indicated for use in patients with multiple fractures, open fractures, animals with a concurrent systemic disease, or geriatric animals.[24]

ESWT also appears to be an effective treatment for human patients with osteonecrosis of the femoral head. Wang and colleagues[81] compared use of ESWT treatment versus core decompression and bone grafting. Patients treated with ESWT had significantly improved pain and Harris hip scores at each follow up. Overall, 79% of patients showed improvement after treatment with ESWT and a trend toward a decrease in size of the lesion at each follow-up interval. In the rabbit model of avascular necrosis of the femoral head, an increase in VEGF mRNA expression was noted as early as 1 week after shockwave treatment and was maintained for 8 weeks after treatment; VEGF protein expression peaked at 4 weeks and was maintained for 12 weeks.[82] Increased neovascularization was noted within regions of new bone formation 12 weeks after initial ESWT treatment in these rabbits, leading to the conclusion that ESWT therapy may promote repair of subchondral bone in avascular necrosis of the femoral head in rabbits.

ESWT may also be useful in treating osteomyelitis. Chronic osteomyelitis was induced in rabbits by injecting *Staphylococcus aureus* into the proximal tibia in one study.[83] Focused ESWT was applied twice to the infected limbs 4 and 5 weeks after inoculation. Signs of bacterial spread were not detectable after ESWT. Systemic indicators of infection were also unchanged. However, histologic scores of osteomyelitis were significantly decreased in the ESWT group compared with the untreated control, although *S. aureus* was still detectable. This study suggests that ESWT may be beneficial as an adjunct treatment of chronic bone infections.

Cardiovascular

Recent investigation of the effects of ESWT for the treatment of cardiovascular diseases has yielded promising results. Low-energy ESWT effectively induced angiogenesis and improved myocardial perfusion and cardiac function in a porcine model.[84,85] Improvement of myocardial perfusion and cardiac function was also investigated in humans with end-stage coronary artery disease and refractory angina pectoris without adverse effects.[86,87] Uwatoku and colleagues[84] recently investigated the effects of ESWT treatment on left ventricular remodeling secondary to acute myocardial infarction. Acute myocardial infarction was induced in a porcine model and then treated with ESWT 3 days or 4 weeks after myocardial insult. Results indicated that treatment with ESWT in the acute phase resulted in improved left ventricular remodeling and increased capillary density in the infarcted myocardial border zone. Improved myocardial perfusion secondary to angiogenesis was associated with improved left ventricular function. ESWT may theoretically be extended for use in vascular complications, such as venous and arterial thrombi dissolution or modification of arterial plaques, as visible histomorphologic changes were documented when used on normal human aortic specimens in vitro.[88] Facilitation of endothelial progenitor cells secondary to ESWT has been associated with increased relative blood flow recovery in chronic ischemia of the myocardium, as well as in the hindlimb.[85,86,89] Neovascularization occurred via upregulation of VEGF as well as stromal cell–derived factor 1, a chemoattractant that plays an important role in hematopoietic stem cell homing and function.

Application of Extracorporeal Shockwave Treatment

Light anesthesia or heavy sedation of the veterinary patient is generally administered before applying focused shockwave treatment to improve patient comfort. In a study investigating the effects of radial shockwaves on the treatment of hip OA, 18 dogs were treated without the use of sedation or anesthesia without evidence of adverse effects related to pain.[8] A study in human patients compared effects of ESWT application in the absence of local anesthetics for the treatment of chronic plantar fasciitis and found that the pain score was significantly less in patients without local anesthetic.[90] The hair should be clipped over the site of application because air attenuates the sound waves, making the treatment less efficacious (Figure 22-5). The use of ultrasound gel between the skin and applicator is also necessary to eliminate air. A thorough understanding of the anatomy of the area where shockwaves are to be applied is necessary. Application of ESWT to a joint should



Figure 22-5 Application of extracorporeal shock wave treatment to an elbow joint of a dog. Note that the hair has been clipped and ultrasound gel has been applied.

be directed at the insertion of the joint capsule rather than the articular cartilage of the joint. Treatment of the entire joint capsule insertion regions may be more efficacious. When applying ESWT, the applicator should be gently moved along the joint capsule insertion sites and mildly rotated to be certain that all areas are treated. When treating ligamentous or tendinous injuries, direction of the wave should be in a proximal to distal direction. The optimal energy density, number of shockwaves applied, and number of sites per application has yet to be elucidated and guidelines for application are generally provided by the manufacturer.[4] In general, 500-1000 shocks are applied for most conditions. Wounds should be treated with low energy levels, whereas nonunion fractures and OA may be treated with higher energy levels. The optimal frequency of application is also uncertain, but generally two treatments 3-4 weeks apart are recommended for most conditions. Improvement of conditions such as OA may occur for several months after treatment.

Adverse Events and Precautions with Use of ESWT

Negative effects may be observed with application of high-energy shockwaves, a large number of shocks administered, or if application is focused on single areas of sensitive structures. Lower energy levels generally reduce the risk of deleterious effects. Caution is advised in young or developing animals, because directing shockwaves over an open physis has been implicated in the development of focal tibial growth plate dysplasia in rats.[91] However, other studies have contradicted Yeaman's results, demonstrating that under human shockwave therapy conditions, damage to the tibial and femoral epiphyses was not evident in a rabbit model.[92,93] Väterlein and colleagues[47] and Lüssenhop and colleagues[94] reported shockwave-induced intramedullary ossification with slight shortening of the treated leg in rabbits; however, this shortening was not clinically significant. Interestingly, cartilage has been found to have less detrimental effects associated with shockwave therapy, even with higher energy levels.[27] The explanation for this is unknown, but it has been postulated that the decreased vascularity of cartilage may decrease deleterious effects, as cavitation bubble formation requires free fluid to form before tissue damage takes place.[27]

The most common side effects after shockwave therapy include petechiae and bruising. High-energy flux densities may be associated with small hematoma formation and local swelling at the application site.[2,4,9] Patients may have discomfort after application, requiring the use of analgesic medications. Corticosteroids and NSAIDs are generally not used unless necessary. Interstitial damage of arterial walls can develop if they are within the focal zone, resulting in rupture, vasoconstriction, or increased permeability. Repetitive action potentials of nerves can result from cavitation induction with ESWT. As a result, major arteries and nerves should be excluded from the focal zone.[2,9] However, nerves and vessels appear to be less sensitive to the effects of ESWT as compared with other tissues.

Indications for Use

ESWT may be beneficial as an ancillary treatment for OA as part of the multimodal approach to therapy of this condition.[9] Improved weight bearing and passive range of motion are similar to results expected with NSAID use. In patients that are unable to tolerate the administration of NSAIDs, extracorporeal shockwaves may serve as a useful, noninvasive alternative for treatment of osteoarthritic conditions. Anecdotally, treatment of conditions affecting the elbow, hip, or back may respond better than other joints. ESWT has been used for delayed or nonunion fractures. Tendinopathies and ligament conditions may also respond to ESWT. Wounds may also benefit from treatment with ESWT, especially if they are large wounds treated in the acute phase of wound healing. ESWT may provide some antiinflammatory treatment of soft tissues, recently attributed to a rapid enhancement of eNOS activity in human vein endothelial cells.[95]

Contraindications to Shockwave Therapy

Extracorporeal shockwaves should not be used in dogs suspected of having immune-mediated joint disease, infectious arthritis, neoplastic disease, diskospondylitis, or unstable fractures, or in dogs with neurologic deficits. Lee and colleagues[96] studied the effects of various doses of ESWT on the spinal cord of rabbits. Neurologic deficits were not apparent after shockwave application; however, varying degrees of microscopic changes were present, including myelin damage and neuronal loss.[96] Use of ESWT near gas-filled cavities or an organ should be avoided, as the risk for damage is high because of potential high-energy release. Delivery of shockwaves over lung fields, brain, heart, major blood vessels, nerves, and a gravid uterus should be avoided. One study investigating the effect of shockwave application to pregnant rabbits at different gestational stages found that congestion and parenchymal hemorrhage was present in the liver, lung, and kidney of fetuses exposed in the early gestational period.[97]

Avoiding application to sites with neoplasia or infection has been recommended because of the potential of inducing metastasis or septicemia from physical displacement of neoplastic cells or bacteria.[2,9] In mouse melanoma and prostate tumor models, lithotripter shockwaves were applied to transplanted tumor cells on the hindlimb of each mouse. Enhanced metastasis was attributed to shockwave treatment secondary to cavitation.[98,99] Conversely, enhancement of chemotherapeutic effects was observed when regional exposure to shockwaves was used in combination

with intravenous bleomycin and cisplatin. Proposed mechanisms for this finding include increased permeability as a result of hydrodynamic flow. Combination therapy resulted in inhibition of cancer cell proliferation and tumor growth in rats, and these effects appeared to be dose dependent.[100-102]

Summary

Extracorporeal shockwave therapy appears to have several applications in canine rehabilitation. Wound healing, osteoarthritis, delayed and nonunion fractures, and ligament and tendon conditions seem to be positively influenced by treatment with ESWT. Further information is needed regarding the energy level, number of shocks, and frequency of application to provide the optimal treatment for various conditions. In addition, additional research is required to further understand the mechanisms of action.

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23 Other Modalities in Veterinary Rehabilitation

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Several alternative and complementary modalities have been used in veterinary rehabilitation, including static magnetic fields, pulsed electromagnetic fields (PEMFs), and shortwave diathermy. Other modalities that have been used in rehabilitation include acupuncture and chiropractic. Acupuncture and chiropractic have been described in detail elsewhere, have their own training programs, and are stand-alone disciplines and are not described here.

Static Magnet Therapy

Magnet therapy has been used as an alternative therapy in human medicine for many years and has gained popularity more recently in veterinary medicine. Magnets used for therapeutic purposes are either static in nature or electromagnets. Static magnets have magnetic fields that do not change, whereas electromagnets generate magnetic fields only when electrical current flows through them. Magnet strength is measured in units of gauss (G) or Tesla (T) (1 T = 10^4 G). The earth's magnetic field is approximately 0.5 G, whereas magnetic resonance imaging machines use magnets of 15,000 G and higher. Static magnets for therapeutic purposes are usually in the 300- to 5000-G range.

History

Magnet therapy in the treatment of various medical conditions dates back as early as 200 AD with Greek healers reportedly using magnetic rings as a treatment for arthritis.[1] During the Middle Ages, magnets were thought to be effective in the treatment of baldness, wounds, arthritis, and gout.[1] In the early 1600s, an English physician, Thomas Browne, may have been one of the first to scientifically evaluate magnets. He concluded that the use of magnets in the treatment of such diseases as gout, headache, and venereal disease amounted to wishful thinking.[1] Because of its continued popularity as an alternative therapeutic modality and the business that it generates, therapeutic efficacy has been studied in various models.

Magnets continue to be in use today, with a myriad of products available claiming various therapeutic values. An Internet search[2] of magnetic therapy for dogs yields products such as beds, collars, and jackets, among others, that claim to have beneficial effects on pain, blood flow, tissue oxygenation, bone and tissue regeneration, inflammation, and sleep. The current literature is sparse, however, and little convincing evidence exists for these claims. For all of these applications, increased blood flow is one theory provided to explain why magnets may have an effect. At this time no solid explanation for why magnets may have a physiologic effect seems to be available. Unfortunately, only weak evidence exists, not only for the effects of pain alleviation and bone or wound healing, but also for the theorized cause, blood flow changes. When studies have shown a potential physiologic effect, the mechanism causing the effect is often poorly explained or may be more strongly associated with a placebo effect.

Effects on Blood Flow

The Lorentz force law states that a magnetic field exerts a force on a moving ionic current.[3] The Hall effect further states that when a magnetic field is placed perpendicular to the direction of flow of an electric current, it will tend to deflect and separate the charged ions.[3] Ions will be deflected in opposite directions depending on the magnetic pole encountered and the charge of the ion.[3] In theory, when a magnetic field with a series of alternating north and south poles is placed over a blood vessel, the influence of the field may cause positive and negative ions to bounce back and forth between the sides of the vessel, creating currents in moving blood similar to those in a river.[3] The combination of the electromotive force, altered ionic pattern, and currents may potentially cause blood vessel dilation with a corresponding increase in blood flow.[4] However, magnetic forces are smaller than the physical forces already present caused by the heart muscle.[3]

The iron in hemoglobin causes blood to be only weakly repelled or attracted by magnets.[5] If they were attracted to a magnet this could have a clotting effect negatively affecting circulation.[3] Static magnets, to be effective, must overcome the strong physical forces of blood flow propelled by the heart and normal thermal induced Brownian random movement of the particles suspended in the blood, which is unlikely.[3]

Increased flow rates of concentrated saline have been observed within a glass capillary tube under the influence

of a magnetic field.[3] The reason for the increased flow is not explained but because the capillary cannot expand, it cannot be related to vasodilation.[3] However, the investigator of this study warns that these findings should not be extrapolated to tissue perfusion.[6] Many studies have shown static magnets to be ineffective in altering circulation both in animal and human studies regardless of the level of magnetic field.[3]

In a study performed on six horses, the effect of static magnetic therapy on blood flow was assessed in the metacarpal region.[7] Red blood cells were labeled with Tc 99m phosphate and dorsal images of both metacarpi were acquired with a gamma camera. One metacarpus was treated with a magnetic wrap with 270 G at its surface, and the other metacarpus was treated with an identical wrap in which the magnets had been replaced with identical size and weighted polytetrafluoroethylene sheet. The metacarpi were treated for 48 hours and the scintigraphy was repeated. No significant difference was noted in perfusion of the metacarpal region between groups. The authors noted that in the commercially available wraps used in this study, the strength of the magnetic field dropped off to 0.5 G at 7 mm from the surface of the magnet, making it unlikely that the underlying blood vessels were actually exposed to a clinically relevant magnetic field. Static magnetic fields have also been shown to have no significant effect on mucosal blood flow in humans,[8] soft tissue healing in rats,[9] or blood flow to the skin of rats.[10]

Pain Alleviation

A variety of theories exist attempting to explain how magnets may provide pain relief. Likewise, a wide variety of protocols with varying magnetic strengths is observed in the literature. One theory to explain pain relief is associated with increased or altered blood flow. As discussed previously, little to no evidence supports the contention that magnets have a measureable effect on blood flow. A second theory to explain pain relief is reduced nerve conductivity. This theory proposes that nociceptive C fibers have a lower threshold potential and that magnetic fields selectively attenuate neuronal depolarization by shifting the membrane resting potential.[11] However, this is an unlikely theory as a 10% reduction in nerve conductivity would require 24 T,[12] a field strength obtained only in the strongest superconducting magnets used for research.[3]

Clinical Effects

Perhaps one of the most successful studies conducted on the use of static magnets to treat pain was done by Vallbona and colleagues[13] on 50 patients with postpolio syndrome who reported muscular or arthritic pain. Based on a hypothesis that magnets applied over trigger points would have the greatest effect, they applied static magnets ranging in strength from 300-500 G to palpated trigger points for 45 minutes while patients remained in the experimental setting.[13] This study was double-blinded and appears to be one of the best-controlled studies using magnets because patients had little opportunity to test for magnetism. Pain levels were assessed by palpation of trigger points before and after application of the magnetic device. The McGill pain questionnaire was used to evaluate subjective pain, elicited by palpation of the single most painful trigger point, even if multiple sites were present. Following application of the magnets, trigger points were again palpated and pain was reassessed by the subjects. Twenty-two patients in the active group showed improvement as compared with four in the placebo group, a significant difference.

In 2007, Pittler and colleagues[14] published a systematic review and metaanalysis of 29 randomized trials in humans on the effect of static magnets on pain and concluded that the evidence does not support the use of magnets for pain relief for low back pain, delayed-onset muscle soreness, or foot pain.[14] The exceptions to this finding were three double-blinded randomized controlled trials assessing pain relief in osteoarthritis (OA) that found pain reductions relative to placebo when treatments lasted 2 to 12 weeks.[14] A separate study evaluated the use of magnets continuously for 24 hours and found that there was no pain relief. The authors concluded that for the condition of OA, there is insufficient evidence to exclude a clinically beneficial effect and believed that further investigation might be warranted.[14]

In a large randomized double-blind study assessing the effectiveness of magnet therapy on pain intensity and opioid requirements in human patients with acute postoperative pain, investigators found no difference in pain intensity level or opioid requirement between groups treated with magnets or sham magnets.[15] Patients with various abdominal surgeries or lipoma excision were treated with quadrapolar static magnets of 1900 G strength placed at the ends of the incisions and around them and left in place for 2 hours. The control group was treated with sham magnets of equal weight and size. Patients rated their pain via a numerical rating scale and the number of rescue doses of morphine was recorded. The authors concluded that magnetic therapy should not be used for treatment of postoperative acute pain or any pain that results from tissue injury.[15]

A comparison of commercially available static bipolar magnetic shoe insoles with identical sham insoles in 101 human subjects diagnosed with plantar fasciitis (heel pain) showed no significant difference in outcome variables studied.[16] Insoles were worn for at least 4 hours a day, 4 days a week for 8 weeks. The sham group reported significant improvement in morning foot pain intensity, and both groups reported an improvement in how much foot pain interfered with their employment enjoyment; however, magnets did not induce additional benefit over

nonmagnetized insoles. The sham insoles used in this study averaged a surface reading of 2.2 G and active magnetic insoles averaged 192.1 G.[16]

In a multicenter, double-blinded, placebo-controlled study investigating the role of static magnetic insoles on diabetic peripheral neuropathy–caused neuropathic foot pain, investigators found that chronic use of 450 G multipolar, static magnetic shoe insoles significantly reduced symptoms of burning, numbness, tingling, and exercise-induced foot pain compared with sham insoles.[17] Subjects wore magnetic insoles or sham insoles for 24 hours per day for 4 months and tabulated validated daily pain scores and unvalidated quality of life scores. No new analgesic drugs were allowed during the study. Significant differences between groups were not apparent until after the second month of treatment. Of note was that the magnitude of the reduction of burning, numbness, tingling, and exercise-induced foot pain, especially in the severe and extreme cases, was comparable or better than those previously reported for gabapentin, tramadol, and lamotrigine studies.[17] The mechanism of action of the magnets is unknown, but this study suggests that long-term use may be required to see improvement in symptoms.

The effectiveness of commercially available magnetic bracelets for pain control in OA of the hip and knee in people was evaluated in a multicenter, randomized, placebo-controlled trial.[18] Three groups were evaluated over a 12-week period: (1) standard strength static bipolar magnetic bracelet, (2) a weak magnetic bracelet, or (3) a nonmagnetic (dummy) bracelet. Mean pain scores using the standardized validated Western Ontario and McMasters Universities score were reduced more in the standard magnet group than in the dummy group (27% change from baseline). However, some patients discovered that the bracelets had a magnetic field, resulting in unblinding, although the analysis indicated that this had a small effect on the results. The authors concluded that pain from OA of the hip and knee decreases when wearing magnetic bracelets, but it remains uncertain whether the effect is due to specific or nonspecific (placebo) effects.

Studies on magnets for therapeutic purposes in animals are limited. Two studies by workers from Hungary demonstrated a reduction of writhing from visceral pain in mice induced by intraperitoneal injection of acetic acid. Mice were treated by placing them in a cage with magnets with nonhomogeneous 2-754 mT strength. The pain reaction was significantly reduced in the treated mice compared with mice injected and placed in cages without the magnetic field.[19,20] The authors hypothesized that the observed effect was somehow mediated by the peripheral opioid system because naloxone antagonized the static magnetic field–induced analgesic response if given peripherally but not centrally.[20]

One study determined the effect of a permanent magnetic field on the progression of OA in a canine model.[21] OA was created by transecting a cranial cruciate ligament. Dogs were subjected to no floor mattress, a floor mattress with ceramic pieces placed between two layers of foam but no magnetic field (sham control), or a floor mattress with ceramic permanent magnets placed between two layers of foam. The magnetic field had a surface field strength of 1100 G, whereas the magnetic field strength at the surface of the mattress was 450-500 G and was equally distributed over the surface. The magnetic mattress group appeared to have less synovitis, less synovial effusion, less disruption of the cartilage surface, and less cartilage ulceration than did the other groups. Histologic scores for OA were less in the magnetic mattress group as compared with the other groups, but the differences were not statistically significant. There were similar trends in immunohistochemical studies for matrix metalloproteinase (MMP)-1 and MMP-3, with the magnetic mattress group having less staining; this group had similar MMP-3 to normal cartilage. The mattress and magnetic field group did not differ from the normal group in MMP-3 as determined by Western blot analysis. The results of this study suggest that cartilage exposed to a magnetic field may have reduced progression of OA early in the disease process. Further studies are needed to confirm this and to delineate the mechanisms of action that magnetic fields may have in reducing the progression of OA.

Use

Although the evidence supporting use of magnet therapy is limited at best, and at worst not generally supported, they continue to be used by owners on their pets. Fortunately there are no known side effects of their use; however, it is recommended that they not be used in patients with pacemakers or any other device that may be adversely affected by the magnetic field. If used, magnets should be placed directly over the affected target area to avoid the rapid decrease in magnetic field over short distances. In addition, any positive effects seem to be associated with the use of strong magnets (50-120 mT). Treatment time should be over prolonged periods if tolerated and magnets should not be considered a sole treatment modality but rather an adjunct to more conventional, proven therapy.

Pulsed Electromagnetic Fields

PEMFs have been used for a variety of purposes, but their main therapeutic purpose is for enhancement of bone or tissue healing and pain control.[22] Piezoelectric potentials are created when bone is stressed as the result of electrolyte-rich fluid moving through bone channels. These electrical currents may act as a stimulus for new bone growth. A similar mechanism occurs with cartilage compression leading to current transduction and subsequent cartilage matrix synthesis. Streaming potentials are created within cartilage when electrolyte-rich fluid flows past areas of

compression and fixed negatively charged sulfated proteoglycans.[23] The maximal production of glycosaminoglycan occurs at a low frequency (less than 15 Hz), and intermittent compression is better than continuous compression. At 15-16 Hz, calcium ion transport occurs and cartilage growth is stimulated, perhaps because of increased transcription and DNA synthesis. The effect occurs quickly, with peak transcription in 20 minutes.

Electromagnetism is generated by running an electric current through a coiled wire (electromagnetic induction).[3] PEMFs are hypothesized to have electric rather than magnetic effects on tissue.[3] PEMF stimulation may act on cell surface receptors, such as parathyroid hormone and transforming growth factor (TGF)-β, and secondary messengers, such as calcium or cyclic adenosine monophosphate. PEMF may have an effect on stabilizing intracellular calcium stores by altering the conformation of calcium channel proteins that may ultimately reduce the production of free radicals by mitochondria and subsequent inflammation.[24]

Wound Healing

Healing may be promoted by using PEMF in chronic wounds,[25] in soft tissue, and in neuronal regeneration.[26] The effects of PEMF on healing of open and sutured wounds was evaluated in dogs.[27] Open and sutured wounds were created in the skin of the trunk of the dogs. The PEMF-treated dogs received treatment twice a day starting the day before surgery and lasting through day 21 after surgery. PEMF treatment resulted in significantly enhanced epithelialization of open wounds 10 and 15 days after surgery. PEMF treatment also shortened wound contraction time. Conversely, PEMF have not been found to have any significant effects in other studies of soft-tissue wound healing in rats[9] or on blood flow in the brains of mice.[28]

Bone Healing

Mechanical and electrical signals may regulate synthesis of extracellular matrix.[29] Endogenous electric current densities produced by mechanical loading of bone under physiologic conditions approximate 1 Hz and 0.1 to 1 uA/cm^2.[6] PEMF therapy stimulates biologic processes pertinent to osteogenesis[30] and bone graft incorporation.[31] In fact, several devices are approved for the treatment of delayed and nonunion fractures. PEMF may also attenuate bone loss during disuse.[32] PEMF therapy study results suggest that therapy may delay healing of fresh fractures, however.[33] One recent metaanalysis suggested that the proportion of delayed and nonunion fractures that united with PEMF was small and not statistically significant, and there was no reduction in pain.[34]

The effect of PEMF on the healing of experimental lumbar spinal fusions was evaluated in dogs. Bilateral posterior facet fusions were performed at L1-2 and L4-5 in adult dogs.[35] After surgery, one group was stimulated with a pulse burst–type signal PEMF for 30 minutes a day, another group was stimulated with the same PEMF for 60 minutes a day, and a third group received no active PEMF stimulation. No difference in the radiographic or histologic appearance of the fusion mass could be detected between the stimulated and control groups, suggesting that PEMF stimulation had no effect on the healing of posterior spinal fusions in this canine model. However, the use of PEMF in the late phase of bone healing in a stable tibial defect model did stimulate bone healing in one study in which dogs were stimulated 1 hour per day for 8 weeks.[36] Load bearing in the PEMF group at 8 weeks was greater than in the control group. In addition, callus area increased earlier in the PEMF group, and maximum torque, torsional stiffness, new bone formation in the osteotomy gap, and mineral apposition rate were greater in the PEMF group.

Osteoarthritis

One of the most common uses for PEMF is the treatment of OA. PEMF treatment has a number of physiologic effects on cells and tissues, including the upregulation of gene expression of members of the TGF-β family, an increase in glycosaminoglycan levels, and perhaps an anti-inflammatory action.[37]

Pipitone and Scott[38] looked at the effects of magnetic pulse treatment for knee OA in human patients. They used a unipolar magnetic device that was used to treat both knees simultaneously for 10 minutes three times a day for 6 weeks. The devices were set at 7.8 Hz for the morning and afternoon treatment sessions and at 3 Hz for the evening session. Outcome measures included evaluation by a rheumatologist, and several subjective pain assessment scales, two of which are disease-specific measures validated for use in patients with knee or hip OA. There were no significant differences between the active- and sham-treated groups in any outcome measures after treatment.[38]

Another review article of PEMF therapy for people with knee OA concluded that PEMF has little benefit in the treatment of OA in people, and that resources might be better directed to other rehabilitation efforts, such as exercise.[39]

Another randomized, double-blind, placebo-controlled clinical trial investigated the effectiveness of PEMF for the treatment of OA of the knee in people.[40] Patients were treated 2 hours daily, 5 days per week for 6 weeks. Comparison of all treated patients with an untreated control group revealed no significant improvements over time. Analysis of patients younger than 65 years old, however, revealed a significant improvement for stiffness on the treated knee. Analysis of activities of daily living score for the PEMF-treated group revealed a significant correlation between less improvement and increasing age.

A Cochrane review evaluated PEMF for OA treatment.[41] Two trials used PEMF, using a noncontact device that delivered three signals in stepwise fashion, ranging from a 5 Hz to 12 Hz frequency at 10 G to 25 G of magnetic energy. The affected knee was treated for 9 hours over a 1-month period. There was a small to moderate effect on outcomes for knee OA, with clinical benefit ranging from 13-23% greater with active treatment than with placebo. The main outcome measures were for pain, ability to conduct activities of daily living in terms of pain or difficulty, joint pain on motion, and tenderness. Although these results suggest that PEMF result in improvement for knee OA, they suggested that further studies should be performed to confirm these results and to establish the clinical relevancy of treatment.

In another Cochrane review of electrotherapies used for mechanical neck disorders, 5 of 11 studies included used magnets as an intervention.[22] Four of the five used pulsed magnets, whereas the fifth used static magnets. Two of the five had significant results and both of these studies used pulsed magnets, one using low-frequency 5-25 Hz 10-25 G, and one using high-frequency 27 Hz.[22] Interestingly, two of the five studies were led by the same author, using a similar intervention with reported significance in the first study followed by no significant difference 2 years later in the second study. Furthermore, another group used a similar intervention and had no significant results. Finally, the subjects of the two studies with positive results retained these benefits for only a short duration.[22]

Another Cochrane review assessed the effectiveness of PEMF compared with placebo for the treatment of knee OA in randomized, controlled trials.[42] Nine studies, including 483 patients, were pooled. No significant difference could be shown for pain or stiffness. There was a significant effect on activities of daily living and scores. The authors concluded that PEMF improve clinical scores and function as an adjuvant (not stand-alone) treatment in patients with OA of the knee, but there is no evidence for an effect on pain. Another study indicated that PEMF does not add additional benefit to patients already receiving treatment for knee OA, including hot packs, therapeutic ultrasound (US), and exercise.[43] Another study also found no added benefit to PEMF therapy for knee OA when patients received hot packs and transcutaneous electrical nerve stimulation and exercise.[44]

A randomized, controlled, blinded, clinical trial consisting of 60 dogs with OA indicated some positive effects of pulsed signal therapy (PST).[45] Dogs in the treatment group were given nine treatments over 11 days. There were 24 dogs in the treatment group and 35 dogs in the control group. Peak vertical force was significantly more improved in the treatment group from day 0 to day 11, but not from day 0 to day 42. The improvement in the canine brief pain inventory (CBPI) score and measurement of joint extension was significantly better in the treatment group for both time points, and the function portion of the CBPI and thigh circumference was significantly improved in the treatment group from day 0 to day 42. This study suggested that PST was useful in treating canine patients with OA.

Pain

Some investigators have evaluated the use of PEMF for pain management after surgery. A randomized, prospective, double-blind study was performed to evaluate the long-term effects of PEMF in patients undergoing arthroscopic treatment of knee cartilage.[46] Patients with knee pain were recruited and treated by arthroscopy with chondroabrasion and/or perforations and/or radiofrequencies. They were randomized into two groups: a control group (magnetic field at 0.05 mT) and an active group (magnetic field of 1.5 mT). Patients were instructed to use PEMF therapy for 90 days, 6 hours per day. The patients were evaluated with a knee injury and osteoarthritis outcome score before arthroscopy, and 45 and 90 days after treatment. Patients were also interviewed for long-term outcome 3 years after surgery. Differences were significant at 90 days. The percentage of patients who used nonsteroidal antiinflammatory drugs was 26% in the active group and 75% in the control group. At 3 years follow-up, the number of patients who completely recovered was higher in the active group compared with the control group.

A randomized controlled clinical trial evaluated PEMF therapy on postoperative pain in dogs following ovariohysterectomy, and the interaction with postoperative morphine analgesia.[47] Following extubation, dogs were divided into four groups: a control group that received no PEMF, a PEMF group, a morphine group with no PEMF, and a PEMF and morphine group. Although no clear benefit was seen in this study, the results suggested that PEMF may augment morphine analgesia following ovariohysterectomy in dogs.

Use

PEMF therapy appears to have benefits for the treatment of certain conditions. Many of the biophysical effects on cells and tissues have been confirmed. However, its use is still limited likely because of confusing and often conflicting results. As with many modalities used in human and veterinary medicine, dose, frequency, and characteristics of the modality used in studies are bewildering, making the meaningful interpretation of studies difficult. In addition, dogs must remain still when in a PEMF unit. This can provide a challenge for many dogs. Although the modality may show promise for certain conditions, its common use in veterinary medicine is hampered by the lack of prospective, blinded, placebo-controlled studies; the expense of the equipment; and the need to maintain dogs in a relatively quiet state during treatment.

Pulsed Shortwave Diathermy

Pulsed shortwave diathermy (PSWD) operates at approximately 27.12 MHz and creates an electrical field, which heats the tissues in its field. It is also called *pulsed shortwave therapy* and *pulsed electromagnetic energy.* It has some advantages over therapeutic ultrasound (US), which include:

- Larger treatment area than US
- Less reflection off bone
- Use over irregular areas such as the paw, where US use is difficult

The disadvantages include a few additional contraindications not seen in US, including metal implants in the field because metal heats up in response to PSWD.

Contraindications and Precautions

- Over the eyes
- Over or near malignancies and tumors
- Over a pregnant uterus
- Over epiphyses of growing bone
- Over the spinal cord after laminectomy
- Vascular thrombus and embolus
- Over the heart
- Electromedical devices (e.g., pacemakers)
- Plastic and metal implants
- Metal in the treatment field and immediate area including collars, or performed on a metal treatment table
- Decreased or absent sensation
- Over acute inflammation
- Over areas of active infection

Another disadvantage is that dogs must remain still during diathermy, which can provide a challenge for many dogs. Most PSWD units involve placing a drum with a built in spacer over a body part and having the patient stay in one place. Newer technology that attaches a sleeve over a body part may have implications for veterinary rehabilitation. Although diathermy has evidence for tissue heating and increases in muscle flexibility in humans, its use in veterinary medicine is hampered by the lack of prospective, blinded, placebo-controlled studies; the expense of the equipment; and the need to maintain dogs in a relatively quiet state during treatment.

In humans, PSWD has been shown to be as effective as US in heating muscle and more effective than US in heating large areas of muscle.[48,49] A well-controlled study compared PSWD and stretch to sham PSWD and stretch in hamstring flexibility. After 5 days of treatment the diathermy/stretch group had an increase in knee extension of 15.8 degrees ± 2.2 degrees, compared with the sham diathermy/stretch, which had an increase in knee extension of 5.2 degrees ± 2.2 degrees. Diathermy with stretch significantly improved hamstring flexibility in comparison with stretching only in this study.[50]

Diathermy has also been investigated for tissue healing. An in vitro study found PSWD given at mean power of 48 W for 10 minutes increased fibroblast proliferation compared with control groups ($P < 0.001$), and chondrocyte proliferation also increased with PSWD exposure of 6 W at 10 minutes' duration ($P = 0.015$).[51]

Summary

Static magnets, PEMF, and diathermy are some of the modalities that are emerging in veterinary rehabilitation. Unfortunately, despite some evidence that there are biologic effects in tissue culture or laboratory experiments, there are few published well-designed clinical studies in veterinary medicine to make firm recommendations for their use. In particular, questions regarding dosage, the length of treatment, and the most effective treatment protocols for specific conditions are lacking. Users should demand proper studies of products before purchasing them, and veterinary funding agencies should provide adequate resources for investigators to test such devices independently.

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SECTION V

Therapeutic Exercise and Manual Therapy

24 Biomechanics of Physical Rehabilitation and Kinematics of Exercise

Joseph P. Weigel and Darryl Millis

Mechanics as developed by Sir Isaac Newton describes how force and mass interact in three-dimensional space and time on the surface of the earth. Biomechanics is the application of Newtonian mechanics to biologic systems. Newtonian mechanics is founded on three classic laws of motion. The first law describes the static state of a body under the influence of balanced force. The second law describes the motion or dynamic state of a body under the influence of unbalanced force. The third law describes the interaction between multiple bodies.

Law I (Law of Statics)

When the sum of all the forces acting on a body equals zero, the body is in a static state (i.e., the body is at rest or in unchanging, constant motion with no acceleration).

$$\Sigma F = 0 F = 0$$

Under certain conditions, the static state is desirable. For example, to enhance the conditions for healing, fractured bones are managed by fixation systems designed to place fracture segments in a static state or at rest. It is therefore the goal of the surgeon to balance, both in direction and magnitude, all the forces that tend to cause motion of the fragments. In the case of a fractured tuber calcis the proximal fragment is under a large tension load caused by the pull of the attached gastrocnemius muscle. Without fixation this pull is unopposed, causing motion of the bone fragments, which disrupts the body's attempt to bridge and stabilize the fracture. When a system of pins and a tension band wire are placed across the fracture, the wire, by virtue of its position and strength, counters the tension load of the gastrocnemius muscle with a corresponding compression load provided by the tension band wire. When the sum of the forces acting across the fracture equals zero, the fracture is in static equilibrium, a condition that fosters bone bridging. It is important to exercise caution when applying physical rehabilitation to animals with healing fractures such that these activities do not result in creating an imbalance of force, causing motion of the fracture. The static state of the fracture must be maintained even though the therapist is focused on motion of joints and the strength of muscles.

Static exercise can be an effective way of stretching and enhancing flexibility by the strategic and controlled application of force without producing motion. Static stretching may improve soft tissue extensibility, which increases the capacity for energy absorption and transfer when running, for example. Practical methods for preexercise preparation in animals have not been established. Dogs are often left sedentary for long periods and then are suddenly released for unrestrained activity, often at high performance levels. After long periods of relative inactivity, ligaments, muscle, tendons, and fascia are "tight," less ductile and less able to tolerate acute strain. Stiff soft tissue has less capacity to absorb impact, lending itself to tearing or rupture. A common thread in the clinical history of cranial cruciate injury in the dog is the occurrence of sudden lameness during vigorous activity following long periods of inactivity.

Law II (Law of Dynamics)

When the sum of all the forces (resultant force) acting on a body is not equal to zero, the body is in a dynamic state (i.e., the body is not at rest and is in a state of changing motion or accelerating).

$$\vec{F} = m\vec{a}$$

Force (F), is a vector and therefore has not only intensity (magnitude) but also direction. Acceleration (a), the change in velocity over time, also has intensity as well as

direction. Newton's second law directly relates the cause of motion to the motion itself. In terms of magnitude, the harder a body is pulled or pushed by an increasing force, the faster it moves as seen with increasing acceleration and velocity. In terms of direction, the body will move or accelerate in the same direction as the applied force that has caused the motion. Therefore motion analysis or dynamics involves the application of arithmetic functions but within three-dimensional space, which also necessitates the use of trigonometric functions. Newton's second law of motion can be expanded to describe the associated concepts of motion such as impulse, momentum, energy, work, and power.

The physical therapist applies Newton's second law of motion when the speed or acceleration (change in velocity over time) of a therapeutic exercise is increased. When speed or acceleration increase, the load (force) on the limb must also be increased to achieve the faster motion. For example, hitting a wall with an automobile at 5 miles an hour might dimple a bumper, but hitting the same wall with the same automobile at 100 miles an hour could result in irreparable damage. Going faster means greater force, which then increases the risk for exacerbating an injury or inducing new injury. For example, repeated rapid, forceful flexion of the stifle in the case of a contracted quadriceps muscle can result in avulsion of the tibial tuberosity. Slow range of motion (ROM) exercises, slow stretching, and slow walking keep the applied forces low, preserving and protecting healing tissues.

Law III (Law of Action and Reaction)

When several bodies interact, the force resulting from the interaction is equal in intensity to the force causing the interaction and will act along the same line but opposite in direction.

Newton's third law describes the event when one body impacts another. Newton observed that when one mass, moving under the influence of a causative force, impacts a second mass, a reaction force occurs. This reaction force is equal to the magnitude of the impacting force (original causative force) but acts in the opposite direction. This is easily observed in the case of a bouncing ball. When a ball is dropped to the floor, it impacts the floor and then reverses direction, moving away from the floor. The initial throwing force is reversed after the impact into a reaction or "bouncing" force. If the bouncing ball is replaced by a bag of sand, the bag will not bounce; however, reaction forces are still created but are absorbed by the sand in the bag. When an animal moves across the floor there is both an elastic response (like the bouncing ball) where force is reflected and an inelastic response (like the bag of sand) where force is absorbed.

Newton's third law is the basis for the use of a force plate in analyzing an animal's gait. When in motion, the animal impacts the plate, which results in a reaction force that is recognized by the plate and analyzed by a computer program. The reaction force, recorded at the point of foot impact (foot strike), is directly related to the force of weight bearing. This reaction force, called the *ground reaction force* (GRF), is transmitted up the limb in part as an elastic reaction where it is reflected by the stiffer tissues, and in a large part as an inelastic response where it is absorbed by the softer tissues. This absorption of force is an important function of the limb and all its components, and as a result, is an important consideration in physical rehabilitation. The therapist can manipulate the reaction force by applying various forms of exercise. For example, the aquatic treadmill mitigates the reaction force by using the buoyancy of the animal in water to reduce its weight. Slinging the abdomen during ambulation also reduces the impact of weight, thereby decreasing the reaction force. Sometimes a reaction force is magnified so it can be a means of "active therapy" during which it can stretch contracted tissues. Exercising down an inclined plane magnifies the reaction force by increasing the magnitude of impact on the front limbs. Also, a reaction force can be magnified by applying a syringe cap to the foot of the sound limb to encourage a shift of weight bearing to the contralateral limb, accelerating its rehabilitation. Conditions of the terrain, such as the stiffness of the floor, obstructions in the gait pathway, or unstable flooring can alter the magnitude and direction of reaction forces. When devising therapeutic plans, the therapist should consider the nature of the reaction forces and how they can be modified for the patient's benefit.

In the case of animal physical rehabilitation, the application of the Newtonian laws of motion can be an ominous challenge. The animal is a complex and sophisticated system of interconnected objects having complex patterns of motion. This chapter attempts to incorporate the fundamental concepts of biomechanics to the practice of animal rehabilitation.

The Body in Equilibrium: Statics

Force is intuitively understood as a measure of intensity but also it must be viewed with respect to direction. For example pulling on a car door causes it to open while pushing on it causes it to close. To know only how hard to pull or push is insufficient in understanding the design, operation, and composition of a car door. Similarly, in biomechanics it is not only beneficial to know how hard the leg impacts during an exercise but also from which direction the force is acting. For example, living bone responds to weight bearing by increasing its mass and this response is not only caused by the intensity of the weight but also by where on the bone the weight load is localized. The classic Wolff's Law can be described as the case where bone increases in mass in response to force that tends to push it together, whereas it decreases in mass where the

force tends to pull it apart. In the case of corrective osteotomies, the goal is to help improve limb alignment, distribution of weight bearing forces, and joint function by altering the anatomy and redirecting weight bearing, creating an improved mechanical environment.

When the animal is at rest (standing still), the forces acting on the body have not disappeared but have been counterbalanced by forces acting in opposite directions. Mechanical analysis of the body in this static state facilitates understanding of the types of forces at play. There are four basic forces: compression, tension, shear, and moment or torque. The kind of force depends on its direction. A force that tends to push on a body is referred to as *compression*; if it tends to pull on the body, it is called *tension*; if it tears or shreds the body, it is *shear*; and finally, if it tends to rotate or turn the body, it is a *moment* or *torque*.

To evaluate direction, a reference or coordinate structure is necessary. Commonly a three-axis rectangular system representing the three dimensions of space (height, width, and length) is used. By convention this axis system is composed of three perpendicular axes, labeled z for the vertical direction, y for the horizontal direction, and x for the transverse direction.

Compression (Push)

When the vertical dimension of the limb is oriented along the z axis, a force parallel to the z axis and directed toward the center of the limb is called *compression.* Compression is the major load on the hard tissue elements, bone and cartilage, as it tends to push them together.

Tension (Pull)

With the vertical dimension of the limb oriented along the z axis, a force parallel to the z axis and directed away from the center of the limb is called *tension.* Tension is the major load on the soft tissue elements, muscles, tendons, and ligaments, as it tends to pull them apart.

Shear (Tear or Shred)

With the vertical dimension of the limb oriented along the z axis, a force perpendicular to the z axis is called *shear.* Shear is a significant force causing the elements of the system to tear or slide apart. For example, the cranial cruciate ligament (CCL) encounters a shear load that occurs across the stifle during weight bearing. When this ligament is torn, the shear is unopposed and cranial drawer occurs. Redirection of the weight-bearing forces by a tibial plateau leveling osteotomy eliminates the shear component, thereby preventing the occurrence of cranial drawer while in weight bearing.

Rotation (Moment of a Force or Torque)

A force can also tend to cause a mass to rotate about a point called the *center of rotation.* The farther away from the center of rotation the force is applied, the greater is the tendency for rotation. This tendency is called "moment of a force," and the distance from the center of rotation to the point of force application is called the *moment arm.* Mathematically, moment is represented by multiplying the force by the moment arm:

$$\vec{M} = \vec{F}\vec{d}$$

where M is the moment of the force, F is the force, and d is the moment arm.

Moment is critical to the understanding of how force acts in the musculoskeletal system. Although forces of body weight and muscle contraction push and pull at the body, they also cause rotation at each of the joints when these forces do not pass through the center of rotation of the joint. For example, there is a tendency toward flexion (rotation in one direction) of the joints in the front limb of a standing animal, which is caused by the body's weight pushing down at a location that is off center of the respective joint. The animal is able to stand because the tendency toward flexion is counterbalanced by a tendency toward extension (counter rotation in the opposite direction) caused by contraction of several muscles, including the supraspinatus, triceps brachii, and extensor carpi radialis muscles. In the rear limb, flexion caused by the body's weight at the hip, stifle, and tarsal joints is counterbalanced by contraction of the gluteal, quadriceps, and gastrocnemius muscles, which causes extension at each of these joints. The actions of these forces of weight and muscle contraction are defined as *moments,* which are very important, especially in the analysis of gait.

Moments of force are important in the effective function of muscle in sustaining weight bearing. For example, in the stifle the normal position of the patella creates an effective moment by the quadriceps about the center of rotation of the stifle such that extension of the stifle is maintained enough to bear weight. However, the function is altered when the patella is not in its normal position and is dislocated medially. In the dislocated position the moment arm of the tension force in the quadriceps is reduced or eliminated (Figure 24-1). The closer the tension force is to the center of rotation, the less extension is possible. Therefore a dog with luxating patellas is weak in the rear limbs and unable to bear weight normally or perform activities, such as jumping.

Lever Systems in the Body (Application of Moments of Force)

A lever is a simple machine using the principle of moments to overcome a resistance load. There are three classes of levers: Class I, in which the fulcrum (point of rotation) is between the effort force (applied force usually resulting from muscle contraction) and the resistance force (load force, which is generally the weight of the body or limb); Class II, in which the resistance is between the effort and

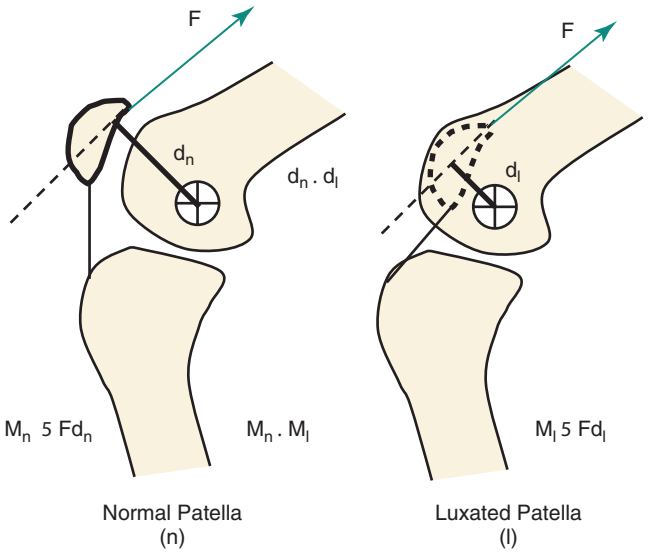


Figure 24-1 The moment arm of the tension force in the quadriceps is reduced or eliminated when the patella is luxated medially. Luxation of the patella compromises weight bearing by lessening the moment (M_l) generated by quadriceps contraction by shortening the moment arm (d_l).

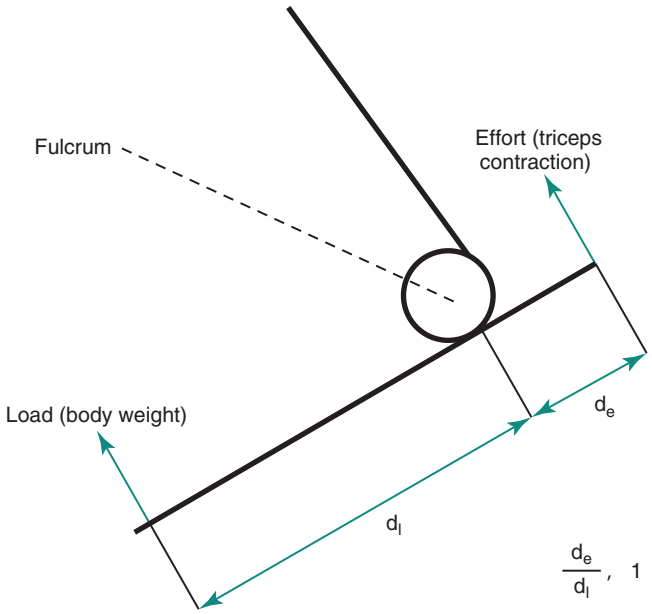


Figure 24-2 Extension of the elbow is a Class I lever system. The fulcrum is located at the center of rotation of the joint, the resistance or load force is located at the point where the foot contacts the ground and the effort or action force produced in the triceps muscle is located at its insertion point on the olecranon.

fulcrum; and Class III, in which the effort is between the resistance and the fulcrum.

A variety of levers are used by the animal to extend and flex the various joints of the limbs. For example, extension of the elbow is accomplished with a Class I lever in which the fulcrum is located at the center of rotation of the joint, the resistance or load force is located at the point where the foot contacts the ground, and the effort or action force, which is produced in the triceps muscle, is located at its insertion point on the olecranon (Figure 24-2). With flexion of the elbow, a Class III lever is in operation in which the effort is located at the insertion of the biceps brachii, which is between the fulcrum (elbow joint) and the resistance force, which is located at the end of the antebrachium (Figure 24-3).

Class II levers are uncommon. An example is the rear foot, in which the weightbearing toes act as the fulcrum, the resistance or load force is the body weight just caudal to and at the foot, and the effort force is the gastrocnemius muscle (Figure 24-4).

In contrast, both extension and flexion of the stifle joint are accomplished with a Class III lever, in which the effort or action load is located at the insertion of the quadriceps muscle on the tibial tuberosity (for extension) or the insertion of the semitendinosus muscle on the proximal tibia (for flexion), which is located between the fulcrum (stifle joint) and the resistance force, which is located at the distal end of the limb.

The relative magnitudes of the effort versus the resistance is the value of understanding lever systems and their

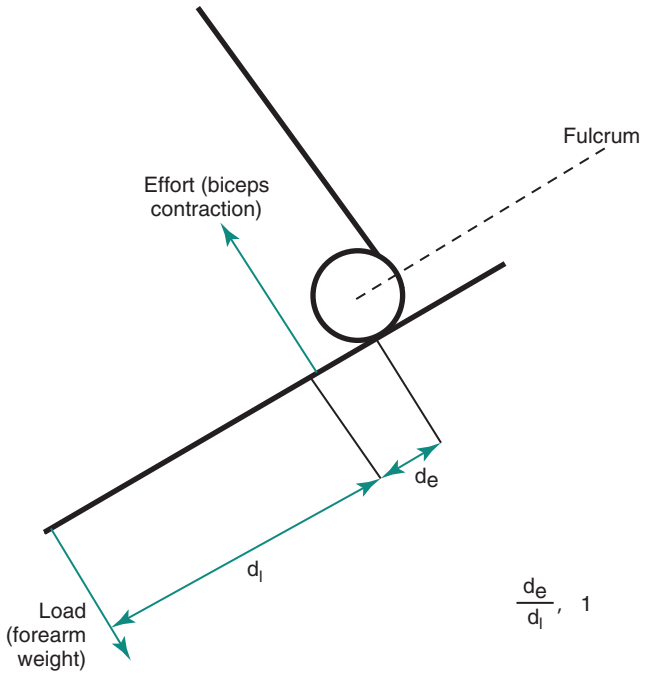


Figure 24-3 A Class III lever occurs with flexion of the elbow where the effort is located at the insertion of the biceps brachii, which is between the fulcrum (elbow joint) and the resistance force, which is located at the end of the antebrachium.

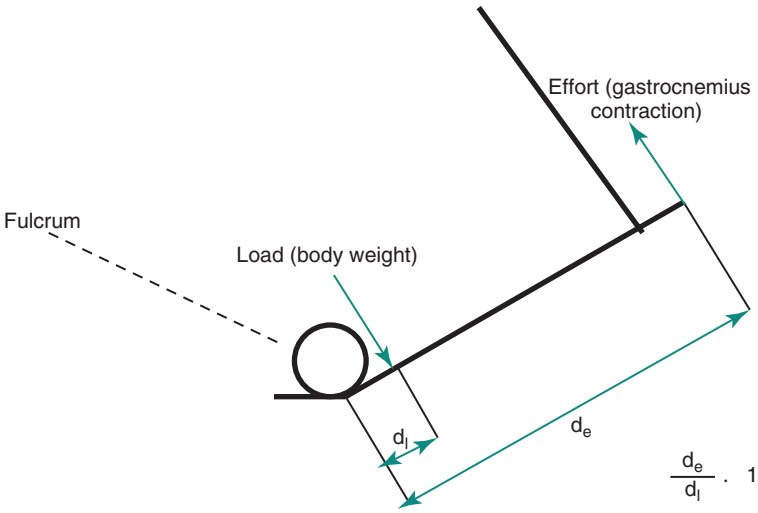


Figure 24-4 An example of a Class II lever is the rear foot, where the weight-bearing toes act as the fulcrum, the resistance or load force is the body weight just caudal to and at the foot, and the effort force is the gastrocnemius muscle.

function in the musculoskeletal system. The effort force multiplied by its distance (moment arm, d_e) to the fulcrum is the effort moment. The resistance force multiplied by its perpendicular distance (moment arm, d_o) to the fulcrum is the load moment. In the balanced state in which the lever is not moving or moving without acceleration, the effort moment must equal the load moment. This Law of the Lever system establishes the relationship between the effort and resistance loads. When comparing the magnitude of required effort to overcome the magnitude of a specified resistance, compare the respective moment arms. If the effort moment arm is longer than the resistance moment arm, then the effort force necessary to overcome the resistance will be relatively less than the magnitude of the resistance.

$$(E)(d_e) = (R)(d_o)$$

In the case in which rotation is inhibited by contracture or fibrosis leading to decreased ROM, therapists use "passive" ROM exercise by pushing or pulling on the lower part of the limb to induce flexion or extension in a target joint. Passive ROM is often used to reduce the possibility of stiffness or to improve the mobility of stiff joints. In some of these exercises the lever action is altered. For example, stretching in flexion of a dog with hyperextension stiffness of the stifle presents the conditions for a Class II lever (Figure 24-5). The point of resistance is the insertion of the quadriceps muscle on the tibial tuberosity, which is located between the fulcrum (stifle joint), and the effort, which is the location of the therapist's hold on the distal crus of the leg. In this case, the effort is relatively far from the stifle when compared with the resistance that is close to the joint. When the quadriceps is adhered to the underlying bone and joint capsule and has lost its elasticity, it has also lost its capacity to absorb energy and load. As

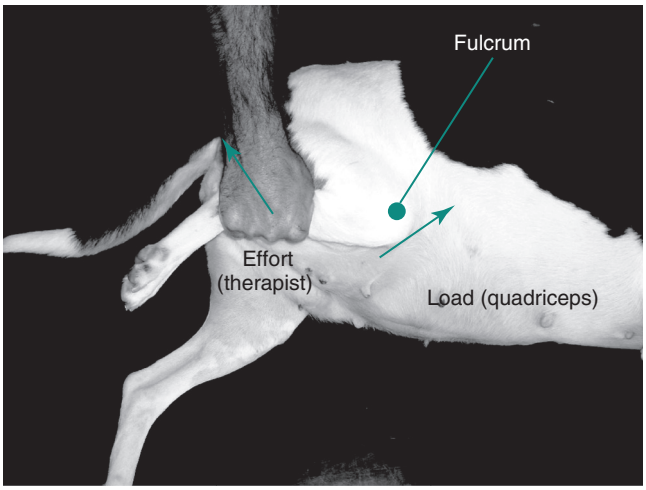


Figure 24-5 Stretching in flexion of a dog with hyperextension stiffness of the stifle presents the conditions for a Class II lever. The point of resistance is the insertion of the quadriceps muscle on the tibial tuberosity, which is located between the fulcrum (stifle joint), and the effort, which is the location of the therapist's hold on the distal shin or crus of the leg. In this case the effort is relatively far from the stifle when compared with the resistance, which is close to the joint.

the therapist pushes on the tibia to produce flexion in the stifle, the resistance load in the stiff quadriceps quickly rises, creating a magnitude of force that has the ability to result in tearing of the quadriceps or even avulsion of the tibial tuberosity. Because the moment arm length of the effort force is great, a large moment can be generated, potentially causing damage. It is important for the therapist to understand the relationship between the effort or action load and the reactive or resistance load when employing certain therapeutic exercises. In the previous example,



Figure 24-6 Wheelbarrowing to increase weight bearing on the front limbs results in the spine becoming a Class II lever in which the resistance (center of gravity of the body) is located between the fulcrum at the shoulder joint and the effort where the therapist is lifting the caudal end of the body. As the spine is raised from the rear, the center of gravity of the body moves to a location that is more dorsal to the shoulder.

moving the hands closer to the stifle joint reduces the moment arm length, and therefore makes it more difficult to generate a large moment, reducing the possibility of tissue damage.

Other exercises such as "wheel barrowing" on the front limbs or "dancing" on the rear limbs employ the use of lever systems. In the case of wheel barrowing or walking down an incline (to increase weight bearing on the front limbs), the spine becomes a Class II lever in which the resistance (center of gravity [COG] of the body) is located between the fulcrum at the shoulder joint and the effort in which the therapist is lifting the caudal end of the body. It is also important to note that as the spine is raised from the rear, the COG of the body moves to a location that is more dorsal to the shoulder, introducing a horizontal component of the weight, which acts as a shear force across the shoulder joint (Figure 24-6). As a result, increased stress in the joint capsule, collateral ligaments, and tendons will likely occur. This horizontal shear force may also cause an additional moment to occur at the level of the elbow joint, which is likely resisted by additional contraction of the triceps muscle. It has been observed that the angle of the shoulder joint becomes more extended with wheelbarrowing,[1] which moves weight bearing from the center of the humeral head to a more cranial location where there is less articular cartilage and bone. Dancing is the reverse of this example; in dancing the spine is raised from the front and used as a Class II lever. As with the effects on the shoulders, the same likely occurs on the hips. For example, the addition of shear force across the hip joint has the potential of increasing mechanical stresses in the articular cartilage, which may be deleterious to a dysplastic joint. Increased moment across the stifle would likely be resisted by increased contraction of the quadriceps muscles, strengthening this muscle group. The effectiveness of physical rehabilitation exercises could be enhanced by detailed mechanical evaluation looking at not only the magnitudes of weight-bearing forces but also the altered direction of these forces.

Tissue Response to Force

To understand how force affects the body, the response of the various tissues to force is important to analyze. Each of the tissues involved respond differently because the composition of each is different. These responses are unique to the tissue and can be called the *material properties of the tissue.* These properties include measures of stiffness, elasticity, plasticity, strength, failure limits, and energy absorption. Basically, a tissue responds to force in two ways: first, an intrinsic resistance to the applied force is called *stress*; second, an accommodation to the applied force by a change in the shape of the tissue is called *strain.*

Stress

Stress is directly related to the magnitude of the applied force (i.e., the greater the applied force, the greater the stress within the tissue). Stress is also inversely related to the area over which the total force is acting (i.e. the larger the area, the lower the stress). This understanding of the nature of stress explains how a scalpel cuts the skin. A sharp blade has an extremely thin edge so that with light pressure the force is concentrated at the edge of the blade and the stress in the skin is very high, causing the skin to break away and separate. A dull blade with a wider edge requires more force to increase the stress to the skin and cause it to separate.

Stress is particularly important in the understanding of how articular cartilage responds to force applied across a joint surface. Under conditions in which the anatomy is normal and the joint is physiologically and mechanically normal, the surface of the joint spreads the applied forces over a wide area such that the stress at any specific point on the cartilage is low. However, if the joint is anatomically deranged, such as with a growth deformity, or is mechanically unstable, such as with hip dysplasia, joint forces are concentrated on smaller surface areas, causing high degrees of stress and subsequent breakdown of the articular cartilage (Figure 24-7).

Motion of a joint is not only important for the physiologic health of the articular cartilage, but also for the mechanical integrity of the cartilage. For example,



Figure 24-7 In a mechanically unstable joint, such as in a dog with hip dysplasia, joint forces are concentrated on small surface areas, causing high degrees of stress and subsequent breakdown of the articular cartilage.

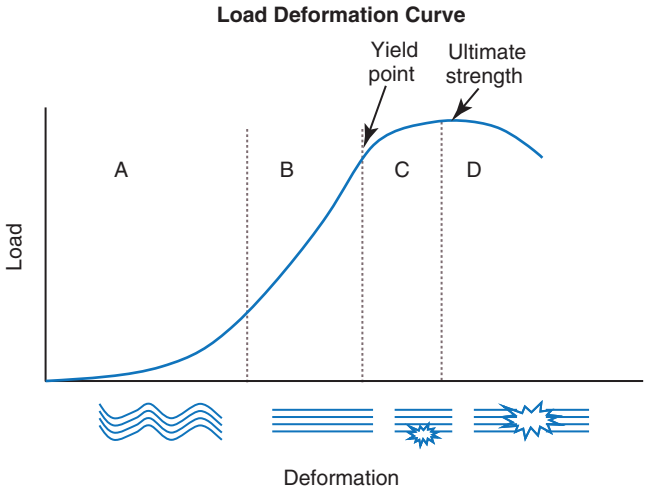


Figure 24-8 A stress versus strain curve demonstrates additional properties of the tissue response to force. **A,** The initial response in the graph is termed the *toe region* of the graph. The collagen fibers are in a completely relaxed state with a crimped or wavy pattern. **B,** The linear portion of the graph describes the elasticity of the tissue. The slope of the graph gives a measure of stiffness of the tissue. **C,** The upper curved portion of the graph describes the plasticity of the tissue. In this range the tissue is deforming, but when the load is removed, the tissue does not return to its original shape, meaning that a permanent change has taken place. **D,** The peak of the curve is the highest level of stress the tissue tested can withstand and is referred to as the ultimate strength of the tissue.

long-term immobilization of a normal joint can induce osteoarthritis. The lack of motion results in a concentration of force over a small area of articular cartilage. This increases the stress, causing a breakdown in the cartilage structure, inducing an inflammatory response and osteoarthritis. Distributing weight-bearing forces over a wider area of the joint surface is one way to minimize articular cartilage stress, but also reducing the weight-bearing force itself while maintaining joint motion should reduce the stress. This may be accomplished with an aquatic treadmill in which the water tempers the weight-bearing force but allows normal ROM.

Strain

As a result of an applied force, a tissue can change shape. For example, a ligament stretched by a tension force will lengthen, and this increase in length is termed *tensile strain.* Likewise, compressive force compacts tissue, creating a compressive strain, and shear force translates portions of tissue, creating a shear strain. For some tissues, strain and strain rates may depend on the direction or orientation of the force causing the strain. Such a property is referred to as *anisotropy.* For example, depending on which direction the force is acting on a section of bone, the strain value is different. Bone is stronger under compression than it is under tension, meaning that the strain is less (i.e., the bone deforms less under a compressive load than it does under the same intensity load in tension). The opposite is true in the case of a ligament: the ligament deforms less under tension than it does under compression.

Stress versus Strain

For any tissue under the influence of force, its response can be graphed as stress versus strain, which demonstrates additional properties of the tissue response to force (Figure 24-8). The initial response in the graph is termed the *toe region* of the graph. This phenomenon is more typical with soft tissue such as a tendon or ligament as opposed to bone. A tendon in its completely relaxed state has a crimped or wavy pattern of the collagen fibers. When the tendon is mounted in the testing machine and then pulled, the initial recording represents a straightening of the crimp and does not represent any part of the strength of the tendon in tension. It is also helpful to pass through this "toe" region by "prestressing" the tendon (i.e., stretching the tendon to eliminate the crimp). The slope of the graph gives a measure of stiffness of the tissue. Steep slopes occur when the tissue is inflexible and brittle like that of bone, whereas a shallow slope represents tissue with flexibility, like that of muscle or tendon. The linear portion of the graph describes the elasticity of the tissue. In this range the tissue is deforming or

undergoing strain, but when the load is removed, the tissue returns to its original length. The upper curved portion of the graph describes the plasticity of the tissue. In this range the tissue is deforming, but when the load is removed the tissue does not return to its original shape, meaning that a permanent change has taken place; therefore the tissue has undergone a "plastic deformation." The peak of the curve is the highest level of stress the tissue tested can withstand and is referred to as the *ultimate strength* of the tissue. Failure under stress is demonstrated as a decreasing capability to maintain the stress and the value begins to drop as the load increases, or complete failure as the stress drops to zero, indicating that the tissue can no longer hold up under any applied load. In addition to these features, the area under the stress versus strain curve represents the amount of energy that is absorbed or stored in the material as it is loaded. Once failure occurs, the stored energy is released in the form of a fracture or tearing event.

For bone, the response is more predictable and consistent as compared with muscle, tendon, and ligament. Bone behaves in a linear way as a elastic solid, but for the soft tissues where the response is more variable and less predictable, the response is nonlinear. As opposed to bone, these tissues behave as a viscoelastic material. A viscoelastic solid's properties, like stiffness, change over time. The response of a viscoelastic material additionally depends on factors like moisture content and temperature. For example, under the condition of cold temperatures, the tissue is stiffer so the stress is greater. When exercise is planned, often the tissues are physically warmed, which has the effect of reducing the stiffness and increasing its flexibility and elasticity. If the ligaments are cold and therefore stiff, a normal load could result in a higher than normal stress and in some cases the stress may be high enough to supersede the capacity of the ligament to remain intact, resulting in ligament damage.

Stress relaxation and creep are also additional phenomena that occur in viscoelastic materials. Stress relaxation is the condition when a tissue is deformed to a consistent state and the stress gradually diminishes over time. Although bone is more like an elastic solid, it exhibits some properties of viscoelasticity when compared with other solids, such as metal. The moisture in bone accounts for this response. In fracture repair, bolts and nuts are not used because of the viscoelasticity of bone. When a bolt and nut are used, the material in between is compressed. This material resists the compression by pushing back on the bolt and nut. This interaction of the applied force by the bolt and nut and the reactive force in the material stabilizes the bolt and nut. In a material that retains its stiffness the fixation remains stable; however, if the material is viscoelastic and its properties change, such as with bone, the stiffness decreases as water is squeezed from the bone. As the stiffness decreases, the push back decreases and the bolt and nut become loose and the fixation is lost. This phenomenon is referred to as *stress relaxation* in which the strain is constant but the stress decreases over time.

The converse event, in which the stress is constant and the strain changes over time, is referred to as *creep*. Creep is used frequently in passive ROM exercises in which a muscle tendon unit is stretched with a constant force and held for a period. During this time the strain in the muscle changes without increasing the force. The therapist can then relax the muscle, rest it, and then repeat the exercise, stretching the tissue to a new length while maintaining the pressure, allowing the tissue to stretch more without increasing the force. Understanding how creep functions, and that force does not have to be continuously increased to achieve lengthening, helps to make injuries, such as avulsion fractures, less likely to occur during therapy sessions.

The Body in Motion: Dynamics

When the forces acting on a body become unbalanced and the sum of these forces is not equal to zero (resultant force), the body will move in the same direction as the resultant force. The displacement, in terms of acceleration, is directly proportional to the resultant force by Newton's second law:

$$\vec{F} = \vec{m}\vec{a}$$

where F is the resultant force, m is the mass, and a is the acceleration.

Evaluation of the left side, or the force side, of this equation is the science of kinetics, whereas evaluation of the right side, or the displacement side, is the science of kinematics.

Kinetics

Kinetic gait analysis can be used to evaluate normal weight bearing, identify alterations in weight bearing, aid in the diagnosis of disorders of locomotion, and evaluate treatment effects. The typical format for evaluating the forces involved in the motion of an animal is the use of the force platform. The force platform is a plate instrumented with a series of strain gauges or crystals and mounted into the floor. Because of their symmetry and convenient speed, the walk and trot are the conventional gaits that are used for evaluation of gait and assessment of lameness. The trot is usually the easier gait to obtain data. At a walk, dogs are more likely to be distracted and have a less consistent gait. When the animal steps on the plate, the sensitive strain gauges or crystals generate an electrical signal that is sent to a data processor and a computer. Multiple signals, collected over time, define the stance phase of the gait, from the time the foot strikes the plate until the instant the foot leaves the plate. The right side of the equation, the



Figure 24-9 The dog is trotting over the force platform, which is connected to a computer for the measurement of ground reaction forces.

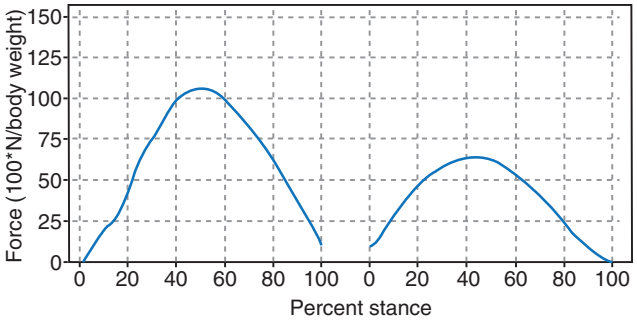


Figure 24-10 Vertical forces of the forelimbs and hindlimbs while trotting. Note that there is only a single smooth curve for the forelimbs and hindlimbs, with the greater forces placed on the forelimb.

displacement side, is kept constant by setting the velocity and acceleration to a well-defined range, for example, trotting at a velocity of 1.7-2 m/s and acceleration of ± 0.4 m/s^2. To generate a readable report, the animal is trotted across the plate such that the front and rear limb, on the same side, fully contact the plate once (Figure 24-9). Often an animal may not lead consistently or may be cautious of the plate by shifting weight, attempting to avoid contact. Therefore a single tracing is not considered reliable by itself. Several identical tracings (usually a minimum of three) are considered more reliable in presenting to the evaluator a more realistic representation of the actual gait routine for that animal.

The force plate is capable of measuring reaction forces in all three dimensions: the vertical (*z* axis), the horizontal (*y* axis), and the transverse direction (*x* axis). Pressure pads or mats have also been developed and have the advantage of being able to evaluate patients too small to participate in force platform analysis and to collect data from multiple limb strikes. This may allow the objective evaluation of small dogs and cats with conditions such as avascular necrosis of the femoral head and medial patella luxation.[2,3] However, they typically only measure relative weight bearing by a limb rather than actual force, and they do not allow the resolution to *z, y,* and *x* forces.

Vertical Ground Reaction Forces

Pressure exerted in the vertical direction on the force plate generates an equal but opposite reaction force in the force plate. This vertical GRF represents the animal's weight-bearing performance and is often measured as a percent of body weight. The term *peak force* represents the maximum value of the GRF during the stance phase of gait, whereas *impulse* represents the generation or dissipation of reaction force occurring over time. Normal peak vertical GRF for the front limb at a trot is 110-125% of body weight, and for the rear limb is 65-75% of body weight,[4] and are typically symmetric with a single peak (Figure 24-10). Walking generates a biphasic Z force curve similar to that seen in humans, resulting in a classic m-shaped graph in which the initial peak represents the vertical force component associated with the initial paw strike in the early stance phase and the second peak represents the increase in vertical force at the time of toe off or propulsion (Figure 24-11).[5] Typical peak vertical force (Z_{peak}) measurements for the fore- and rear limbs while walking are lower than those measured while trotting and are typically 60% and 30% of body weight, respectively. Z_{peak} is affected by the gait, velocity, and acceleration of the dog, the dog's body weight, conformation, and musculoskeletal structure.[6-8] In both the walk and the trot, Z_{peak} increases as the forward velocity of the animal increases.[6,9]

Impulse, the area under the force-versus-time curve, is the change in momentum over time. As velocity increases at a trot, the vertical impulse (VI) increases as a result of the increased force.[6,9,10] When comparing only peak vertical GRFs between individuals, how rapidly the limb is loaded and how long the limb bears the load are overlooked. These factors affect the impulse value and are not reflected in the peak force. If the limb bears load longer, for a similar peak load, the impulse will be significantly greater (Figure 24-12). Z_{Peak} of a rear limb is greater while trotting than while walking, but because the stance time is longer at a walk, the impulse is greater while walking than while trotting. Therefore changes in both peak reaction force and impulse should be evaluated when rating gait performance, and repeated evaluations may only be

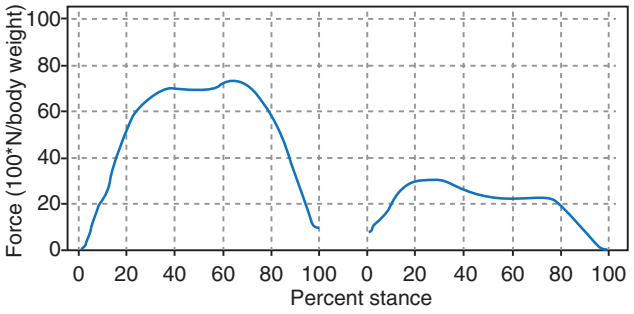
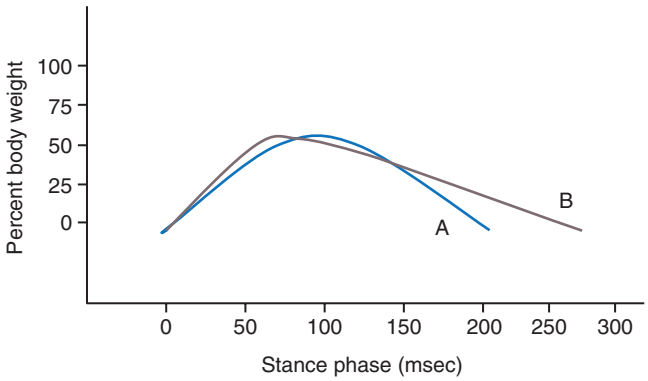


Figure 24-11 Vertical forces of the forelimbs and hindlimbs while walking. Note the biphasic nature of the force curve.



	Peak 100*N/N	Impulse 100*N-sec/N	Time msec	Average rise slope N/msec
A	50.97	5.56	207	1.34
B	52.45	8.49	283	4.77

Figure 24-12 The rate of limb loading and stance time affects the impulse value and is not reflected in the peak force. If the limb bears load longer, for a similar peak load, the impulse is greater.

compared if the dog is ambulating at the same gait, and velocity, and there is no acceleration or deceleration while measuring GRFs.

The animal's COG is particularly important in weight distribution at a stance or while in motion. Traveling uphill tends to shift the balance of forces toward the hindlimbs, and moving downhill shifts the forces to the forelimbs.[8] These factors may affect the forces generated during muscle contraction and be used advantageously during strengthening programs. Further evaluation of kinetic information includes symmetry of the tracing, and comparisons of tracings to the contralateral limb and previous tracings. In cases of lameness, the peak vertical GRF and VI are significantly reduced. Coefficients of variation between trials range from 5% to 9% for Z_{peak} and VI, whereas variation is higher for braking and propulsive forces. Most of the variability is due to dog and trial variation, with little influence on variation by experienced handlers.[11] The symmetry of the tracing can also be altered with lameness. If the shape of the tracing is shifted to the left or right, the animal is quickly loading the limb and peaking early in the stance phase and then slowly unloading the limb throughout the remainder of the stance phase, or slowly loading the limb and quickly unloading it. The clinical significance is not clearly understood regarding this shift, but one study has suggested that a combination of Z_{peak} and the falling slope (unloading the limb) is more valuable in assessing lameness caused by cruciate ligament disease than either measure by itself.[12] Changes in the shape of the tracing could represent lameness because it is reasonable to surmise that a painful limb would not be allowed to increase its load all the way to midstance but would peak early, or the limb would be rapidly unloaded, when the limb pain reaches an intolerant level. However this shift has been seen in contralateral limbs that are compensating for lameness in other limbs. In the compensation mode, loading of the limb peaks early to reduce the load on the painful limb. The difference may be related to the Z_{peak}. In the case of the shift occurring with lameness, the Z_{peak} is less than normal, whereas in the case of compensation, the left shift in the compensating limb is normal or higher than normal.

The stance time may also be assessed. The stance time of the gait cycle is the time when the limb is in contact with the ground and depends largely on the velocity of the subject. As the velocity increases, the stance time decreases.[9,10] In addition, a lame limb typically has a shorter stance time compared with the unaffected limb.[7,8,10,12]

Horizontal Ground Reaction Forces

Pressure exerted in the direction of the animal's motion occurs when the animal places the foot on the plate. This creates a GRF that is equal to the applied pressure but in the opposite direction. This GRF pushes back against the forward motion of the animal resulting in a braking action. As with the vertical force, the intensity of this braking force increases to a peak value then begins to decrease (Figure 24-13). However, there are important differences. Unlike the Z_{peak}, the braking force rises and falls in the first half of the stance phase, such that the horizontal GRF is zero at the midpoint of the stance phase. In addition, the braking force is opposite to the direction of animal motion and is therefore negative and appears below the horizontal axis. After the animal passes the midpoint of the stance phase, the pressure exerted by the foot on the plate causes a GRF that is in the same direction as the animal's motion. This GRF pushes the animal along its motion and is therefore a propulsive force. Similar to braking, the propulsive force increases to a peak value and

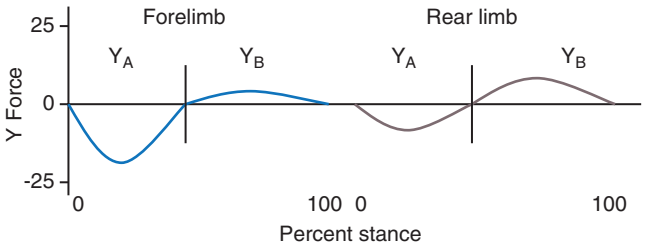


Figure 24-13 Braking and propulsion forces of the forelimbs and hindlimbs while trotting. Note the biphasic nature of the force curve, with the negative deflection representing braking, and the positive deflection representing propulsion.



Figure 24-14 A computerized system may be used to measure weight bearing on each limb while standing. In addition, the distribution of weight to various limbs may be assessed during therapeutic exercises, such as weight shifting.

then decreases. The propulsive force cycles completely within the second half of the stance phase. These forces of braking and propulsion occur along the *y* axis. Normally, the forelimbs have greater braking forces than propulsive forces, and the hindlimbs have greater propulsion as compared with braking.

Transverse Ground Reaction Forces

Some side-to-side pressure is exerted by the foot during stance phase; however, in the case of the quadruped animal moving in a straight line, the intensity is minimal when compared to the *x* and *y* forces. As a result this GRF is seldom used in the evaluation of lameness. The forces may be quite significant, however, in an animal that is turning rapidly, such as negotiating vertical weave poles during an agility competition.

Measurement of Static Weight Bearing

Force plate and motion analysis systems are costly and require a relatively large dedicated workspace. Therefore their use in private practice is limited. Subjective lameness scales have traditionally been used, but have significant limitations. Weight bearing at a stance may be one method to obtain quantitative data regarding the relative amount of force placed on each limb while standing. A computerized system to objectively evaluate weight bearing at a stance in dogs may be useful for this purpose (Figure 24-14). Preliminary studies indicate that there is good correlation between Z_{peak} at a trot determined by force plate analysis and static weight-bearing pressures in dogs with rear-limb lameness.[13] In fact, the difference between the lame and contralateral sound limb may be greater at a stance because there are three other limbs to shift the weight to, and the limbs are in contact for a longer time than at a trot, allowing additional time to shift the weight.

Kinematics

Kinematics is the science of the right side of Newton's fundamental equation of motion. The right side of the equation relates displacement to its cause, the unbalanced force. Typically the equation is stated using acceleration as the unit of displacement. Acceleration is the rate at which the velocity changes with respect to time. Velocity is the rate at which the displacement occurs with respect to time. When viewing an animal in motion, linear motion is easily understood as the animal is seen moving in a straight line from one point to another. However, when the internal mechanics of the animal's muscles and bones are studied, the initiating action is more circular than linear. This is analogous to the motion of an automobile. When viewing the car in motion, the action is linear as the car moves from one point to another. However, when the internal mechanics of the car are studied, the action is circular involving drive shafts, axels, and wheels. In the car, the force is generated by the up-and-down motion of pistons and rods. The action of this force is transmitted to a rotating camshaft to circular gears and rotating drive shafts and ultimately to the wheels, which rotate to propel the car forward in linear motion. In the animal, the force is generated by the up-and-down contraction of actin and myosin elements in the muscle. The contraction of these muscle elements causes rotation of the bones about the joints, propelling the animal forward in linear motion. A car is rated in terms of torque (i.e., its ability to generate force for motion). Similarly, in the analysis of the forces that generate motion in the body, torque or angular moment is important. Because circular motion is important in the generation of body motion, angular displacements must be measured and from these measurements angular momentum can be derived.

Real-time photographic, optoelectric technology is effective in measuring angles during continuous motion.

Optoelectric technology involves the use of infrared cameras positioned for views in three-dimensional space. The animal is prepared with reflective markers secured to the body at predetermined points (Figure 24-15). These points are anatomic features that represent joint anatomy. Infrared light emitted from individual cameras is reflected from these markers back to the camera (Figure 24-16). As the joints move, the cameras record the displacement of the markers. Data points are analyzed with respect to a three-dimensional grid set up and calibrated at the beginning of the test. Such a system can record joint angle excursion throughout a full stride. Typical data acquired include stride length, stance and swing times, joint angles in all planes of motion, and linear and angular joint velocity and acceleration.

A stride includes a single swing and stance phase. The stance time of gait is the period when the foot is in contact with the ground, and the swing phase occurs when the foot is off the ground between stance phases. The swing phase has three distinct movements. The limb initially swings caudally following propulsion. The limb is then pulled cranially, and finally travels briefly caudally toward the ground in preparation for the next stance phase.[14,15] The distance from initial contact of one limb to the point of second contact of the same limb is termed *stride length,* and a *gait cycle* is a series of events that includes one stride for each of the four limbs.[5,14,15]

The joint angle data can be plotted over the period of a single stride (Figure 24-17). These patterns of motion are specific for each joint and gait. Some results have been obtained both for normal gait and for lameness associated with cranial cruciate deficiency[16] and hip dysplasia.

Combining Kinetics and Kinematics

The evaluation of force (kinetics) and of displacement (kinematics) can be combined or connected via Newton's second law of motion. This connection is possible when kinetic and kinematic data are synchronously obtained.

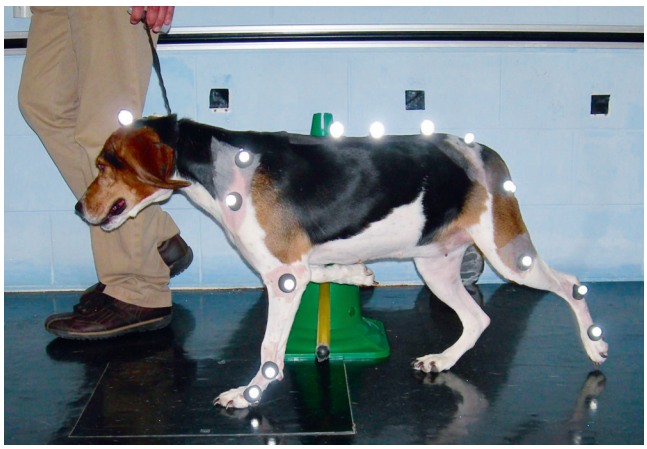


Figure 24-15 The animal is prepared with reflective markers secured to the body over anatomic features that represent joint anatomy.

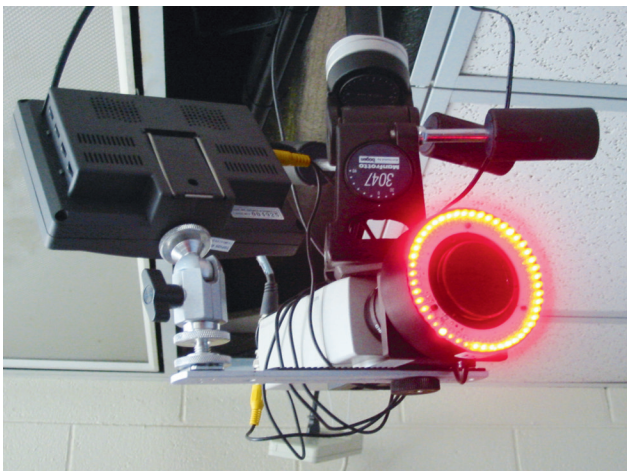


Figure 24-16 Cameras that detect infrared light reflected back to the camera are used to capture the motion of reflective markers as the animal moves through the test area.

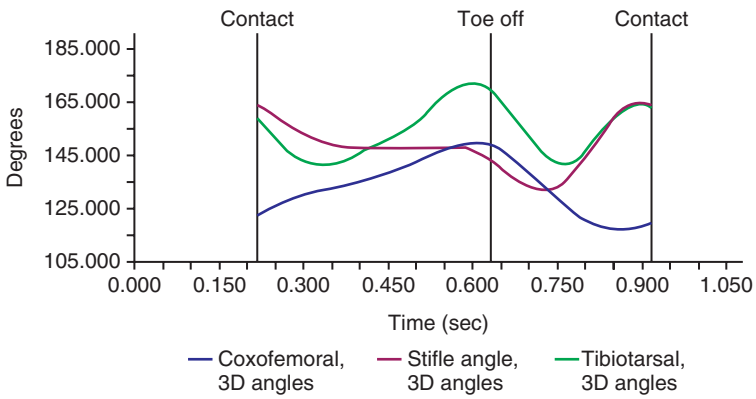


Figure 24-17 Angular data from each joint can be plotted over the period of a single stride. This graph represents motion of the hip, stifle, and hock joints at a walk for a dog.

Such effort requires special technology to coordinate data acquisition. Once this is achieved, the constant in Newton's equation, mass, must be determined for the body segments involved in the analysis. This includes not only the mass value itself, but also the distribution of mass (mass moment of inertia). Therefore Newton's second law may be expressed specifically for linear motion (the study of the entire animal moving in a linear fashion):

$$\vec{F} = m\vec{a}$$

where F is the force (kinetics: evaluation of force), m is mass, and a is acceleration (kinematics: evaluation of linear displacement). Specifically for angular motion (the study of the interior forces of the muscles and the angular movements of the bones):

$$\bar{M} = \vec{I}\bar{\alpha}$$

where $\bar{M}$ is the moment (kinetics: evaluation of moment of force), I is the mass moment of inertia, and $\bar{\alpha}$ is the angular acceleration (kinematics: evaluation of angular displacement).

When kinetics and kinematics are connected mathematically, forces and moments of individual joints can be determined by calculating the values from force plate ground reaction and joint motion values. This approach is referred to as *inverse dynamics.*

Kinetic and Kinematic Gait Analysis Research

Normal Gait

Knowledge of typical weight-bearing patterns in normal dogs and those with orthopedic conditions provides information that may be valuable in the rehabilitation of patients. In a normal standing patient, each forelimb bears approximately 30% of the dog's body weight, and each rear limb bears 20% of body weight. The relative proportion of weight bearing on the fore- and rear limbs is relatively consistent at the walk and trot, which are symmetric gaits. However, because of the relationship of velocity and acceleration to the forces placed on the limbs during the stance phase of gait, significant increases in absolute forces during weight bearing occur with increasing speed at various gaits. For example, a dog may have Z_{peak} measurements of 55% and 30% of body weight at a walk in each fore- and rear limb, respectively. The forces may increase to 100%, 118%, and 125% in the forelimbs and 70%, 80%, and 85% in the rear limbs while trotting at 1.5-1.8, 2.1-2.4, and 2.7-3 meters per second, respectively.

Several studies have evaluated normal GRFs and kinematic motion of dogs at the walk and trot.[4,6,7,9-12,14,17-27] Studies in normal dogs indicate that each joint has a characteristic and consistent pattern of flexion and extension during the walk, but complex joint movements may occur during the swing phase.[14,19,27] Two additional studies described kinematic motion of the joints of normal dogs at a trot.[17,18] The coxofemoral and carpal joints were characterized by a single peak of maximal extension and the femorotibial, tarsal, scapulohumeral, and cubital joints had two peaks of extension, with one peak occurring prior to the stance phase, and a second peak occurring during the stance phase.[17,18] There were insignificant differences between trials and few differences among dogs of similar body type. It was concluded that kinematic gait analysis may provide a reliable description of joint motion in dogs of similar size and conformation. A recent study has evaluated not only the joint angle excursions at a walk, but also defined angular velocity and acceleration rates of those movements. This study indicated that these parameters are consistent and repeatable and helps to further characterize the normal walking gait of hound-type dogs.[27] Additional studies are warranted to determine the sensitivity and specificity of alterations in these parameters as indicators for specific causes of lameness.

Cranial Cruciate Ligament Disease

Following cranial cruciate ligament rupture (CCLR), Z_{peak} may be only 50% of normal at a walk, and dogs may be non–weight bearing at a trot.[28] By 7 months after extracapsular surgical repair, weight bearing is usually equal in both rear limbs at a walk. Experimentally, Z_{peak} at the trot with an extracapsular repair technique was normal by 20 weeks after surgery.[29] Weight bearing in the contralateral rear limb initially increases, likely as a result of redistribution of weight from the affected limb to normal limbs, then returns to normal as weight bearing improves on the affected limb.

Dogs with CCLR have variable degrees of lameness and demonstrate altered movement in the hip, stifle, and hock joints.[16] The stifle joint angle in the cruciate-deficient state was more flexed throughout the stance and early swing phase of stride and failed to fully extend in late stance, when limb propulsion is typically developed. In addition, extension velocity was negligible. The hip and hock joint angles, in contrast, were extended more during the stance phase, perhaps as a result of compensatory changes.[16] Paw velocity and stride length were also significantly reduced in dogs with CCLR walking on a treadmill as compared with the contralateral side, and to normal dogs.[30]

Changes in kinematics were evaluated over a 2-year period in dogs with CCL-deficient stifles.[31] All changes occurred primarily between 6 and 12 months. Peak cranial tibial translation increased by 10 mm and the range of abduction and adduction motion nearly doubled (from 3.3 degrees to 6.1 degrees) after CCL transection and did not improve with time. These changes may overload secondary restraints such as the medial meniscus. This objective information further defines the pathologic gait of CCLR patients.

More sophisticated techniques have been used to quantify net joint moments, joint powers, and joint reaction forces (JRFs) across the hock, stifle, and hip joints in Labrador retrievers with and without CCL disease using inverse dynamics calculations.[32] Kinematic, GRF, and morphometric data were combined in an inverse dynamic approach to compute hock, stifle, and hip net moments, powers, and JRF while trotting. Vertical and braking GRF and JRF were decreased in CCL-deficient limbs. In fact, the braking/propulsion ratio shifted from 50%:50% in normal limbs to 33%:66% of the stance phase in CCL-deficient limbs. It is possible that reduced braking may result in decreased cranial tibial thrust. In affected limbs, extensor moments at the hock and hip, flexor moment at the stifle, and power in all three joints were less than normal. Greater joint moment and power of the contralateral limbs compared with normal were also identified. Other compensatory changes included increased time spent with the sound foot on the ground and an increased amount of force applied to all joints of the sound limb, especially during propulsion, compared with normal and CCL-deficient limbs. Propulsion generated around that joint at push-off was three times greater than normal, reflecting increased stifle extensor muscle contraction. Lameness predominantly affected forces during the braking phase and extension during push-off, and a greater contribution of the contralateral limbs to propel the dog forward was identified. These changes may be somewhat protective against damage to the articular cartilage and joint and may be mediated, in part, by the nervous system and a response to pain. In one study using a CCL transection and hindlimb deafferentation model, extension of the hip, knee, and ankle joints of the unstable limb was increased, and braking of the unstable knee was delayed or attenuated.[33] These changes are different than models that have not used deafferentation. The model used in this study results in rapid onset and progression of osteoarthritis with cartilage erosions present by 13 weeks, perhaps because there is no pain sensation or joint proprioception is altered. Stifle hyperextension resulting from limb deafferentation, and knee instability resulting from CCL transection may work synergistically to create increased tibiofemoral motion and changes in the loading of articular surfaces that result in rapid joint breakdown.

Hip Dysplasia

Dogs with hip dysplasia also have reduced weight bearing on the affected limbs. In addition, hindlimb propulsion is reduced in dysplastic limbs. These differences persist 1 month after total hip replacement, and in many instances, weight bearing is initially lower after surgery in the operated limb as compared with presurgical values.[34] By 3 to 6 months after surgery, weight bearing is improved at a trot. In most dogs, hip dysplasia is bilateral, and weight may actually be transferred from the unoperated side to the side with the total hip replacement. In addition, young dogs having triple pelvic osteotomy for treatment of hip dysplasia have weight bearing while walking on the operated side that is similar to preoperative levels by 10 weeks, and have significantly greater weight bearing on the operated side by 28 weeks after surgery as compared with preoperative values.[35]

A recent study reported that dogs with hip dysplasia have complex gait alterations that may not be manifested as overt clinical lameness, making subjective evaluation of lameness difficult. Dynamic flexion and extension angles and angular velocities have been calculated for the coxofemoral, femorotibial, and tarsal joints.[36,37] In the late stance phase of the gait cycle, the hindlimb gait of dogs with hip dysplasia was characterized by a more extended coxofemoral joint, and more flexed femorotibial and tarsal joints throughout stance and early swing phases of the stride. All joints flexed more rapidly in the early swing phase. These changes may be the result of pain in the hip joint early in the stance phase when maximum weightbearing and propulsion would be expected, with the increased extension at the end of the stance phase the result of a relatively passive sudden extension of the hip to complete the gait cycle. Other characteristic changes in the swing phase of the gait cycle, such as an increased stride length and decreased Z_{Peak} measurements, were also evident in dogs with hip dysplasia. Another study determined that dogs with hip dysplasia had a greater degree of coxofemoral joint adduction, greater range of abduction-adduction, and greater lateral pelvic movement, compared with controls.[26] These differences were thought to be indicative of compensation in gait of affected dogs as a result of discomfort or biomechanical effects attributable to hip dysplasia and degenerative joint disease.

Joint motion while walking on a treadmill was evaluated in dogs with borderline radiographic signs of hip dysplasia and normal GRFs. Affected dogs had a later time for maximal flexion of the hip joint and more flexion and ROM of the stifle joint, compared with dogs with no radiographic signs of hip dysplasia. In addition, maximal angular velocity of the stifle and tarsal joints was significantly greater during the swing phase in dogs with borderline hip dysplasia, suggesting that dogs with borderline hip dysplasia have altered joint kinematics.[38]

Elbow Arthritis

The compensatory load redistribution caused by osteoarthritis of the elbow joint was determined by evaluating GRFs in one study.[39] Dogs with osteoarthritis had a compensatory gait pattern that reduced the stress on the affected limb. The load was reduced on the lame limb and increased on the contralateral hindlimb. A symmetry index indicated a weight shift to the contralateral forelimb and diagonal hindlimb, which resulted in a more balanced weight distribution compared with normal dogs. It is possible that

forelimb lameness could overload nonaffected extremities and the vertebral spine.

Lumbosacral Disease

Thoracolumbar spinal movement has been evaluated in normal Belgian Malinois dogs and those with subclinical radiographic changes of the lumbosacral region.[40] Markers were placed on the spinal processes of C7, T6, T13, L3, L7, and S3, and dogs were walked on a treadmill. ROM in the transverse and vertical direction and the time of occurrence of the maximal marker position were calculated. Significant kinematic changes were detected between clinically sound dogs with radiographic lumbosacral changes and dogs with no radiographic abnormalities.

Golden Retriever Muscular Dystrophy

Various forms of muscular dystrophy in people cause altered gait kinematics. Stifle and tarsal kinematics of the pathologic gait of dogs with golden retriever muscular dystrophy (GRMD) were evaluated while walking at a self-selected speed and compared with carrier littermates (controls).[41] GRMD dogs walked significantly slower than controls. At the stifle joint, both groups displayed similar ROM, but GRMD dogs walked with the stifle joint relatively more extended. At the hock joint, GRMD dogs displayed less ROM and walked with the joint relatively more extended compared with controls.

Dynamic Gearing

As described previously, the biomechanics of the quadruped animal is similar in a general sense with the mechanics of vehicles such as automobiles and bicycles. These vehicles produce rotary motion, which is transferred to linear movement. The efficient transfer of energy in these machines is modulated by gearing mechanisms. The bicycle has a simple gearing mechanism and can be used as an analogy to the quadruped animal. The cyclist generates motion by pedaling. In this action the cyclist pushes on the pedal, producing a moment of force that turns the sprocket, which turns a second sprocket in the rear wheel. The relationship between the pedal sprocket and the wheel sprocket is described as a *gear ratio.* Sprockets with different diameters can affect the energy transfer, making it more or less difficult for the cyclist to move the bicycle. Gearing mechanisms have been proposed for biologic systems, including people and animals.

In the body, muscles produce forces that function as moments that rotate bones about joint centers of motion. A second set of forces, those that resist gravity, the GRFs, also figure into the production of motion. These muscle moments can be labeled as *internal moments* as opposed to those produced by the GRFs, which can be referred to as *external moments.* Each set of these forces has associated moment arms. With the hip, for example, body weight causes the femur to rotate into flexion while the extensor muscles of the hip cause the femur to rotate into extension. Therefore for every unit of moment causing hip flexion, there is a corresponding moment causing hip extension. Like a bicycle gear, for every unit of turn by the pedal, there is a corresponding turn of the rear wheel. This relationship can be described in terms of a gear ratio. A low ratio means that the turn of the pedal gear generates nearly the same turn in the wheel, whereas a high gear ratio means that for every turn of the pedal gear the wheel gear turns many times. One can conceptualize the interplay of external moments (GRF moment) and internal moments (muscle force moment) as a gearing mechanism with a gear ratio. This ratio is found by dividing the external moment arm by the internal moment arm.

Gear ratios are important especially when acceleration is initiated. In the case of the bicycle the cyclist turns the pedals faster to generate more force for more speed. In the animal it might be assumed that the speed of muscle contraction is increased to generate more velocity. Some have observed that as the speed of muscle contraction increases there is a decrease in the force of contraction; however, it has also been observed that as the speed of contraction increases, the power (rate of work) generated is increased. When the cyclist accelerates the bicycle, there is an upshifting of the gears (i.e., a transition from a low gear to a high gear that reduces the requirement for additional force produced by the cyclist). That is, for a given pedal revolution per minute (a constant force), the bicycle can still accelerate because the gear ratio is increased. It has been theorized and demonstrated that in the dog there is a similar change in gear ratios as the animal accelerates. This is called *dynamic gearing.* For a given velocity of muscle contraction (the optimal velocity), the ratio between the external moment arm and internal moment arms increases, facilitating acceleration. These moment arms change because of the change in the angles of the joints.

In the case of running, dynamic gearing has been demonstrated to occur about the shoulder and stifle joints.[42] Such a phenomenon was not demonstrated when evaluating the gear ratios about the carpus, elbow, hock, and hip. Dynamic gearing facilitates motion, especially acceleration and running, by maximizing the efficiency of muscle contraction and the transfer of energy from one segment to another and eventually to the ground. Alterations in mass and its distribution in limb segments, imbalance in the contraction of muscle groups, disruption of angular motion in the joints, and changes in limb dimensions can potentially influence the relationship between the various joint moments (i.e., the gearing ratios), resulting in suboptimal performance and maybe creating more stress and strain on the elements of the system. For example the effects of patella instability and luxation can be better visualized from the perspective of the efficient or inefficient

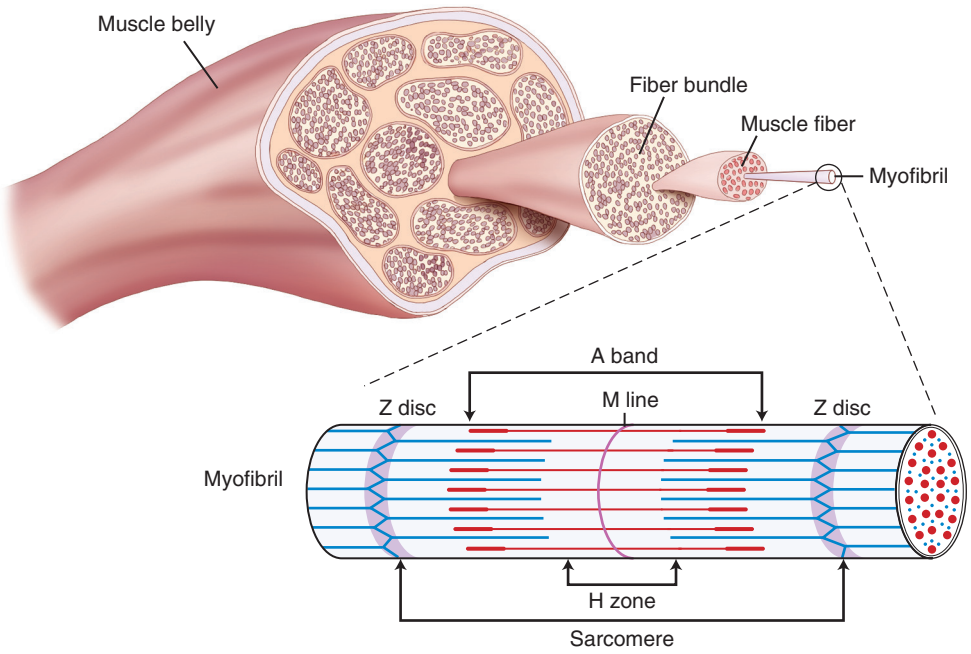


Figure 24-18 Muscle fibers are composed of smaller subunit myofibrils. The sarcomere is the unit of the contractile system of muscles found in the myofibrils and consists of actin and myosin arranged in transverse bands.

transmission of energy across abnormal segments. It has been shown that in the process of running, hock joint excursion is 58 degrees, whereas stifle excursion is 36 degrees; however, the quadriceps is not only responsible for extending the stifle, but it is also partially responsible for extending the hock.[42] This is related to the muscles that cross both joints and to the various moments that these muscles produce. When the moment arm decreases in the stifle because of a dislocated patella, the gear ratios are changed and the quadriceps is less efficient, which could translate to less efficiency in extension of the stifle and hock, resulting in poor performance. Therefore what happens in the stifle has an effect on the hock. Such theories at this stage are assumptions that require more testing by combined kinetic and kinematic measures.

Energetics

Isolating analysis to specific biomechanical phenomena, such as joint moments and GRF, does not reflect the influence of a whole host of variables that are involved in the generation of gait. Another perspective in the study of animal motion is that of energy flow. Throughout motion and gait, energy is transferred from the muscle to bone and then from the bone back to muscle. This transfer of energy depends not only on the forces of weight bearing, and linear and angular motion, but also on the physiologic and metabolic status of the muscles, the integrity of the connective tissue structures, the mobility of the joints, and the neurologic control of gait. Energy flow can be analyzed by assessing the mechanical energy used by the body as work. Work can be assessed mathematically by determining power values for physical activities such as walking, running, jumping, and so on. The core of physical rehabilitation is the use of these activities as a means of therapy. Determining work and power values of the animal's performance can facilitate decisions regarding which activities and their intensity are appropriate for the treatment of specific conditions.

In mechanical systems such as the musculoskeletal system of the body, energy is neither created nor lost but transferred between a stored state (potential energy) and an expended state (kinetic energy). In the body, this transfer physically occurs between muscle and bone, and the result is body movement, such as walking, trotting, running, or swimming.

Biomechanics of Muscle

Muscle is the principal source of the mechanical energy used in the generation of motion. It is important to understand the biomechanics of muscles so that strategies may be designed to restore function in animals with decreased muscle force or movement caused by disease. A brief review of basic muscle contraction is necessary to more completely understand the biomechanics of skeletal muscle. Muscle fibers are composed of smaller subunit myofibrils (Figure 24-18). The sarcomere is the unit of the

contractile system of muscles found in the myofibrils, and consists of actin and myosin arranged in transverse bands. The myosin filaments have crossbridges that attach to the actin filaments. During muscle contraction, it is the relationship of the crossbridges to the actin that results in shortening of the myofibrils, similar to an oar that is rowing to advance a boat through the water. This is also known as the *sliding filament theory.*[43] The force of muscle contraction depends on the relative number of crossbridges formed by the interaction of the actin and myosin filaments via the crossbridges.

Muscle not only expends this energy with contraction of myofibrils but also stores and absorbs this energy during the process of weight bearing and gait. The elastic components of muscle (epimysium, perimysium, and endomysium) and tendon store potential energy as the muscle is passively stretched. For example, as the hamstrings contract, the quadriceps is stretched. After contraction in the hamstrings is terminated, the quadriceps releases the stored potential energy and springs back to its resting length. This spring-like function of muscle-tendon units prevents overstretching and damage to the muscle. When an animal stands motionless, the muscles contract in a coordinated and balanced fashion, without the production of motion. Muscle contraction under such conditions is termed *isometric contraction.* In isometric contraction, all the mechanical energy is stored in the muscle as potential energy. Muscle also absorbs energy from impact and dampens vibration, protecting and preserving the moving parts of the skeletal system. Although the function of muscle in expending energy (i.e., kinetic energy in the generation of motion, which is the focus of the physical therapist) is important, the function of muscle in storing energy is equally important. Muscle fibrosis and the loss of elasticity compromise its capacity to store and absorb energy, rendering joint structures and surfaces more vulnerable to additional injury and wear. In treatment plans of osteoarthritis, the focus is naturally on the condition of the articular cartilage, subchondral bone, internal and external ligaments, and the status of the joint capsule; however, the supporting muscles must also be included. The therapist can take advantage of this opportunity to fill the gap in the management of osteoarthritis by focusing attention on the condition of the muscle and applying therapeutics designed to improve muscles' capacity to store, expend, and transfer energy.

The term mechanical energy represents the ability of a mechanical system to do work, such as producing motion.[44] Work represents the flow of mechanical energy between the kinetic and potential states, between the internal parts of the system, and between the system and its environment. The motion of the internal components such as muscle and bone represents internal work, which leads to motion of the body, and represents external work. Work is mathematically calculated by multiplying the force that generates the motion by the distance moved. The rate at which work is performed represents the power of the system. Power, therefore, is the rate of flow of mechanical energy and is mathematically calculated by dividing work by time (W/t), or the rate of work.[44] Power is a sensitive indicator for abnormal gait because it depends on the function of all components of the musculoskeletal system.

Positive Work: Concentric Muscle Contraction

When muscle contracts, it produces a moment that causes a bone to rotate. To calculate the work done, the moment is multiplied by the displacement (angular displacement). The product of angular moment and acceleration is positive when the direction of the angular moment and acceleration is the same. This positive product represents positive work and is associated with concentric muscle contraction, which is defined as the circumstance in which the direction of the motion is the same as the contraction. For example, when a dog jumps a barrier, the quadriceps muscle is producing positive work by extending the stifle joint. The direction of the contraction (i.e., the shortening of the muscle) is in line with the action of extension of the stifle. Co-contraction and positive work are ideal for muscle function and pose less risk for muscle injury as compared with eccentric muscle contraction.

Negative Work: Eccentric Muscle Contraction

Eccentric contraction occurs when the direction of the contraction or shortening is opposite the direction of the motion of the bone to which the muscle is attached. Because the directions are opposite each other, the product of the moment and acceleration is negative, and therefore the work is negative. For example, when a dog is landing on its front limbs after jumping a barrier, the triceps muscle is contracting, trying to extend the elbow in an attempt to cushion the landing. However, the force overpowers the muscle and causes the elbow to flex. The work the muscle does in this circumstance is negative and is more likely to result in muscle injury, such as delayed onset muscle soreness. When formulating and designing physical activities, the therapist should consider the nature of the work the muscles will endure. Positive work scenarios tend to build muscle strength, whereas negative work could inhibit rehabilitation efforts by contributing to muscle soreness.

Zero Work: Isometric Contraction

Isometric contraction is defined as a contraction that is not associated with any movement. Because no displacement takes place, the product of joint moment and displacement is zero, so work is not done. Biomechanics traditionally involves the "inverse solution" to problems, in which displacement (the outcome of applied force) is measured first and the generating force is then calculated. Assessment of the nature of isometric contraction in terms of its causative

force is difficult because no displacement occurs except within the muscle itself and at a microscopic level. To understand these forces and energy conditions, direct measurement is required. However, isometric contraction is an important function of muscle and can be used by the therapist to increase strength and elasticity.

Muscle Force Generation

The force generated by the muscle varies with the muscle's length. Muscles generate relatively little tension when a muscle is maintained in a very long or very shortened state. Muscles generate greater force with the muscle at intermediate, or optimal, lengths. For example, the biceps muscle generates the greatest force in humans when the elbow is at an angle of approximately 90 degrees. The tension generated in muscle is a function of the overlap between the actin and myosin filaments. This phenomenon is known as the *sarcomere length-tension relationship.*[45] With a long muscle length (full extension), there is little overlap between actin and myosin filaments, and because there are few cross-bridges, little tension is generated. As muscle length decreases, there is overlap of actin and myosin, and greater tension may be generated. This reaches its maximum effect when the muscle is at its optimal length. As the muscle further shortens, a point is reached at which further decreases in muscle length do not result in greater tension because there is so much actin-myosin overlap that additional cross-bridges cannot be formed. With even further shortening of the muscle, actin filaments begin overlapping with other filaments on the opposite side of the sarcomere, and there is interference with cross-bridge formation, resulting in decreased muscle tension.

The length-tension situation is altered when a muscle is stretched to various lengths without stimulation, or with passive movement. Near the optimal length of muscle, passive tension in the muscle is almost zero. As the muscle is stretched to longer lengths, however, passive tension increases. Therefore passive tension by stretching the muscle can play a role in providing resistive force even in the absence of muscle activity. The phenomenon of passive muscle tension may be due to titin, a large protein within the myofibrils that connects the thick myosin filaments end to end.[46] This protein may also stabilize the myosin lattice so that high muscle forces do not disrupt the sarcomeres.[47]

The effect of passive tension is greater in muscles that cross two joints, such as the hamstring or caudal thigh muscles. In this situation, it is difficult to fully extend the stifle joint while the hip is flexed maximally because the passive tension of the stretched hamstring muscles prevents further stifle extension.

In contrast to the length-tension relationship, the force-velocity relationship seen in muscle contraction does not have a precise anatomically identified basis. The force-velocity relationship establishes that the velocity of muscle contraction is inversely proportional to the load applied to the muscle.[45] For example, as the load applied to a muscle increases, the muscle shortens more slowly. If the external load equals the maximal force of muscle contraction, muscle contraction velocity is zero. If the load is further increased, eccentric muscle contraction and muscle lengthening occur.

The force of muscle contraction is also proportional to the contraction time.[43] The longer the contraction time, the greater the force of contraction. Although maximum force may be developed relatively quickly in the contracting muscle, longer contraction times generate additional forces in the elastic components of the muscle-tendon unit.

The arrangement of the sarcomeres affects muscle contractile properties. Muscle velocity and excursion are proportional to the number of sarcomeres in series, whereas muscle force is proportional to the total cross-sectional area of sarcomeres.[45] Muscles designed for generation of high forces often have muscle fibers slanted at an angle, an arrangement called *pennate muscles.* Various muscle groups appear to be designed for either the production of high forces or velocity and high excursions. The quadriceps muscles seem to be designed more for production of force, whereas the sartorius muscles, with their longer length and lower cross-sectional areas, are more adapted to high excursions. The caudal thigh muscles are also designed for large excursions.

Muscles generate force and transmit this force via tendons to the bones. If the muscles generate adequate force, the bones rotate about joint axes. This activity can be defined as a *torque,* which clinically represents strength. Torque is a mathematical relationship between force and the length of the moment. Torque, or strength, may be changed by changing the magnitude of force (tension-producing capability of muscles), changing the length of the moment (changing the location of the muscle insertion site), or changing the angle between a force, such as a loading force and the magnitude of the moment generated by a muscle. When a joint is fully extended, there is an unfavorable mechanical advantage to the muscle because the moment is relatively small.[45] Much of the force generated by muscles in this situation compress the joint surface rather than rotate the joint. The maximum moment of most joints occurs with the joint flexed to 90 degrees. However, studies of torque generation have demonstrated that the joint angle at which the muscle generates maximal force is not the same angle at which the moment arm is maximum. In reality, both the moment arm and muscle force are constantly changing during normal joint motion, making the measurement of torque very complex. A thorough understanding of the principles regarding generation of force by muscles may allow development of treatments for specific muscle groups that have a rational physiologic basis and that result in measurable improvements. For example, when administering neuromuscular electrical stimulation

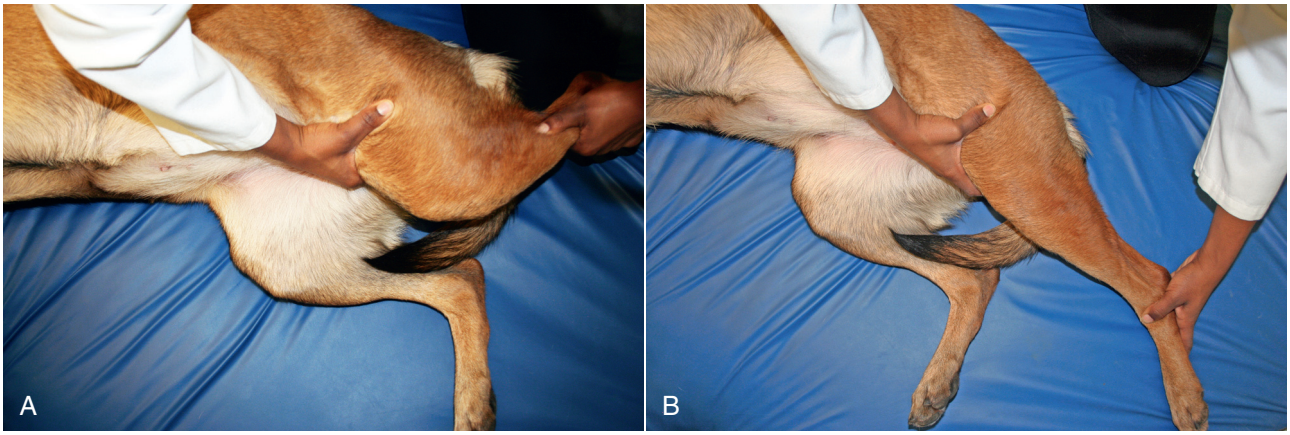


Figure 24-19 If hip joint extension is desired, the contribution of the rectus femoris muscle to resistance may reduce hip extension if the stifle is kept flexed during hip extension (*A*), as compared with extending the stifle (*B*), because of the influence of the rectus femoris muscle on hip extension.

for quadriceps muscle strengthening in a patient with muscle weakness, it may be beneficial to perform the treatment with the stifle in mild flexion as compared with full extension or flexion.

Biomechanics of Exercise Modification

Knowledge of the alterations in body posture or positioning that patients undergo while recovering from surgery or during rehabilitation of chronic conditions helps the therapist to appreciate and take advantage of these factors to improve the effectiveness of therapeutic exercises. In patients with proprioceptive disorders, the size of the base of support affects stability. For example, it is more difficult to balance with the feet close together than it is with the feet spaced wide apart. Similarly, the stability of the ground surface may progress from a static or stable surface, such as the floor, to a more mobile base, such as a balance board or trampoline. The tactile and proprioceptive input may also be altered by standing and walking on various surfaces, such as foam rubber.

Increasing the external load that a limb experiences is important for muscle strengthening. Although it is obvious that increasing the weight that a limb carries (e.g., by using weights or resistive elastic bands) increases the magnitude of resistance, there may also be increased feedback from muscle and joint receptors, enhancing the response. The length of the moment arm undergoing an increased load also affects the resistance that a limb undergoes. Placing the weight or elastic band distally on the limb increases the length of the moment arm and increases the force necessary to move the limb. Therefore if a muscle is relatively weak and a limb is not able to withstand a high load, a leg weight or elastic band should be placed relatively proximal on the limb. As muscle strength improves and additional strengthening is desired, the weight or band may be moved further distally. The speed of the exercise should also be considered. It is usually easier to perform an exercise at a medium rate of speed than a very slow or very rapid rate.

Consideration of limb position is also important when targeting muscles that cross two joints. The tension exerted by a muscle spanning more than one joint depends on the position of the second joint over which it passes, because this determines the total length of the muscle. The semitendinosus and semimembranosus muscles are more effective flexors of the stifle when the hip is also flexed as compared with the hip joint extended. If hip joint extension is desired (with concurrent shortening of the gluteal muscles), the contribution of the rectus femoris muscle (which crosses both the hip and stifle joints) to resistance may be minimized if the stifle is kept extended during hip extension as compared with flexing the stifle (Figure 24-19).

Likewise, hip flexion is limited if the stifle is concurrently extended, but hip flexion increases if the stifle is simultaneously flexed (Figure 24-20). Similarly, hock flexion is greater when the stifle is concurrently flexed, but is limited if the stifle is kept extended (Figure 24-21). These biomechanical considerations are particularly important when planning stretching and ROM exercises.

Sensory facilitation or inhibition may be used to alter muscle responses. For example, compression of a joint may stimulate the joint receptors and facilitate extensor muscle activity and stability around a joint. One method of increasing extensor muscle activity with joint compression is rhythmic stabilization. This activity may be accomplished by placing the dog in a standing position on a compressible surface, such as an air mattress or exercise ball. Weak dogs may be supported with a sling to be certain that they do not collapse. While keeping the dog in a standing position, the dog is gently and rhythmically pushed down (Figure 24-22). As the joints become compressed,

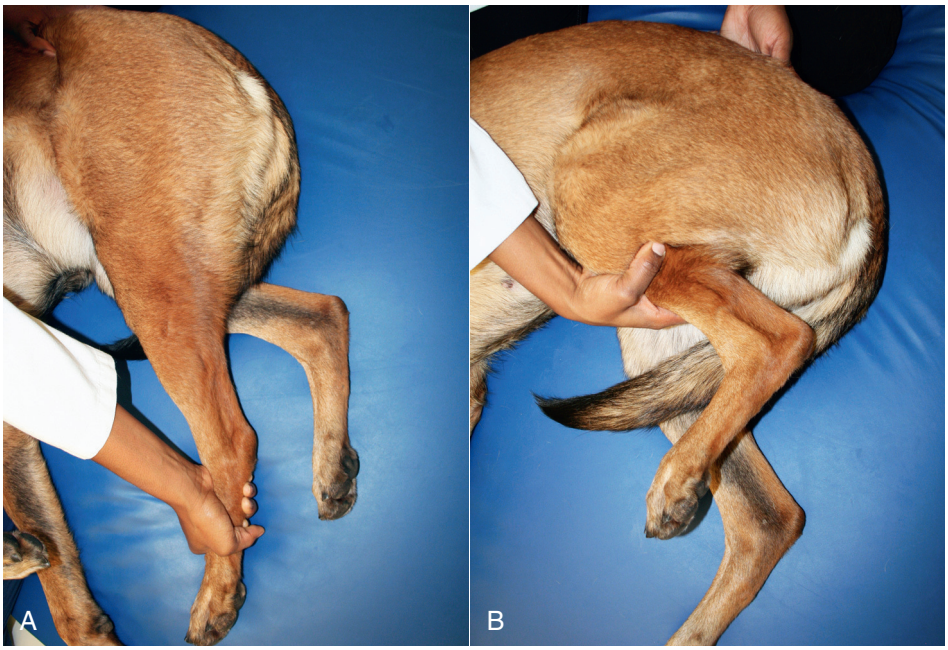


Figure 24-20 Hip flexion is limited if the stifle is concurrently extended (*A*), but hip flexion increases if the stifle is simultaneously flexed (*B*).

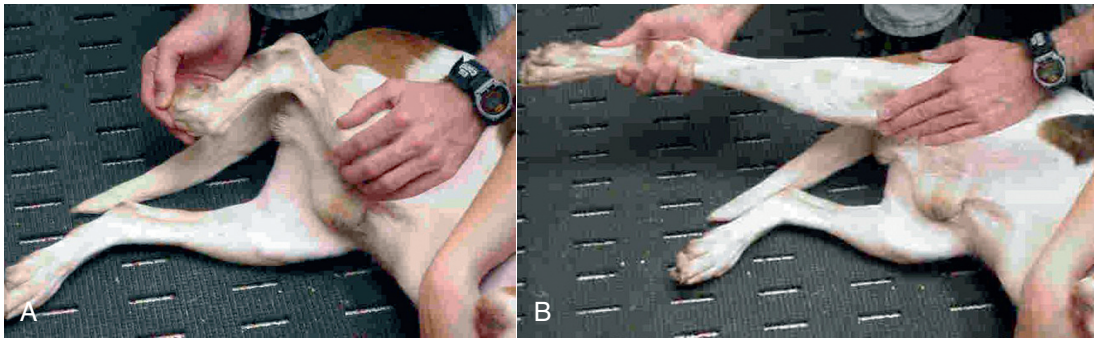


Figure 24-21 Hock flexion is greater when the stifle is concurrently flexed (*A*), but is limited if the stifle is kept extended (*B*).



Figure 24-22 Rhythmic stabilization may be accomplished by supporting the dog in a standing position on an exercise ball and gently pushing the dog down in a rhythmic fashion to stimulate the extensor muscles to contract to maintain body posture.

the extensor muscles responsible for maintaining posture are stimulated to prevent collapse. Conversely, traction separates joint surfaces and is useful if increased ROM is desired.

Biomechanics of Therapeutic Exercises

Walking

Walking is perhaps the most simple therapeutic exercise, but one of the most valuable in the early phases of rehabilitation. Z_{Peak} measurements of the forelimbs and hindlimbs are approximately 54% and 40% of body weight, respectively (Table 24-1). Total motion excursion in the shoulder, elbow, and carpus at a walk is approximately 30, 45, and 90 degrees, respectively (Table 24-2).[14,27] In the rear limb, motion in the hip, stifle, and hock is approximately 35, 40, and 35 degrees, respectively.

Table 24-1 Kinetic Characteristics of Therapeutic Exercises

	Walking Forelimb	Walking Hindlimb	Trotting Forelimb	Trotting Hindlimb	Wheelbarrowing Forelimb	Dancing Forward Hindlimb
Peak vertical force	54%	40%	111%	66%	91%	76%
Vertical impulse	21%	15.2%	20%	12%	17%	16%

Values are a percent of body weight.

Table 24-2 Kinematics of Walking and Trotting in Dogs

Joint	Position	Walk[53]	Walk[14,19]	Trot[54]	Trot[17,18]
Shoulder					
	Extension	148	146	138	137
	Flexion	120	121	119	107
	ROM	28	25	19	30
Elbow					
	Extension	144	143	140	140
	Flexion	94	97	94	88
	ROM	50	46	46	52
Carpus					
	Extension	192	65	199	80
	Flexion	99	–20	84	–20
	ROM	93	85	115	100
Hip					
	Extension	136, 140	132	142	132
	Flexion	103, 103	100	105	100
	ROM	33, 36	32	37	32
Stifle					
	Extension	154, 155	146	152	150
	Flexion	106, 109	111	88	97
	ROM	48, 51	35	63	53
Tarsus					
	Extension	162, 165	158	163	155
	Flexion	125, 123	128	97	120
	ROM	37, 42	30	67	35

ROM, Range of motion.

Trotting

Trotting is frequently used to increase the speed of muscle contraction and the forces placed on the limb, with increased strength of muscle contraction. Z_{Peak} measurements of the forelimbs and hindlimbs at the trot are approximately 115% and 66% of body weight, respectively (see Table 24-1). Increasing the speed to a trot results in little change in shoulder joint motion, increases elbow joint excursion slightly, and increases carpal joint motion approximately 15 to 20 degrees (see Table 24-2).[17] At a trot, hip and hock motion is similar to that at the walk, but motion in the stifle increases to approximately 55 degrees.

Treadmill Walking

Walking on a ground treadmill is a commonly performed exercise for conditioning and to encourage limb use after surgery. One study determined that dogs have an increased stance time and greater stride length when walking on a ground treadmill as compared with walking over ground, but there were no differences in swing time.[48] Furthermore, maximum extension, flexion, and ROM angles are similar for the forelimb and hindlimb joints between ground and treadmill walking (Table 24-3). Maximum joint flexion velocity tended to be lower in dogs walking on ground treadmills, perhaps because of the active assisted nature of

Table 24-3 Joint Motion during Selected Therapeutic Exercises

Joint		Walking	Walking Ground Treadmill	10% Incline Ground Treadmill	Wheelbarrowing	Dancing Forward	Dancing Backward
Shoulder	Ext	148	146†	—	162*	—	—
	Flex	120*	121		131†		
	ROM	28	25†		31*		
Elbow	Ext	144	146*	—	130†	—	—
	Flex	94†	93		80*		
	ROM	50†	53*		50†		
Carpus	Ext	192†	194	—	198*	—	—
	Flex	99*	100		112†		
	ROM	93	94*		86†		
Hip	Ext	136†	138	141	—	140	164*
	Flex	103*	107	103*		120	135†
	ROM	33	31	38*		20†	29
Stifle	Ext	154	156*	153	—	155	129†
	Flex	106	111†	109		110	88*
	ROM	48*	45	44		45	41†
Hock	Ext	162	163	166*	—	163	157†
	Flex	125	128	125		134†	115*
	ROM	37	35	41*		29†	37

Ext, Extension, *Flex,* flexion, *ROM,* range of motion in degrees.
Values represent mean flexion and extension angles, respectively.
For each joint, the most joint extension, flexion, and ROM (*) or least joint extension, flexion, and extension (†) are indicated.

walking on a treadmill. Therefore if increased weightbearing on an affected limb with similar joint angle excursions but less rapid joint motion is desired, walking on a ground treadmill should be considered.

Walking on a treadmill with a 10% incline results in joint motion similar to walking on a level treadmill, probably because dogs can adjust their stride length while propelling the body up an incline to achieve similar joint motion to that obtained with walking over level surfaces.[49] The mean maximum hip extension and total hip ROM were 3 and 7 degrees greater with incline treadmill walking, whereas mean stifle extension was 3 degrees less with incline treadmill walking.

Another study evaluated dogs walking on a treadmill at inclines of 5%, 0%, and –5%.[50] Stance or swing phase durations were not affected by treadmill inclination. When the inclination increased from –5% to 5%, hip joint ROM increased and stifle extension decreased significantly (Table 24-4).

Table 24-4 Joint Motion while Walking on a Treadmill with an Incline at 5%, 0%, or –5%[50]

Joint	Joint Position	Decline	Level	Incline
Hip	Extension	120.9	120.8	121.8
	Flexion	95.8	95	94.5
	ROM	25.1	25.9	27.3
Stifle	Extension	146.2	144.9	143.1
	Flexion	93.7	93.4	91.6
	ROM	52.6	51.5	51.5

ROM, Range of motion.

Incline Ramp Walking

In general, walking up an inclined ramp results in greater ROM of the forelimb joints as compared with ascending stairs or trotting on a level surface.[51] Shoulder extension, ROM, and carpal flexion are greater when walking up a ramp as compared with ascending stairs or trotting over level ground (Table 24-5). Shoulder ROM while ascending the ramp was 66 degrees, compared with 20 degrees while ascending stairs. Elbow flexion and extension are greater when walking up a ramp as compared with trotting over level ground. In another study, incline walking on an 11% slope resulted in increased hip flexion and decreased stifle joint flexion.[52]

Decline Ramp Walking

Walking down a ramp results in less femorotibial flexion and tibiotarsal flexion and extension compared with walking down stairs (see Table 24-5).[53] ROM of the hip, stifle, and hock joints are also less with decline ramp walking as compared with descending stairs. Decline

Table 24-5 Kinematics of Stair and Ramp Ascent and Descent

Joint	Joint Position	Trotting	Stair Ascent	Ramp Ascent	Stair Descent	Ramp Descent
Shoulder	Extension	138	126	152		
	Flexion	119	106	85		
	ROM	19	20	66		
Elbow	Extension	140	149	157		
	Flexion	94	57	46		
	ROM	46	92	111		
Carpus	Extension	199	189	187		
	Flexion	84	68	36		
	ROM	115	121	151		
Hip	Extension	142	147		123	120
	Flexion	105	102		96	97
	ROM	37	45		27	23
Stifle	Extension	152	145		158	157
	Flexion	88	61		62	77
	ROM	63	83		96	80
Tarsus	Extension	163	171		159	152
	Flexion	97	61		61	77
	ROM	67	108		98	76

ROM, range of motion.
Hindlimb trotting and stair ascent from Durant et al.[54]
Stair and ramp decline from Millard et al.[53]
Forelimb trotting, stairs and ramp from Carr et al.[51]

walking at a slope of 11% resulted in decreased hip flexion in another study as compared with overground walking.[52] Therefore ramp descent may be easier than stair descent for dogs with musculoskeletal disease of the pelvic limb and limited motion of pelvic limb joints.

Stair Ascent

ROM of the pelvic limb joints is significantly increased during stair climbing compared with trotting on level ground, with increases of 10, 20, and 40 degrees for the hip, stifle, and hock joints, respectively (see Table 24-5).[54] Extension of the hip and hock joints is increased with stair climbing (approximately 5 and 8 degrees, respectively), whereas stifle extension is decreased by 7 degrees. Flexion is also significantly increased in the stifle and hock by 26 degrees and 35 degrees, respectively.[54] Hip flexion does not contribute to stair ascent, but the stifle and hock joints contribute significantly to raising the limb up to the height of the step.

Another study also found that tarsal flexion, extension, and ROM; stifle flexion and ROM; and hip extension and ROM were greater when ascending stairs as compared with walking on level ground.[55] However, they found that stifle extension increased with stair climbing. The difference may be due to different stair parameters and a convention of joint angle measurement used in people.

Regarding the forelimb joints, there are some differences between stair ascent and trotting.[51] These differences are due to changes in extension and flexion of forelimb joints. Shoulder extension is less with stair climbing as compared with trotting over ground, whereas flexion is greater with stair climbing. Because of these opposite effects, there is no difference in total shoulder ROM. Elbow extension, flexion, and ROM are greater with stair ascent as compared with trotting over ground, and carpal flexion and ROM are greater with stair climbing. Ramp ascent, however gives, greater extension, flexion, and ROM of all forelimb joints as compared with stair ascent.

Stair Descent

Stair descent results in significantly greater stifle flexion and hock flexion and extension compared with walking down a declined surface (see Table 24-5).[53] Significantly greater ROM was also achieved in the coxofemoral, femorotibial, and tibiotarsal joints during stair descent. Another study, using different stair parameters and a convention of joint angle measurement used in people, agreed that tarsal flexion, extension and ROM, stifle flexion and ROM, and hip flexion were greater when descending stairs as compared with walking on level ground.[55]

Wheelbarrowing

Raising the rear limbs off the ground and walking the dog forward is a therapeutic exercise known as *wheelbarrowing* (Figure 24-23). The main objective is to increase the use of the forelimbs and increase weight bearing on them.



Figure 24-23 Wheelbarrowing exercise is performed by supporting the rear limbs of the dog and walking forward on the forelimbs.



Figure 24-24 Dancing exercise is performed by supporting the front limbs of the dog and moving the dog in a forward or backward direction. A muzzle is recommended if the dog is painful or resists the exercise in any way.

However, several specific events occur during wheelbarrowing. The forelimb is a complex structure, with multiple joints continuously undergoing joint angular acceleration and deceleration, with the limb held at changing angles to the ground during the stance phase. During wheelbarrowing, the dog must move its forelimbs to keep from falling and in doing so, the stride length changes as the dog attempts to keep its balance. In one study of hound-type dogs, wheelbarrowing resulted in significantly shorter stride length (0.66 vs. 0.86 m), stance time (0.27 vs. 0.37 s), and swing time (0.22 vs. 0.27 s) as compared with walking at the same speed.[1] Dogs had a mean Z_{Peak} of 91% of body weight, which is intermediate between that expected of walking (approximately 55%) and trotting (approximately 100% to 110%) (see Table 24-1). Mean VI was 17% of body weight, which is similar to walking (approximately 23% of body weight), but greater than trotting (approximately 13% of body weight) because of the shorter stance time while trotting. So although some of the dog's body weight is shifted to the forelimbs, the forces placed on the forelimbs are only intermediate between walking and trotting, likely because of the shorter stride length and stance time. In addition, some of the weight is also transferred to the handler.

Regarding joint kinematics, dogs that wheelbarrowed had significantly more shoulder extension, elbow flexion, and carpal extension, and less shoulder flexion, elbow extension, and carpal flexion as compared with walking (see Table 24-3).[1] These differences are approximately 10 to 15 degrees.

Dancing

Raising the forelimbs off the ground and walking the dog forward and backward is known as *dancing* (Figure 24-24). The main goal is to increase weight bearing on the rear limbs. A comparison of Z_{Peak} measurements and VI of nine hound-type dogs that danced forward and were trotted across a force plate indicated that dancing resulted in greater Z_{Peak} measurements than trotting (75.6% vs. 65.4% body weight) and greater VI (16.1% vs. 12%) (see Table 24-1).[1]

Dancing forward may result in different kinematic characteristics compared with dancing backward or walking over ground. There was significantly less hip flexion, total hip ROM, hock flexion, and hock total ROM with forward dancing compared with walking (see Table 24-3).[1] The stride length (0.56 vs. 0.84 m, respectively) and swing time (0.18 vs. 0.26 s, respectively) were shorter with forward dancing as compared with walking, but the stance times were similar (0.32 s). Dancing backward also differed from walking over ground. Hip extension and stifle flexion were greater with backward dancing as compared with walking, whereas hip flexion, hip ROM, and stifle extension and stifle ROM were less (see Table 24-3). This information may be useful in rehabilitating dogs with various conditions. For example, in the early phases of a therapeutic exercise program for a dog with hip dysplasia that is painful with hip extension, gluteal muscle strengthening may be more comfortable by dancing the dog forward rather than backward.

Cavaletti Rail Walking

Walking over cavaletti rails results in significant increases in flexion of various joints, and the increase is related to the height of the cavaletti rails (Table 24-6).[56] Specifically, the elbow, stifle, and hock have increased joint flexion and

Table 24-6 Joint Motion while Walking over a Cavaletti Rail

		Walking	Low (at Carpus)	Medium	High (mid antebrachium)
Shoulder	Extension	149	152	153	153
	Flexion	115	125	124	124
	ROM	34	26	29	29
Elbow	Extension	147	145	142	143
	Flexion	89	72	60	59
	ROM	58	72	82	86
Carpus	Extension	182	187	187	187
	Flexion	81	86	79	81
	ROM	101	101	108	106
Hip	Extension	140	138	137	138
	Flexion	103	100	97	95
	ROM	36	38	40	43
Stifle	Extension	155	154	153	154
	Flexion	104	83	71	68
	ROM	51	71	82	86
Tarsus	Extension	165	153	154	155
	Flexion	123	84	71	68
	ROM	42	69	84	87

ROM, Range of motion.

subsequent increases in ROM while walking over cavaletti rails as compared with overground walking. There is a mild increase in hip flexion. There are no differences in joint extension, however. The shoulder surprisingly had no changes. The biceps brachii muscle is a flexor of the elbow and extensor of the shoulder, and its dual role may result in no net increased movement of the shoulder. In another study, walking over cavaletti rails at the level of the carpus resulted in increased elbow, carpal, stifle, and tarsal flexion, and increased carpal and stifle joint extension as compared with walking.[52] Stifle and tarsal ROM were also greater walking over cavaletti rails as compared with overground walking.

Sit-to-Stand Exercise

Compared with walking, sit-to-stand exercise results in greater flexion of the tarsus and nearly twice as much flexion of the stifle and hip, while extension of these joints is less[57] (Table 24-7). Others have also found that tarsal and stifle flexion and ROM are increased with sit-to-stand exercise, whereas extension of these joints is less with sit to stand as compared with walking[58] (see Table 24-7). Although the values for hip flexion and extension differed between the two studies, the ROM was similar regarding sit-to-stand and walking exercise.

Jumping

Jumping over obstacles results in significant increases in forces placed on the limbs during landing. Hound-type

Table 24-7 Joint Motion during Walking or Sit-to-Stand Exercise

Joint		Walk	Sit to Stand
Shoulder	Extension	125	119
	Flexion	88	91
	ROM	37	27
Elbow	Extension	146	147
	Flexion	91	109
	ROM	55	37
Carpus	Extension	239	202
	Flexion	128	133
	ROM	111	70
Hip	Extension	147, 140	115, 135
	Flexion	111, 103	49, 104
	ROM	36, 36	66, 31
Stifle	Extension	146, 155	108, 133
	Flexion	111, 104	46, 46
	ROM	35, 51	62, 87
Tarsus	Extension	145, 165	131, 131
	Flexion	111, 123	95, 38
	ROM	34, 42	36, 94

ROM, Range of motion.

Data from Feeney LC, Lin C-F, Marcellin-Little DJ, Tate AR, Queen RM, Yu B: Validation of two-dimensional kinematic analysis of walk and sit-to-stand motions in dogs. Am J Vet Res 2007;68:277-282; and McEachern GL, Millis DL, Headrick J, Hicks DA. Kinematic assessment of sit-to-stand exercise in dogs with chronic extracapsular cranial cruciate repair. Proc 2nd World Veterinary Orthopedic Congress/Veterinary Orthopedic Society, Keystone CO, Feb, 2006.

Table 24-8 Kinetics of Jumping

	Peak Vertical Force	Vertical Impulse	Peak Braking Force	Braking Impulse	Peak Propulsion Force	Propulsion Impulse
HIGH JUMP						
First limb strike	185.8	17.4	–26.0	–0.054	20.9	1.5
Second limb strike	168.1	22.6	–40.2	–2.8	12.2	0.7
LONG JUMP						
First limb strike	192.2	17.3	–29.4	–0.85	17.0	1.0
Second limb strike	155.1	20.5	–41.4	–3.1	9.8	0.5

dogs were jumped across a high jump (61 cm or 24 inches) and a long jump (two 38-cm or 15-inch tall hurdles placed 61 cm or 24 inches apart). Z_{Peak} was significantly higher for the first limb strike than the second limb strike in both high jump and long jump (Table 24-8). VI was lower for the first limb than the second limb for the high jump and long jump because of reduced stance time of the first limb strike. There were no differences in Z_{Peak} or VI between high and long jumping on the first limb strike. However, Z_{Peak} and VI were greater for the high jump than long jump when comparing the second limb strike.

There were also significant differences between braking and propulsion force and impulse for both first and second strike for the high jump when compared with the long jump. There was less peak braking force and braking impulse of the first limb strike as compared with the second limb strike for both high and long jumping. There were no differences in peak braking forces between high and long jumping in either the first or second limb strikes. Braking impulse was greater for the first limb strike with long jumping compared with high jumping, but not with the second limb strike. In addition, there was greater peak propulsion and propulsion impulse in the first limb strike as compared with the second limb strike for high jumping and long jumping. There was greater peak propulsion force and propulsion impulse with the first limb strike for high jumping versus long jumping, and for the second limb strike.

Dogs apparently strike harder with the first limb strike for both high and long jumping, but stay on the second limb strike longer for support, resulting in greater impulse on the second limb. There are minimal differences between high and long jumping regarding the vertical forces placed on the first limb strike, but the second limb strike is slightly greater in dogs with high jumping. Dogs apparently brake primarily with the second limb strike. High jumping results in more vertical drop, perhaps obliterating the need for more active braking because the landing is more vertical. Conversely, there is greater propulsion in the first strike as compared with the second. Dogs that undergo high jumping have greater propulsion than long jumpers for both the first and second limb strikes. This may be a result of a spring-like effect in dogs jumping more vertically, with more potential energy stored in the muscles and soft tissues. As they prepare to move forward, this potential energy may be suddenly released, resulting in greater propulsion. With information about GRFs, trainers may be able to adjust jumping techniques to minimize injury to sporting dogs. More work is needed to study differences in jumping styles and possible predisposition to injury.

The land-based exercises that result in the greatest and least flexion, extension, and ROM are indicated in Table 24-9.

Aquatic Biomechanics and Exercises

One of the principal advantages of exercising in water is the buoyancy that the water exerts, resulting in less force on limbs. Buoyancy is an important force in aquatic therapy because animals in water are lifted upward so that the effect of gravity is partially canceled. Therefore the need for antigravity muscle action is greatly reduced. The amount of unloading is related to the height of the water in relation to the height of the individual. One study evaluated the changes in body weight on ground compared with different water levels while standing.[59] Dogs standing in water to the level of the tarsus, stifle, and greater trochanter weighed 91%, 85%, and 38% of body weight, respectively, as compared with standing on dry ground, indicating that buoyancy can be a significant factor in reducing loads placed on limbs. This may be of particular advantage in animals with lower motor neuron conditions that have difficulty supporting their weight, or in patients with arthritic joints to help reduce the weight-bearing forces on painful joints.

Movement of a body through water is complex and involves forces generated by the individual, and counterforces to the movement by the water.[60] *Thrust* refers to the forces in the direction of velocity of movement, and may be thought of as the force that the body generates to push

Table 24-9 Exercises That Have the Greatest and Least Joint Motion

		Greatest Motion	Least Motion
Shoulder	Extension	Wheelbarrowing	Sit to stand
	Flexion	Ramp ascent	Wheelbarrowing
	ROM	Ramp ascent	Stair ascent
Elbow	Extension	Ramp ascent	Wheelbarrowing
	Flexion	Ramp ascent, stair ascent, cavaletti rail	Sit to stand
	ROM	Ramp ascent, stair ascent, cavaletti rail	Sit to stand
Carpus	Extension	All exercises similar	All exercises similar
	Flexion	Ramp ascent	Sit to stand
	ROM	Ramp ascent	Sit to stand, wheelbarrowing
Hip	Extension	Dancing backward	Sit to stand, ramp descent, ramp ascent, stair descent
	Flexion	Sit to stand	Dancing backward
	ROM	Sit to stand	Dancing forward
Stifle	Extension	Ground treadmill, stair descent, ramp descent, dancing forward	Dancing backward, sit to stand
	Flexion	Sit to stand, stair ascent, stair descent	Dancing forward, ground treadmill, inclined treadmill
	ROM	Stair ascent, stair descent, cavaletti rail	Dancing backward, walking
Tarsus	Extension	Inclined treadmill, stair ascent	Sit to stand
	Flexion	Sit to stand, stair ascent, stair descent	Dancing forward
	ROM	Sit to stand, stair ascent, stair descent	Dancing forward

ROM, Range of motion.

the water backward. This helps to accelerate the body and keep it moving forward.

Drag is a force that is opposite to the velocity of movement and opposes the motion between the body and the fluid.

$$\text{Drag Force} = (\text{Constant}) \times (\text{Area}) \times (\text{Drag Coefficient}) \times (\text{Velocity})^2$$

The constant is determined by the density of water, and area is the cross-sectional area of the body in the direction of the motion. The drag coefficient is affected by the shape of the body and the surface of the body. Of particular importance is the velocity of the body part moving through the water, which creates drag related to an exponential function of the velocity. Therefore if the velocity of the dog moving in water doubles, the amount of drag quadruples. The major resistance to moving through water is the turbulent drag, with formation of eddies and currents behind the body.

Another important source of drag exists at the surface of the water, at the water-air interface. There is high surface tension of the water here, and movement just under the surface tends to create waves. The energy loss with this motion results in an increase in forces that resist movement close to the cube of the velocity rather than the square. Therefore with movement just beneath the surface of the water, nearly eight times as much energy must be expended to double the velocity of the body.

One study determined stifle joint ROM and angular velocities during swimming and walking in healthy dogs, and in dogs that had undergone surgical treatment of a ruptured CCL.[61] For dogs in both groups, swimming resulted in significantly greater overall ROM of the stifle joint than did walking, primarily because of greater joint flexion. However, dogs had significantly less stifle extension while swimming compared with walking.

Two-dimensional kinematic analysis of gait has been used to evaluate joint motion in dogs walking on ground and underwater treadmills. In addition, the effect of different water levels on active joint ROM was evaluated when patients walked underwater.[62] Hip, stifle, hock, shoulder, and elbow flexion are greater in dogs walking in water compared with walking on land (Table 31-3). Joint flexion is generally greatest with water levels at or higher than the joint of interest. Maximum joint extension is similar for underwater and ground treadmill walking. Maximum and minimum joint flexion and extension are related to the level of water. These effects are likely a result of the sudden action of a limb to break the surface tension of the water at different water heights.

Summary

Technology will continue to advance, broadening the options for physical rehabilitation. Understanding how these technologies are applied depends on an

understanding of the fundamental concepts of the physics of motion and the transfer of energy. The musculoskeletal system is a machine, based on applied force and motion, with the transfer of energy resulting in work and power. These concepts can be used to predict and evaluate performance of the body and to evaluate the effectiveness and outcomes of therapies.

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25 Range-of-Motion and Stretching Exercises

Darryl L. Millis and David Levine

Range of motion (ROM) and stretching exercises are very important to achieve improved motion of joints after acute injury or surgery, or in patients afflicted with chronic conditions. They are also important to help increase flexibility, prevent adhesions between soft tissues and bones, remodel periarticular fibrosis, and improve muscle and other soft tissue extensibility to help prevent further injury to joints, muscles, tendons, and ligaments.

As with many types of rehabilitation activities, it may be beneficial to administer analgesic medication, such as a nonsteroidal antiinflammatory medication, 30 to 60 minutes before beginning any rehabilitation exercises to allow peak absorption of the medication and take advantage of its maximal analgesic effect.

Range of Motion

The full motion that a joint may be moved through is termed the *ROM*. The structure of the joint and the volume, integrity, character, and flexibility of the soft tissues that surround the joint affect joint motion.[1] ROM is commonly measured with a goniometer, and each joint has characteristic angles, such as flexion and extension of the stifle, and flexion, extension, abduction, adduction, and internal and external rotation of the hip (Appendix 3).

Muscles also have a ROM, which is termed the *functional excursion of muscles.*[1] This is the distance that a muscle is able to shorten after it has been maximally elongated. The functional excursion may be affected by the joint it crosses, particularly if joint motion is restricted. Some muscles, such as the biceps brachii and the rectus femoris muscles, cross two joints. In general, muscles that cross multiple joints may lose functional excursion easier than those that cross only one joint.

ROM exercises are useful to diminish the effects of disuse and immobilization.[2] To maintain ROM, the joints and muscles must be periodically moved through their available ranges. Movement may be passive, active assisted, or active. In each situation, a load is produced on the soft tissues that helps to maintain the articular cartilage, muscles, ligaments, and tendons in a healthy state. It is generally appropriate to initiate passive ROM as soon after injury or surgery as possible provided there are no contraindications. Progression through active assisted and active ROM follows, and when appropriate, some resistance to motion may be introduced for strengthening.

Passive Range of Motion

Passive ROM is motion of a joint that is performed without muscle contraction within the available ROM, using an external force to move the joint. In animals, passive ROM is performed by the therapist. Additional force applied at the end of the available ROM is defined as stretching. Passive ROM and stretching can be performed in conjunction with each other to help maintain and improve joint ROM.

The benefits of continuous passive ROM immediately after joint surgery were initially described by Salter and colleagues[3] and include decreased pain and improved rate of recovery. The scar tissue deposited after an injury or surgery is laid down in a random fashion, and ROM helps this scar tissue to align along the lines of stress that the tissue normally undergoes. This results in a stronger scar and may help prevent future injury. However, it is critical that the therapist maintain a ROM that is comfortable to the patient and not injure tissues by exceeding their limits.

Tissues limiting passive ROM may be normal or pathologic and include the joint capsule, periarticular soft tissues, muscles, ligaments, tendons, skin, and scar tissue. For example, open wounds over joints that are allowed to heal secondarily by contraction and epithelialization may result in limitations to passive ROM. Surgical incisions may result in adhesions and fibrosis between skin, subcutaneous tissues, fascia, muscles, and bone, limiting the ability of tissues to glide over one another. Musculotendinous tissue may also be relatively shortened as a result of spasm or contracture. Any restriction of motion may result in resistance to joint movement and pain.

Passive ROM is used whenever a patient is unable to move joints on its own, or if active motion across a joint may be deleterious to the patient, such as with a tenuous articular fracture repair. Passive ROM is sometimes used to help relax an anxious patient. The most common indications for passive ROM exercises are immediately after surgery (before active weight bearing) to help prevent joint

contracture and soft tissue adaptive shortening, maintain mobility between soft tissue layers, reduce pain, enhance blood and lymphatic flow, and improve synovial fluid production and diffusion.[2]

Another indication for passive ROM is the prevention of joint contracture during healing and recovery in paralyzed patients. Ideally, exercises should be performed two to six times per day to help maintain normal joint mobility. However, if the patient does not regain functional neuromuscular control (active motion), it is likely that joints will undergo some degree of contracture despite aggressive efforts. Although passive ROM may help slow muscle atrophy, it will not prevent muscle atrophy, increase strength, improve endurance, or be as effective in improving vascular and lymphatic flow as active ROM techniques.

Proper technique for performing passive ROM is important. The patient should be relaxed and comfortable. If active muscle contraction is not desired, it is especially important to be gentle and not create pain or discomfort. The bones proximal and distal to the joint should be supported to avoid excessive varus and valgus stresses on the joint. The therapist should gently hold the limb and avoid painful areas, such as incisions and wounds. The location of the therapist's hands on the limb is also important; the closer the hands are to the joint, the less torque will be produced at the joint. This may decrease pain and the risk of patient injury. The motion should be smooth, slow, and steady with motion generally occurring by moving the distal limb with the proximal limb held steady. The patient should be continually monitored for any discomfort, and the technique altered if necessary to enhance comfort.

Active Assisted Range of Motion

Active assisted ROM occurs when the therapist guides joint motion and the patient's muscle activity assists joint motion to some degree. In animal patients, the amount of muscle activity provided by the patient is difficult to control. In reality, because it is difficult to avoid muscle activation in patients that are not paralyzed, most ROM exercises in small animal patients involve a degree of active assisted ROM. In anxious patients or in those with upper motor neuron conditions, muscle contraction of antagonist muscle groups may oppose joint ROM exercises.

Active assisted ROM exercises are most useful for those patients that are weak or recovering from lower motor neuron conditions. In addition to assisted ROM with the patient in lateral recumbency, exercises may be performed with the patient supported by a sling, with the therapist assisting limb movement and joint ROM during ambulation. This may be performed while the patient is slowly walked over ground, on a ground treadmill, or on an underwater treadmill. Another form of active assisted ROM exercise is performed while swimming. Patients with limited ability to ambulate on the ground may benefit from controlled swimming, during which the therapist assists movement of the animal's limbs and joints while in the water with the patient. The buoyancy of the water helps to support the weight of the limbs while the therapist concentrates on assisting the limb through a normal cycle of movement.

Active assisted ROM helps to combat the negative effects of immobilization on limbs, similar to those achieved with passive ROM. Some degree of active muscle contraction allows muscle strengthening and strengthening of bone at muscle origin and insertion sites. In addition, some neuromuscular reeducation, and proprioceptive and gait training may be achieved.

Active Range of Motion

Active ROM is the motion of a joint that may be achieved by active muscle contraction. In addition to increasing strength, coordination between muscle groups is necessary because the guidance of assisting the patient through ROM is no longer provided. The active ROM may be performed during a regular gait cycle, in which the excursion of joint motion is relatively limited, or under special conditions designed to expand motion and encourage more complete use of the full available ROM. Examples of these activities include swimming; walking in water, tall grass, snow, or sand; climbing stairs; crawling through a tunnel; and negotiating cavaletti rails.

As the patient improves ROM of a joint, it is helpful to continue to perform passive ROM and stretching to achieve as complete ROM as possible, and then perform active ROM through this increased motion to emphasize more complete use of the limb. Greater strength is required for patients to perform active ROM, and some of the special conditions require more muscle strength than normal ambulation during walking or trotting. Therefore for those exercises, a transition between active assisted and active ROM may be necessary. Active ROM exercises may be a prelude to other strengthening activities. Owners may also be involved with active ROM exercises in helping with a home care program for their pet.

Precautions and Contraindications to Range of Motion

All forms of ROM exercises are contraindicated when motion may result in further injury or instability. Such examples include unstable fractures near joints and unstable ligament or tendon injuries. Communication between the surgeon and therapist is critical to be certain that appropriate ROM exercises are performed and that the limits of the range are not exceeded, which may result in damage to the tissues. In most cases, early passive ROM is felt to be beneficial if the therapist stays within a ROM that is reasonable for the patient and condition being treated. In

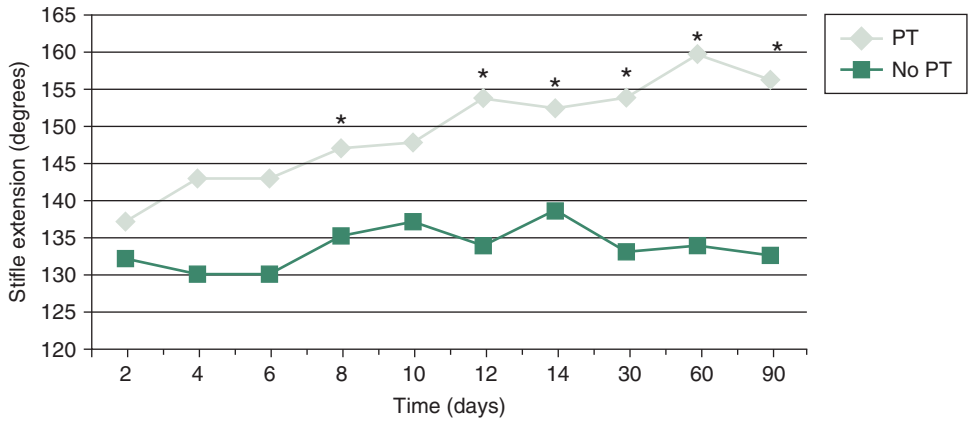


Figure 25-1 Effect of physical rehabilitation on stifle extension. Note the return of normal stifle extension by 2 weeks after surgery, in those dogs receiving rehabilitation and the loss of complete stifle extension in some dogs not receiving postsurgical rehabilitation. $*P < 0.05$.

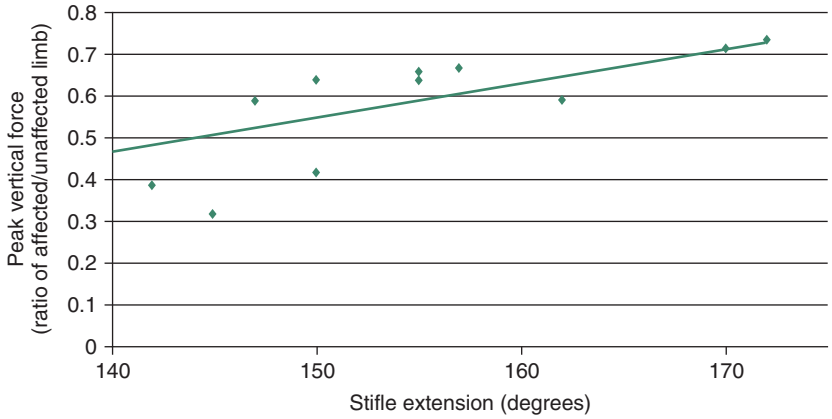


Figure 25-2 Correlation between stifle extension and peak vertical force. Relationship of comfortable angle of stifle extension to peak vertical force in dogs 10 weeks after cranial cruciate ligament transection and stifle stabilization. Dogs with normal stifle extension have greater weight bearing on the limb at a trot than those with limitation to stifle extension. Although a cause-and-effect relationship cannot be definitively identified from these data, there is a significant relationship. $r = 0.78$; $P < 0.001$.

addition, the therapist should perform the exercises at a reasonable speed that is not painful for the patient.

Range of Motion Studies

In some canine conditions, active ROM is a prerequisite to a successful outcome. For example, it is critical that puppies with distal femoral physeal fractures have early passive and active ROM exercises to avoid fracture disease and tiedown of the quadriceps muscle group.[4] Similarly, dogs with condylar fractures of the distal humerus must begin active ROM to prevent permanent joint stiffness.

In one study of dogs following surgery for cranial cruciate ligament rupture, patients not receiving early passive and active ROM exercises had reduced stifle extension (Figure 25-1).[5] This loss of motion appeared to be permanent in some dogs if extension was not achieved within 2 weeks. In contrast, those dogs receiving appropriate rehabilitation had return of near-normal stifle extension. There also appears to be an association of stifle extension with weight bearing, with improved weight bearing in dogs with near-normal stifle extension (Figure 25-2). However, the cause and effect of this relationship are unknown.

The association of stifle ROM with function following surgery for cranial cruciate ligament disease in clinical patients has been further evaluated.[6] In 412 dogs treated with tibial plateau leveling osteotomy (TPLO) for treatment of cranial cruciate ligament rupture, dogs were determined to have no loss of extension or flexion (n = 322), less than 10 degrees loss of extension or flexion (n = 78), or 10 degrees or more loss of extension or flexion (n = 12). Loss of extension or flexion greater than 10 degrees was associated with significantly greater lameness in comparison with no loss, or loss of extension or flexion less than 10 degrees. In addition, osteoarthritis of the stifle joint was significantly correlated with loss of extension, and loss of extension greater than 10 degrees was less tolerable and less amenable to physical rehabilitation than flexion loss. The authors concluded that loss of extension or flexion should be assessed in dogs with persistent lameness after TPLO so that early intervention can occur.

An explanation for the restricted ROM in clinical cases may include physical restrictions, such as joint capsule fibrosis, soft tissue approximation, or bony impingement that may occur with osteophytes as a result of joint disease.

Another possible explanation is an increase in intraarticular pressure with joint motion, resulting in stretch of the joint capsule and creation of pain. One study evaluated the intraarticular pressure and volume relationships in the knee joint of dogs.[7] In general, infusion of fluid into the joint did not result in intraarticular pressure changes between 125-110 degrees of stifle motion. However, increasing flexion from 110 to 50 degrees resulted in an increase in pressure, which was greater with increased intraarticular volume. Increasing the intraarticular volume also resulted in decreased total ROM of the joint.

The functional and structural consequences of remobilization of the shoulder joint after 12 weeks of immobilization were studied in 10 beagle dogs.[8] It was found that after 12 weeks of immobilization, the passive ROM was markedly impaired, intraarticular pressure increased during movement, and the filling volume of the joint cavity was reduced. On histologic examination, the capsule showed hyperplasia of the synovial lining and vascular proliferation in the wall, but there was no increase of collagen in the capsular wall. Both the functional and structural changes were unaltered after 4 weeks of remobilization, but after 8 weeks they began to reverse, and they returned to normal after 12 weeks, indicating that functional and structural changes after immobilization of an uninjured shoulder joint are potentially reversible. The timing and degree of improvement may not be similar in patients with pathologic injury to the shoulder.

Another study of dogs evaluated the effects of joint mobilization treatment on carpal joints of dogs.[9] The right carpal joints of 12 dogs were immobilized for 6 weeks, which resulted in joint hypomobility. The treated group received mobilization therapy daily for 4 weeks following the immobilization period. In control and treated groups, passive ROM, peak angles of extension and flexion of the carpal joint, and the amount of time required in the gait cycle to reach these peak points were evaluated cinematographically during gait before immobilization and once weekly for 4 weeks after immobilization. The treated group had improved passive ROM and motion during gait.

Continuous passive ROM has been assessed in several studies to evaluate its use in the management of cartilage defects. Salter and colleagues[10] demonstrated that young rabbits with a full-thickness defect in the articular cartilage managed with continuous passive motion had healing of 52% of the defects with hyaline-type cartilage. Similar results were obtained in adult rabbits, in which 44% of the defects were repaired with hyaline cartilage. Passive motion may also help to reduce cartilage destruction following infection of a joint.[11,12] The optimal daily dose of passive ROM and the total duration of treatment are unknown. However, one study of rabbits found that at least 8 hours of passive motion per day was necessary to achieve the level of healing obtained by Salter.[13] The effect of active motion on cartilage healing is unknown in animals because of the difficulty in achieving consistent limb use after cartilage injury.

Continuous passive motion has also been recommended to prevent contracture of periarticular tissues and to restore joint function. Immobilization results in shortening of fibrous tissues and loss of motion, depending on the position of immobilization. There is a loss of collagen mass after immobilization, and there is collagen cross-linking of periarticular connective tissues, which may lead to increased stiffness. Passive motion may reduce these changes and prevent joint stiffness. In one study, 16 hours or more of continuous motion prevented increased joint stiffness following articular cartilage damage.[14] Longer durations of passive motion may be necessary to help maintain normal quantities and turnover of collagen and glycosaminoglycans (GAGs), as well as to help maintain the normal alignment of collagen fibers and prevent shortening and cross-linking.

It has been suggested that continuous passive ROM may be somewhat antiinflammatory in patients with inflammatory joint disease. In one study, the effects of continuous passive ROM on early inflammatory events were evaluated in meniscal fibrocartilage of rabbits with antigen-induced arthritis.[15] Rabbit knees injected with bovine serum albumin and Freund's complete adjuvant were treated with 24 or 48 hours of continuous passive ROM or were immobilized. Within 24 hours, immobilized knees had marked degradation of GAGs in meniscal fibrocartilage. Inflammatory mediators, including matrix metalloproteinase 1, cyclooxygenase 2, and interleukin (IL) 1b, increased within 24 hours and continued to increase. Knees undergoing continuous passive ROM had reduced GAG degradation and inflammatory mediators during treatment, and increased synthesis of the antiinflammatory cytokine IL-10. These results suggest that continuous passive ROM suppresses the deleterious effects of inflammatory arthritis more than immobilization and provide information regarding the involved molecular events.

The duration of daily passive motion treatment has varying effects. For example, relatively short daily passive motion may reduce stiffness immediately after treatment, but there may be greater long-term stiffness.[14] In fact, shorter durations of passive motion may be detrimental.[14,16] One study indicated more stiffness with 10 to 30 minutes of passive motion per day than if the joints were immobilized,[14] whereas another indicated increased stiffness with less than 8 hours of daily passive motion.[16] Although somewhat surprising, these results suggest that there may be increased trauma associated with breakdown of soft adhesions that form when motion is not occurring, and that very frequent motion is necessary to keep the tissues mobile.

Both continuous motion and as little as 4 hours of daily passive motion help to preserve muscle mass in rabbits.[14,17]

In fact, 5% to 15% greater limb muscle mass may occur in rabbits with passive motion as compared with those that are immobilized.

The effects of passive motion on bone loss are less clear. It is well documented that immobilization results in a decrease in bone mineral content; however, 16 or 24 hours of passive motion resulted in the loss of greater bone mineral than immobilization.[14] It was suggested by these authors that the increased joint stiffness that resulted from less than 16 hours of motion may have increased the bending stresses and load on the bones, whereas greater duration of passive motion maintained normal joint stiffness and prevented the additional loading on the bones.

In people, the use of active and passive knee motion in the immediate postoperative period and a treatment plan for early postoperative limitations in knee motion has proven highly effective in restoring motion after anterior cruciate ligament reconstruction.[18] In one study of 207 knees, 91% regained full ROM. The remaining patients did not regain motion as rapidly and were placed in an early postoperative phased treatment program of serial extension casts, early gentle manipulation under anesthesia, or arthroscopic treatment of intraarticular adhesions and scar tissue. Most of these patients regained full ROM. Those patients who failed to follow the rehabilitation program had permanent and significant limitation of motion. The incidence of postoperative motion problems was related to the extent of the surgical procedure. Patients who had more extensive surgery, such as a concurrent meniscus repair or a medial collateral ligament repair, had a higher incidence of restricted motion.

Clinical Application of Range of Motion

Passive Range of Motion

Passive ROM treatment should be administered in a quiet and comfortable area, away from distractions, such as loud noises, other pets, and other people who are not involved with the treatment. This will allow the patient to be calm, relaxed, and more receptive to the treatment.

It is recommended that patients have a muzzle applied for the initial treatments, or if they are painful, resistant to treatment, or overly anxious. The patient should be placed in lateral recumbency with the affected limb up (Figure 25-3). Help may be required to restrain the animal and to help keep it quiet and relaxed. In all forms of ROM activities, therapists should be comfortable and use proper body mechanics to avoid injury to themselves.

After the animal has relaxed, gently place both hands on the injured limb and begin a very gentle massage to help further relax the patient. Massage for 2 to 3 minutes, and then gently move one hand to the portion of the limb above the joint. Place the other hand on a portion of the limb below the affected joint. Be certain that the entire limb is supported to avoid any undue valgus, varus, or rotational stresses to the involved joint.



Figure 25-3 Before performing range-of-motion or stretching exercises, the patient should be placed in lateral recumbency with the affected limb up.

After the hands are in the correct position and the limb is supported, begin by slowly and gently flexing the treated joint (Figures 25-4 to 25-7). In general, the other joints of the limb should be allowed to remain in a neutral position (a position as if the animal were standing). Try not to move the other joints while working on the affected joint because some joints may be restricted by the position of the joints above or below the target joint. In some situations, the position of one joint may greatly affect the motion in another joint. In these cases, the other joints should be placed in a position that will allow as complete a ROM as possible to the target joint. For example, maximal hock flexion cannot be obtained while the stifle is maintained in an extended position. In this case, placing the stifle in a flexed position allows more complete flexion of the hock. Slowly continue to flex the joint until the patient shows initial signs of discomfort, such as tensing the limb, moving, turning the head toward the therapist, or trying to pull away, but do not cause undue discomfort, indicated by behaviors such as vocalizing or attempting to bite.

With the hands maintained in the same positions, slowly extend the joint. Again, try to keep the other joints in a neutral position and minimize any movement of the other joints. Slowly continue to extend the joint until the patient shows initial signs of discomfort.

Alternatively, a number of joints may be simultaneously placed through a ROM, a technique sometimes referred to as *ROM through functional patterns* or bicycling movements. This form of ROM exercise may be appropriate as an animal nears active use of a limb. Flexing and extending all of the joints of a limb in a pattern that mimics an exaggerated normal gait pattern may also be beneficial for neuromuscular reeducation.

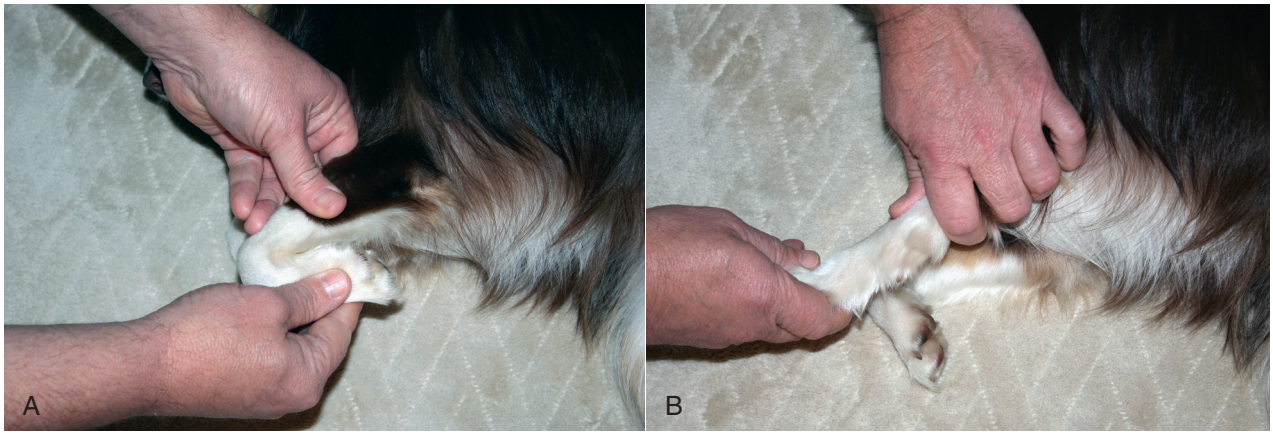


Figure 25-4 **A,** Carpal flexion. While supporting the radius and ulna in one hand and the foot in the other, the carpus is gently flexed. **B,** Carpal extension. While supporting the radius and ulna in one hand and the foot in the other, the carpus is gently extended.

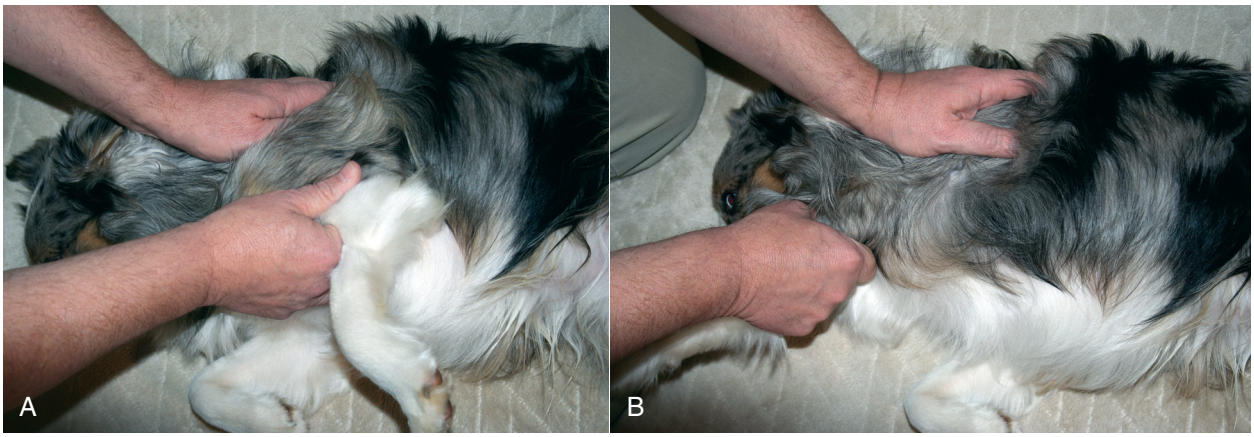


Figure 25-5 **A,** Shoulder flexion. While supporting the distal scapula in one hand and the humerus in the other, the shoulder is gently flexed. **B,** Shoulder extension. While supporting the distal scapula in one hand and the humerus in the other, the shoulder is gently extended.

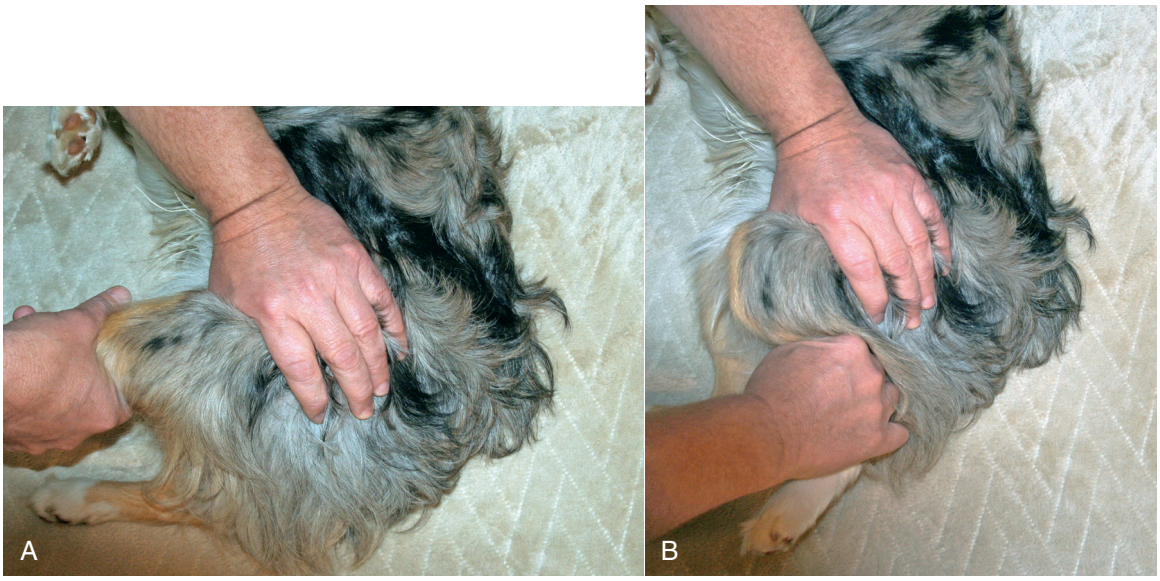


Figure 25-6 **A,** Stifle flexion. While supporting the distal femur in one hand and the tibia in the other, the stifle is gently flexed. **B,** Stifle extension. While supporting the distal femur in one hand and the tibia in the other, the stifle is gently extended.

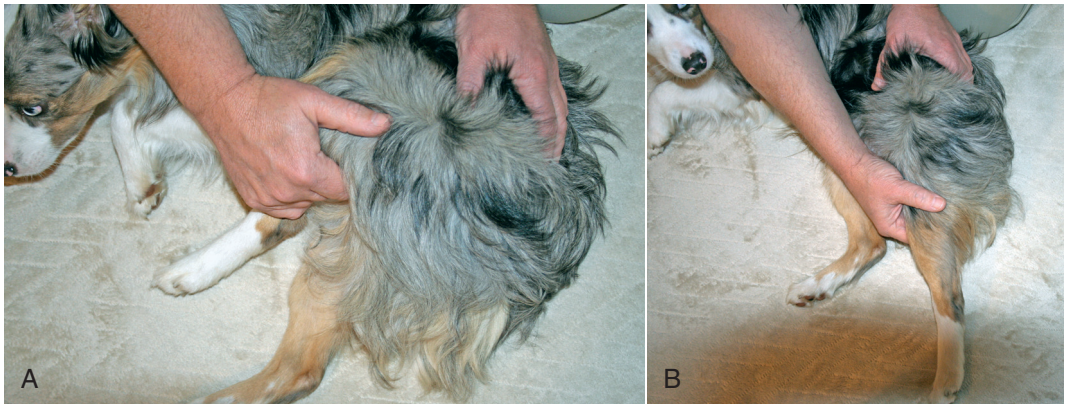


Figure 25-7 **A,** Hip flexion. While supporting the femur in one hand and the pelvis in the other, the hip is gently flexed. **B,** Hip extension. While supporting the femur in one hand and the pelvis in the other, the hip is gently extended.

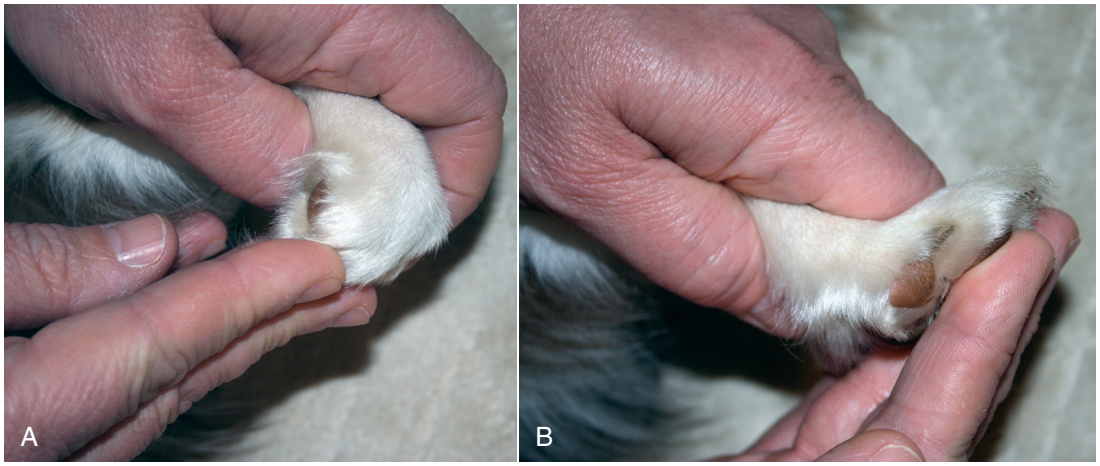


Figure 25-8 **A,** Digit flexion. While supporting the carpus in one hand and the digits in the other, the digits are gently flexed. **B,** Digit extension. While supporting the carpus in one hand and the digits in the other, the digits are gently extended.

The number of ROM repetitions and the frequency of the treatments depend on the condition treated. In general, for most routine postoperative conditions, 15 to 20 repetitions performed two to four times per day are likely adequate. As the ROM returns to normal, the frequency may be reduced. For more challenging conditions, such as articular fractures, physeal fractures in young dogs, joints with contractures, or joints that have been immobilized for a time, an increase in the number and frequency of repetitions may be necessary.

It is also important to maintain normal ROM in the other joints of the affected limb. After completing ROM of the affected joint, keep the limb in a neutral position and slowly move the hands distally to the digits (or if already working on the digits, move proximally). Be certain to keep the injured joint supported. Performing ROM of the digits requires additional consideration because it is difficult to passively flex and extend a single joint at a time because of the proximity of the other joints of each digit. In addition, it is more efficient to perform ROM in all joints simultaneously (Figure 25-8). Place the fingers or palm on the dorsal surface of the digits and slowly flex the digits until the point of initial discomfort. Then place the fingers or palm on the pads of the patient's foot and slowly extend the toes until the point of initial discomfort. After ROM is performed on the digits, ROM exercises are performed on the other joints.

The ROM session may be ended with a gentle massage to the injured limb. Gently and slowly massage for approximately 5 minutes. This helps to maintain a relaxed state. An ice pack may be applied to the injured joint after the ROM and massage.

Active Assisted Range of Motion

Active assisted ROM is the next step in the progression of joint motion during rehabilitation. In most situations, veterinary patients will have some muscle tension during ROM activities, and there is an equally likely chance that

an agonist or antagonist muscle group, or likely a combination of both, will contract with joint movement. Patients with neurologic conditions may benefit from assisted movement of limbs while in lateral recumbency, or while standing and generating a gait pattern.

Active assisted ROM may be performed while the patient is ambulating on a ground treadmill or during swimming activity, during which the therapist helps move the limb at the appropriate phase of the gait cycle. Similarly, placing the animal in an ambulatory sling and slowly moving the animal with the sling while assisting the animal to advance the limb are appropriate active assisted ROM activities. Another technique involves the use of an elastic band placed around a limb or the foot. As the animal advances the limb, tension on the elastic band helps to advance the limb.

Active Range of Motion

When the animal is able to ambulate and move the affected limb, active ROM exercises may be initiated. Dogs only use a small amount of their available joint ROM during normal walking and trotting, so the joints do not go through a complete normal ROM during these activities. If joint restriction is present, patients may benefit by performing activities that encourage a more complete ROM. Swimming or walking in water results in greater flexion of joints. Decreased joint extension may occur with swimming, but walking in water maintains relatively normal active joint extension while increasing joint flexion, resulting in greater overall joint ROM. Other activities that may be performed include walking in snow, sand, or tall grass and crawling through a play tunnel. Climbing stairs may increase joint excursion, while also increasing strength. Walking over cavaletti rails is an excellent method of achieving normal limb extension for walking, while increasing joint flexion of certain joints, especially the elbow, stifle, and tarsus as the patient negotiates the rails. In addition, the rails may be raised or lowered to encourage increased or decreased flexion of these joints, based on the needs of the individual patient.

Stretching

Stretching techniques are often performed in conjunction with ROM exercises to improve flexibility of the joints and extensibility of periarticular tissues, muscles, and tendons.[2] Conditions that result in adaptive shortening of tissues, including immobilization, reduced mobility, injury, and fibrosis of periarticular tissues, or neurologic conditions, may respond favorably to stretching.[1] Muscle weakness may occur with adaptive shortening of tissues because peak muscle tension cannot occur, and joint ROM may be decreased as a result of periarticular fibrosis.

Flexibility refers to the ability of tissues, particularly muscle, to relax and respond to an elongation force. Overstretching refers to the elongation of tissues beyond normal limits to allow greater joint ROM than normal. Overstretching may be detrimental if there is inadequate soft tissue support to maintain joint stability and prevent injury. Tightness implies mild shortening of a musculotendinous unit, with no specific pathologic cause. Two-joint muscles, such as the rectus femoris and gastrocnemius muscles, are especially susceptible to tightness. The unit may be lengthened over time with stretching exercises.

Contracture occurs when muscles or other soft tissues that span a joint shorten and limit ROM. Contractures may be defined by the types of soft tissues involved.[1] Myostatic contractures have a musculotendinous junction that has adapted to a shortened position. There is no pathologic condition, but the joint has significant loss of motion. Scar tissue adhesions between normal tissues, such as muscles and fascia, tie down the tissues and prevent normal gliding motion between the tissues, resulting in reduced ROM. Most scar tissue adhesions may be prevented with appropriate stretching, exercise, and massage in the early tissue-healing phase following injury. If the adhesion continues to mature without tissue modification, a fibrotic adhesion may form. These types of adhesions dramatically reduce joint ROM, and the resultant contracture is very difficult to treat and restore normal function to the joint. When there is an excessive amount of fibrous tissue, cartilage, or bone that forms in the area, there may be permanent loss of soft tissue extensibility. Damage to the central nervous system with upper motor neuron signs may result in hypertonic muscles and a pseudomyostatic contracture. In this situation, the muscle seems to be in a chronic state of contraction, resulting in reduced ROM. Appropriate ROM, stretching, and massage may help to improve this form of contracture.

The various tissues that are involved, such as muscle, ligaments, tendons, joint capsule, and skin, respond to stretching differently. Interestingly, in a study of rat denervated gastrocnemius muscle-tendon units, the muscle tissue accounted for 95% of the total increase in length of the unit with loading, whereas the tendon contributed only 5% to the length.[19] Therefore the therapist must consider which tissues are most likely limiting mobility and choose the appropriate techniques to help achieve more normal function. If stretching occurs within the normal physiologic limits of the tissue, elastic deformation occurs in which the tissues return to their normal resting length after the stretch is completed. If the tissue is stretched beyond its limits, permanent deformation of the tissues remains after the stretch is completed, resulting in an increased resting length; this is called *plastic deformation.*

Stretching is a general term that is used to indicate maneuvers to elongate tissues shortened as a result of a pathologic condition and to increase flexibility and joint motion in normal and abnormal tissues. Stretching differs from ROM exercises in that stretching takes tissues beyond

the normal ROM. Passive ROM only takes structures through the available range. Passive stretching techniques are most commonly used in veterinary medicine because of the inability to verbally communicate instructions to the patient to selectively relax and contract various muscle groups. It may be possible, in some situations, to apply neuromuscular electrical stimulation to various muscle groups to mimic active muscle contraction of an individual muscle for certain active stretching techniques, but this requires coordination of efforts, and usually two or three people must be involved in the treatment session.

Several stretching techniques have been described, including static stretching, prolonged mechanical passive stretching, ballistic stretching, and proprioceptive neuromuscular facilitation stretching. The acute effect of stretching is an immediate elongation of the elastic component of the musculotendinous unit. A chronic stretch may be applied with the limb immobilized with the target tissues in an elongated position. Chronic stretching over time may result in sarcomeres being added to lengthen muscle tissue at the musculotendinous junction.[20] The number of sarcomeres in series increases, resulting in an increased resting muscle length. This form of stretching is used to lengthen shortened tissue and to decrease muscle stiffness.[2] Immobilization of a muscle in a shortened position results in a decrease in sarcomeres and increased connective tissue; stretching may correct this process.

The muscle spindle monitors the velocity and degree of muscle stretch. When a muscle is stretched very quickly, the muscle spindle is activated, which stimulates the primary afferent fibers and results in increased muscle tension. This is termed the *monosynaptic stretch response.* Caution should therefore be used to avoid stretching too rapidly, which may cause stimulation of the muscle spindle and an increase in muscle contraction instead of relaxation.[1] The Golgi tendon organ senses muscle tension during a contraction and provides a protective function to the muscle by inhibiting contraction of the muscle when tension is applied slowly. When excessive muscle tension develops during contraction, the Golgi tendon organ responds, and the muscle relaxes.

Noncontractile soft tissues, such as tendons and ligaments, are composed primarily of collagen. Initial stretch applied to these tissues results in straightening of the collagen fibers with relatively little force. As the tissue is stretched to the end of a ROM, the tissue remains elastic, and release of the stretch results in return of the collagen to its normal resting position. If the stress on the tissues continues, the bonds between the collagen fibrils and fibers may be damaged, resulting in plastic deformation. Caution must be used during stretching to avoid tissue damage in most instances. Low-magnitude forces placed on the tissues over time allow collagen fibers to rearrange, perhaps resulting in a safer mode of tissue elongation. In people, 15 to 20 minutes of low-intensity sustained stretch, repeated for 5 days, has increased the length of the hamstring muscles.[21] Cyclic submaximal loading may also increase resting tissue length through tissue remodeling. Hamstring length was increased in people following a series of 10-second stretches and 8-second rest periods for 15 minutes per day, for 5 days.[21]

Clinical experience suggests that in uncomplicated cases of muscle tightness, 5 to 10 degrees of ROM may be gained per week, and 3 to 5 degrees may be gained in cases with joint capsule tightness. Other factors may alter these expected responses, including the animal's weight-bearing status, other medical conditions such as Cushing's disease, the extent of trauma to the tissues, and the type of surgical procedure performed.

There is no consensus regarding which stretching technique is most effective, and some controversy exists regarding the efficacy of various stretching techniques for certain conditions. However, if benefits are achieved, the effects of a consistent stretching program may be maintained over time, even if stretching is temporarily discontinued. A stretching program performed three to five times per week may result in measurable increases in flexibility in patients with stiffness. After flexibility improves, the frequency of stretching may be reduced. In fact, stretching too frequently may be harmful in some situations. The natural cycle of stress and adaptive remodeling may be interrupted if adequate time is not allowed for healing between intense stretching sessions. Additional caution is required in aged animals because collagen becomes less elastic, and less stretch is tolerated before plastic deformation.

Static Stretching

Static stretching involves placing the joint or joints in a position so that the muscles and connective tissues are stretched while held in a static position with the tissues at their greatest length. Stretches should be held for 15 to 30 seconds. There is probably little advantage to stretching for longer periods in healthy patients.[22] One advantage of this form of stretching is that less force is applied, reducing the possibility of iatrogenic damage to the tissues. There is also less stimulation of the IA and II spindle afferent fibers, which increase the muscle's resistance to stretch.[2] It is important to be certain that the stretch is gentle, comfortable, and well tolerated by the patient. A low-intensity stretch applied for a longer duration may be more comfortable for some patients. A slow static stretch is less likely to induce contraction in the muscle being stretched.[1] The gains in ROM are relatively transient with static stretching, and the increases are generally attributed to elastic changes in actin-myosin overlap.[1]

When performing this form of stretching, it is important to have the patient as relaxed as possible to allow the maximal stretching of the muscle and tissues with as little resistance as possible. It is important to properly support



Figure 25-9 A dog with contracture of the left carpus and digits after immobilization of the forelimb as a result of a fracture. Note the upright stance of the foot in comparison with the normal contralateral side.

the limb and perform the stretch with the joints in the proper alignment to reduce any abnormal stresses on the joints. After the stretching period, the tissues are allowed to return to a neutral position, and then the stretch is reapplied for up to 20 repetitions in a session.

Prolonged Mechanical Stretching

Prolonged mechanical stretching is similar to static stretching in that a low-intensity stretch is applied. It differs, however, in that the stretch is prolonged. The stretch, applied for a minimum of 20 minutes and up to several hours, is effective in increasing ROM in many patients.[23] In people, a low-intensity prolonged stretch applied 1 hour per day was more effective than static stretching over a 4-week period in patients with chronic knee flexion contractures.[24]

In animals, an effective manner of achieving prolonged mechanical stretching is the use of splints or other coaptation devices to provide prolonged stretch to tissues. For example, dogs with a fracture of the radius and ulna treated with a cast often have contracture of the carpus following fracture healing (Figure 25-9). Pain and reduced limb use are frequent sequelae. A dorsal fiberglass splint, which has been contoured to the normal contralateral side, may be placed on the affected limb over several layers of cotton cast padding that have been placed over the limb. Gauze bandaging material may then be used to help pull the foot up to the contoured splint with mild tension, applying controlled prolonged gentle stretch to the palmar carpal tissues. The wrap may be changed several times per day, and static stretching and ROM exercises performed. When the wrap is replaced, additional stretch may be applied by increasing the tension on the gauze bandaging material. At no time should the patient feel undue discomfort. After several days of this form of treatment, the carpus generally returns to a normal ROM and function and pain are improved.

Other devices to provide prolonged mechanical stretch in veterinary medicine include the use of a dynamic flexion apparatus to apply a flexion stretch to the stifle in patients with fracture disease and quadriceps contracture.[25,26] In some instances, it may be necessary to surgically release adhesions and scar tissue. A dynamic flexion apparatus may then be applied to help prevent recurrence of the contracture. The apparatus consists of transcutaneous pins placed in the pelvis or proximal femur, and in the tibia, with elastic bands placed between the pins to provide prolonged stretch with the stifle in flexion. In addition, the patient may also actively extend the stifle against the resistance of the elastic band, but when the quadriceps muscle relaxes, the elastic band applies stretch to the stifle in flexion.

It is believed that when muscles are maintained in a lengthened position for several weeks, sarcomeres are added.[20,27] This increase in length persists if the new length is actively used. Other connective tissues may also lengthen as a result of plastic deformation of the tissue.

Ballistic Stretching

Ballistic stretching (sometimes referred to as *dynamic stretching*) differs from static stretching in that a series of movements are used to stretch the muscles and connective tissues.[2] This is a high-intensity, short-duration bouncing stretch.[1] The stretch that occurs often results in contraction of the antagonistic muscles to the tissues being stretched. Although effective stretching may occur with this form of stretching, there is a risk of tissue injury, and this form may not be appropriate for patients early in the postoperative period. In fact, the muscle tension that occurs with ballistic stretching is approximately twice that obtained with low-intensity prolonged stretching.[28] In general, a period of light warm-up activity and static stretching exercises should precede balistic stretching. In humans, ballistic stretching is sometimes used by individuals who need to perform quick motions such as in martial arts or hurdling. There may be similar applications in canine athletes, but this has not been investigated.

Proprioceptive Neuromuscular Facilitation Stretching

Proprioceptive neuromuscular facilitation stretching is more complex and takes advantage of neuromuscular stretch activation. The stretches use a contract-relax

sequence, an agonist muscle contraction, or a contract-relax-agonist muscle contraction sequence.[2] Unfortunately, these stretches require the patient to actively and consciously contract a muscle group. Because we cannot verbally communicate to an animal when the muscle should contract, the therapist must use the other forms of stretching. There is a possibility that neuromuscular electrical stimulation may be used to provide active muscle contraction of a particular agonist muscle group, but clinical experience in this technique is limited.

Precautions and Contraindications to Stretching

Joints should not be forced into an uncomfortable position beyond the available ROM. Patients should not be painful during or after stretching. Caution should be exercised if a limb has been immobilized for a length of time. The goal should be to stretch and realign connective tissue, not rip and tear tissues. Stretching should not be performed if a ligament or tendon has been injured until the fibrous tissue can withstand some stress. Similarly, caution should be exercised during stretching in regions of relatively new fractures after repair has been performed.

Stretching Studies

Although there is a relative paucity of data and scientific knowledge regarding stretching to improve flexibility, especially in animals, it is a widespread practice in both human and animal rehabilitation. A study in Labrador retrievers with elbow and stifle osteoarthritis investigated the effects of a home passive stretching program carried out by owners on joint ROM. Owners were carefully insructed on how to perform the stretches and received supervised practice. ROM measurements were obtained at the start of the study and after a 21-day home program. The home program consisted of owners performing 10 stretches of 10-second duration each two times daily. After 21 days, there was a significant increase in ROM, with an average increase of 14.6 degrees.[29]

Studies of stretch behavior of normal human muscle have yielded interesting results. One study evaluated the passive properties of the hamstring muscle group during stretch while measuring passive hamstring resistance and electromyographic activity.[30] Resistance to stretch was defined as passive torque of the hamstring muscle group during passive knee extension. The knee was passively extended to a predetermined final position where it remained stationary for 90 seconds (static stretch). Alternatively, the knee was extended to the point of discomfort (stretch tolerance). Passive energy and stiffness were calculated. Torque decline in the static phase was considered to result from viscoelastic stress relaxation of the tissues. A single static stretch resulted in a 30% viscoelastic stress relaxation. With repeated stretches, muscle stiffness temporarily decreased, but returned to baseline values within 1 hour. Stretching over a 3-week period increased joint ROM because of a change in stretch tolerance rather than in the passive properties of the muscle. Strength training resulted in increased muscle stiffness, which was unaffected by daily stretching. Inflexible and older subjects have increased muscle stiffness, and a lower stretch tolerance compared with subjects with normal flexibility and younger subjects. The concept of an increase in stretch tolerance was confirmed in another study of people in which individuals underwent a stretching protocol of the hamstring muscles for 6 weeks.[31] Although there was no increase in the extensibility of the hamstring muscles, there was an improvement in stretch tolerance, which resulted in an increase in joint ROM.

Another study evaluated the effects of one 10-minute stretch on muscle stiffness in people with short hamstrings.[32] The subjects were unable to touch the floor when bending forward. One group performed static stretching exercises for 10 minutes interspersed with relaxing, and an untreated group served as controls. One 10-minute stretch resulted in a significant increase in passive muscle moment, ROM, and elongation of the hamstrings. There was no significant change in passive muscle stiffness, however, with respect to the prestretch stiffness curve. The authors also concluded that one session of static stretching does not influence muscle stiffness, and the increased ROM and extensibility of the hamstrings resulted from an increase in stretch tolerance.

Acute changes in the flexibility of hamstring muscles were evaluated in older adult humans.[33] A single 32-second stretch was performed by men and women with a mean age of 65 years. Although the changes were moderate, the increase in flexibility was significantly greater in the stretch group as compared with the controls (4 degrees vs. 1 degree).

Although most muscle stretching studies have focused on defining the biomechanical properties of isolated elements of the muscle-tendon unit or on comparing different stretching techniques, one group of investigators evaluated stretching properties in an entire muscle-tendon unit.[34] Rabbit long digital extensor and cranial tibialis muscle-tendon units were evaluated using common stretching techniques. The effect of varying stretch rates was also evaluated. Similar to other studies, the muscle-tendon units responded viscoelastically to tensile loads. Both cyclic and static stretching resulted in sustained muscle-tendon unit elongations, suggesting that greater flexibility can result if these techniques are used clinically. With repetitive stretching, there was little additional alteration of the muscle-tendon after four stretches, implying that a minimum number of stretches leads to most of the elongation in repetitive stretching. Also, greater peak tensions and greater energy absorptions occurred at faster stretch rates,

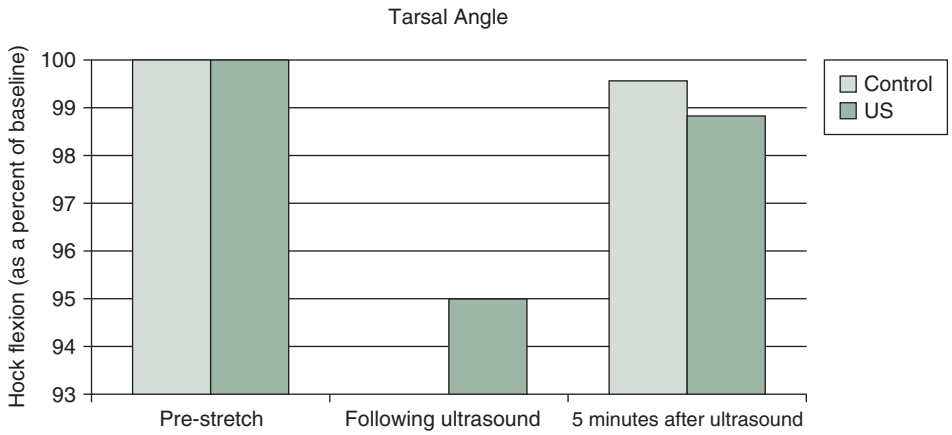


Figure 25-10 A mild stretch applied to the common calcaneal tendon immediately after ultrasound treatment (10 minutes of 3-MHz continuous ultrasound heating at 1.5 W/cm^2) resulted in a 5-degree increase in hock flexion, but this increased flexion was lost within 5 minutes after the ultrasound was discontinued. Repeated stretching of control tendons without ultrasound resulted in no increase in hock flexion.

suggesting that the risk of injury in a stretching regimen may be related to the stretch rate. Indeed, one group of investigators found that during slow (or static) muscle stretching, factors inhibiting motor neurons contributed to the lengthening of the muscle.[35]

Passive stretching may also help to prevent muscle atrophy by allowing constant change in the length of the muscle fibers. Intermittent muscle stretching helped to prevent atrophy and loss of sarcomeres in muscles of mice that were immobilized in a shortened position.[36] In this model, a static stretch for as little as 30 minutes per day helped to preserve sarcomeres. In addition, there was less development of intramuscular connective tissue with some stretching. In a study of rat tail tendons, a low-force stretch over a prolonged period resulted in effective elongation of the tendon.[37] Also, the proportion of prolonged tendon lengthening was greater with low-force, long-duration stretching.[23] Heating tissue before stretching has been advocated to help improve the effectiveness of stretching. Elevating the tissue temperature before stretching the rat tail tendons resulted in significantly less tissue damage; and lower loads applied with heating for a prolonged period resulted in significantly greater elongation. One researcher concluded that approximately three times as much force is needed to elongate tendons at temperatures of 20° to 30° C than at 43° C.[38]

Therapeutic ultrasound is frequently mentioned as an adjunctive treatment to improve the effectiveness of stretching through its ability to warm tissues. However, the specific effects of heating on tissues and the manner in which stretch is affected is unclear. In a study of ultrasound treatment of the common calcaneal tendon of dogs, it was found that pulsed-mode ultrasound was not effective in increasing tendon temperature, but continuous-mode ultrasound was.[39] The amount of heating with 3-MHz continuous-mode ultrasound was related to the power, with 1.5 W/cm^2 increasing tendon temperature more than did 1 W/cm^2. Interestingly, the greatest temperature increase occurred within 3 minutes, with little increase thereafter. Tendon cooling occurred very rapidly, with return to baseline temperature within 1 minute after stopping ultrasound. A 4-kg stretch applied to the tendon immediately after ultrasound treatment (10 minutes of 3-MHz continuous ultrasound heating at 1.5 W/cm^2) resulted in a 5-degree increase in hock flexion, whereas two bouts of stretching of the contralateral nontreated calcaneal tendon within 5 minutes did not result in increased hock flexion. However, the increased hock flexion was lost within 5 minutes after the ultrasound was discontinued (Figure 25-10). Clearly, methods of maintaining the increased stretch are needed to improve tissue extensibility.

One study performed on humans also evaluated the effects of (1) static stretch and ultrasound, (2) static stretch alone, or (3) no treatment on triceps surae muscle extensibility.[40] Static stretch and ultrasound consisted of 7 minutes of continuous ultrasound at 1.5 W/cm^2 to the muscle with stretch applied during the seventh minute. Static stretch alone consisted of a 51-lb load applied for 1 minute. Static stretch and ultrasound treatment increased elbow flexion an average of 1.2 degrees, or 20%, more than static stretch alone, whereas static stretch alone increased flexion an average of 1.3 degrees, or 27%, more than no treatment. Both increases were statistically significant.

A similar study on people confirmed the short-term benefit of heating with ultrasound before stretching.[41] One group received ultrasound (1.5 W/cm^2) to the musculotendinous junction of the triceps surae and then stretching, and another group received stretching alone. Ultrasound and stretch increased mean ROM more than did stretch alone, but there were no long-term residual effects.

Another study evaluated whether heating with continuous-mode ultrasound would augment the effect of stretching of the medial collateral ligament of the knee in normal humans.[42] Treatment consisted of 3-MHz, 1.25 W/cm^2

continuous ultrasound or sham treatment over the medial collateral ligament. A valgus stretch of the knee (10 ft-lb for 2.5 min) was performed after ultrasound treatment. Stretching alone resulted in a 1-degree valgus displacement of the knee, whereas stretching with ultrasound resulted in a displacement of 1.24 degrees. However, these differences were not statistically significant.

The effect of stretching on athletic performance such as agility competitions, field trials, Schutzhund, etc. is controversial. Traditionally, static stretching precedes athletic performance. However, the effectiveness of stretching on athletic performance has come into question in recent years based on research. Several studies have shown a decrease of muscle force output around a single joint associated with static stretching.[43-45] Proposed mechanisms for this decrease in muscle force include a decrease in muscle stiffness,[45] muscle inactivation during muscle stimulation,[43] and decreased muscle activity after maximum voluntary contraction. In addition, because stretching can reduce muscle stiffness, it may have a negative effect on the ability to generate a forceful contraction.[45] Stretching also appears to decrease motor neuron excitation.[46] One study evaluated the effect of stretching on agonist and antagonist muscle activity and muscle force output with joint isometric contractions.[47] The authors concluded that stretching appears to decrease muscle force output by up to 20% in a single joint isometric contraction and the rate of force development in a multiple joint isometric contraction in people. Stretching may cause a decrease in muscle force generation up to 16 minutes after stretching and may not be recommended as a general warm-up procedure, especially prior to strength or power activities. Current recommendations for stretching during warm-up activities should be carefully considered. It is not clear how applicable these results would be when combined with warm up, practice drills, and low-intensity dynamic exercises.

Another traditional belief is that stretching prior to activity may result in fewer injuries associated with performance. This topic has been recently reviewed in people.[48] The purposes of stretching before athletic performance include ensuring that there is sufficient ROM in the joints to perform the activity, and to reduce muscle stiffness, theoretically reducing the risk of injury. The effect of stretching on injury prevention has shown mixed results. In general, it is believed that stretching along with warm-up does not affect the incidence of overuse injuries. However, stretching may reduce the incidence of muscle strains. In general, it may be beneficial to perform static stretching when a large ROM of various joints is required for activity. A combination of stretching and warm up is likely needed to reduce the risk of injury in these situations. It is most beneficial to target the stretching to those muscle groups known to be at risk for a particular activity. At least four to five 60-second stretches to the point of initial discomfort are recommended to reduce resistance to stretch, followed by some sport-specific warm-up activity. However, stretching prior to an activity that has more overuse injuries rather than muscle strains is unlikely to be effective in reducing injury.

Clinical Application of Stretching

In preparation for stretching, it is beneficial to perform some low-intensity active exercise if possible, such as walking and light jogging. In addition, application of superficial heat or therapeutic ultrasound before stretching may improve tissue extensibility. There may be less damage to tissues, and less force may be required to attain increased tissue length with stretching if the tissues are warmed. The combination of muscles, tendons, skin, and joint capsule should be considered and their relative contributions to restricted motion assessed. For example, the target tissue for lengthening a muscle that is contracted is the muscle belly, not the tendon. The patient should be in a comfortable position, which is generally in lateral recumbency, and should be on a padded surface. It is important that the patient be as relaxed as possible, and, in some cases, mild sedation or tranquilization may be beneficial.

Static Stretching

One hand should stabilize the bone proximal to the joint, and the other should stabilize the bone distal to the joint. In general, the distal bone should be moved relative to the proximal bone. The affected limb and joint should be slowly placed through one end of a ROM (usually beginning in flexion) until a restriction to motion is felt. Very gentle traction may be applied to the joint while slowly stretching to the point of initial restriction. The patient may indicate that it feels mild discomfort, such as by turning the head or mildly tensing the muscles in the affected limb. Under no circumstances should more severe pain be inflicted, which might be indicated by vocalizing, trying to move away, or attempting to bite. The stretch should be prolonged, ideally for at least 15 seconds. During the stretch, a conscious effort should be made to try to increase the joint excursion without increasing the level of discomfort. There should be no bouncing motions during the stretch with static stretching. Following the stretch at one end of the motion range, the pressure is slowly released and the opposite end of the ROM should be stretched. In some situations, dogs that resist manual stretching may tolerate other stretching techniques, such as placing the dog in ventral recumbency with the limbs flexed under the body and slowly pressing down on the body to achieve greater elbow flexion (for example, Figure 25-11).

In some situations, muscles that cross two joints, such as the rectus femoris and biceps brachii muscles, may be restricted. In these situations, it is beneficial to put an individual joint through a stretching session, and then place the other joint through a stretching session. As ROM and flexibility improve, both joints may be simultaneously



Figure 25-11 A mild stretch is applied to increase elbow flexion using the dog's body weight. The antebrachium is comfortably supported on a table with padding.

placed through stretching exercise. For example, the hip and stifle joints may be stretched in extension. This is more challenging, and there will usually be less joint excursion with simultaneous extension of both joints than if each joint is individually stretched in extension. However, these motions more completely reflect the use of these joints and muscles during normal gait, and it is valuable to perform these maneuvers.

The therapist should be patient and not try to achieve full ROM in one or two sessions. The ideal daily frequency of stretching is unknown for dogs. In general, two to four sessions per day may initially be required, with the frequency decreased as normal ROM and tissue extensibility improve. The process must be applied consistently and regularly to obtain good results, and it may take 2 to 3 weeks to see noticeable improvement.

Prolonged Mechanical Stretching

The most problematic joints that are affected with immobilization of a limb in a cast or splint are the digits, carpus, and tarsus, which may develop flexural contracture. This may occur in either the front or the rear limb joints as a result of fractures of the radius and ulna or tibia and fibula, or in the digits with surgical fusion (arthrodesis) of the carpus or tarsus. After 4 weeks in a coaptation device, the joints are often contracted in a flexed position and are painful when attempts are made to place them in a functional walking position.

Normal static stretching of the affected joints should be performed. It is usually only practical to stretch all of the digits at the same time. Between stretching sessions, a dorsal fiberglass splint, molded to the normal contralateral side, is placed on the affected limb to help maintain a prolonged mechanical stretch. The splint may be manufactured using fiberglass casting material folded back and forth on itself, using the normal limb as a template. Several layers of cotton cast padding are placed over the affected limb. The dorsal splint is applied, and gauze bandaging material is rolled on to the limb under mild tension to pull the foot up to the splint, applying controlled prolonged gentle stretch to the restricting tissues. The wrap is changed one to three times per day, and static stretching and ROM exercises should be performed while the wrap is removed. When the wrap is replaced, additional stretch may be applied by increasing the tension on the gauze bandaging material. In general 5 to 10 days is usually adequate to regain normal ROM of the digits. Similar principles may be applied to more proximal joints, such as the elbow and stifle. In some situations, it may be difficult to use a fiberglass splint, and placement of transcutaneous pins in the bones proximal and distal to the affected joint may be necessary to provide good fixation. Elastic bands may be placed to achieve flexion or extension as needed.

Ballistic Stretching

To perform ballistic stretching, the patient should be comfortable and the affected limb stabilized. The joints are positioned until a gentle stretch of the target tissues occurs, and then the therapist applies gentle "bouncing" of the tissues. The therapist should be careful to use only gentle movements to avoid damage to the tissues and pain. Because of the risk of tissue injury, this technique should not be performed in traumatized tissues, after surgery, or on tissues that are inflamed or edematous.

Ballistic stretching may be beneficial to normal dogs that undergo ballistic-type activities, such as agility dogs that jump and turn sharply, or dogs that play Frisbee. A technique of ballistic stretching in sporting dogs may be performed using a therapy ball or roll, trampoline, or inflatable mattress. The trampoline and mattress provide greater stability so that a single person can perform the stretches, whereas two people are generally needed when a therapy ball or roll is used. With the animal in a standing position, the animal is gently bounced up and down. Depending on the situation, the dog may also be placed in a sternally recumbent position on the device. Gentle bouncing of the dog will allow ballistic stretching of the joints in a flexed position. Ballistic stretching may also be performed with a joint in extension using a therapy ball or roll. For example, a dog may have its forelimbs on the ground with the rear limbs pulled back in extension over a therapy roll (Figure 25-12). Rapid, gentle rolling of the therapy roll back and forth or gentle bouncing of the rear limbs on the roll results in ballistic stretching of the hip and stifle joints. Similar principles may also be used for ballistic stretching of the forelimb joints in extension.

Summary

ROM and stretching exercises are basic parts of a complete physical rehabilitation program. The goals of ROM and stretching should be to lengthen muscles, improve joint

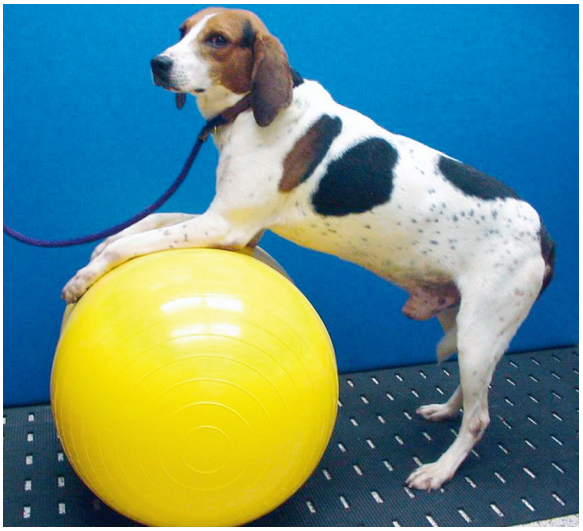


Figure 25-12 A physioball is used to increase hip extension by gradually rolling the ball forward to the point of a stretch on the cranial aspect of the hip joint and the periarticular tissues.

ROM, increase tissue mobility, and improve functional use of the affected joints and limb.

The therapist must be patient and be satisfied with small gains and slow improvement. To properly monitor patients, frequent measurement of ROM should be obtained using goniometry, and the results should be recorded to chart the patient's progress. Impatience and overzealous treatment may ultimately slow improvement and may actually reduce joint motion and worsen contracture of soft tissues. This is because tissue damage and pain may delay the active use of a limb through the available ROM, resulting in increased fibrosis of tissues and decreased flexibility.

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26 Joint Mobilization

Deborah Gross Saunders, J. Randy Walker, and David Levine

Manual therapy techniques are skilled hand movements intended to improve tissue extensibility, increase range of motion (ROM), induce relaxation, mobilize or manipulate soft tissue and joints, modulate pain, and reduce soft tissue swelling, inflammation, or restriction.[1] The primary techniques included in manual therapy are mobilization and manipulation of joints and associated soft tissues. Mobilizations are passive movements that are either oscillatory or sustained stretch performed in such a manner that the patient can prevent the motion if so desired. These motions are performed anywhere within the available ROM. The intent of this chapter is to provide an overview of the principles of manual therapy, followed by selected treatment techniques for the hip, stifle, elbow, shoulder, carpus, and the thoracic and lumbar spine. The techniques of GD Maitland,[2,3] an Australian physical therapist who developed a clinically based approach in the 1960s and 1970s, are emphasized in this chapter. Maitland described four grades of mobilization (I-IV) (Figure 26-1) and manipulation. Manipulation, a grade V mobilization, is a high velocity, low amplitude passive movement that cannot be prevented by the patient and is typically performed near the end of the available ROM.

There have been numerous randomized controlled trials (RCTs) in humans that have demonstrated the efficacy of manual therapy for treating patients with a variety of disorders in the spine and peripheral joints. Many of these studies have compared "traditional treatments," such as exercise, pharmaceutical interventions, rest, and placebo, with manual therapy. Other RCTs have compared these traditional forms of therapy to groups receiving both the traditional therapies plus manual therapy. The majority of these studies have found that manual therapy is as effective if not superior to traditional therapies.[4-15] Common outcome measures assessed have been pain, functional scales, disability levels, ROM, number of treatments needed, length of time in treatment, and cost effectiveness of treatment. Despite the growing body of evidence for manual therapy, it is viewed as a complementary therapy in human medicine by many, even though its effectiveness seems well substantiated and the risks are low. The most likely reason for its slow acceptance is that the skill level required to properly apply these techniques is higher than with traditional therapies such as exercise or modalities. Rationales for the reasons manual therapies may work have been investigated and include reducing muscle inhibition,[16] decreasing pain,[17,18] improving intervertebral disk hydration,[19] correcting joint displacement, adjusting joint subluxations, restoring bony alignment, reducing nuclear protrusion,[20,21] and placebo effect.[8] More recent theories include evidence supporting the need for adequate stresses and normal movement as being critical to maintain the integrity of collagenous tissues, muscles, and bones.[22] Evidence of the effectiveness of manual therapy must be established in small animals. Although many anatomic similarities exist between humans and small animals, we cannot assume the techniques will yield the same results.

Indications for Manual Therapy

Manual therapy is indicated for pain and loss of motion that occur secondary to neuromusculoskeletal dysfunction. The patient may have pain with motion or rest; pain in the midst of available ROM or at the end of available ROM; or pain or stiffness resulting from postural changes. Specific examples include dogs with limited motion secondary to canine hip dysplasia, elbow dysplasia, intervertebral disk disease, and osteoarthritis. Manual therapy differs from stretching in that when a stretch is applied, a low load is placed on the tissues for a specified amount of time (usually 10 to 30 seconds) to help elongate them. In manual therapy the force is applied in an oscillatory manner rather than in a sustained manner.

Contraindications and Precautions for Manual Therapy

The list of contraindications presented by Dutton[23] includes spinal instability, bacterial infection, malignancy, systemic localized infections, sutures over the area, recent fracture, cellulitis, febrile state, hematoma, acute circulatory condition, an open wound at the treatment site, osteomyelitis, advanced diabetes, hypersensitivity of the skin, inappropriate end feel (spasm, empty, bony), constant severe pain,

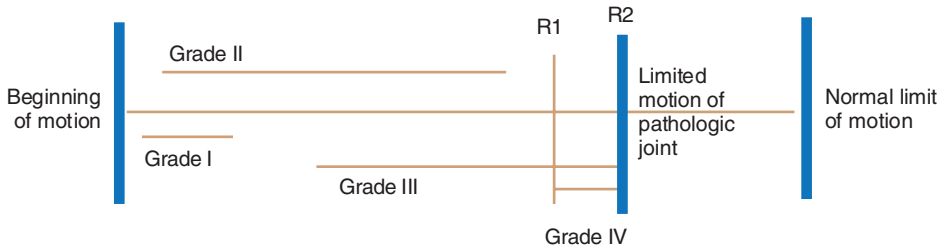


Figure 26-1 Graphic description of grades of joint mobilization.

extensive radiation of pain, pain unrelieved by rest, any undiagnosed lesion, and severe irritability (pain that is easily provoked, and that does not go away within a few hours). Additional contraindications specific to dogs include contractures such as fibrotic myopathy and quadriceps contracture, or in other circumstances in which manual therapy is unlikely to effect any change and may be painful. An overly aggressive or fearful dog that may bite the therapist or unrelaxed dogs are also contraindications. Total elbow and hip replacements should be considered a contraindication for grades III-V accessory mobilizations until the appropriateness of joint mobilization techniques are further evaluated for these procedures.

Precautions (proceed with caution) identified by Dutton[23] include joint effusion or inflammation, rheumatoid arthritis, presence of neurologic signs, osteoporosis, hypermobility, pregnancy (if the technique is to be applied to the spine), and steroid or anticoagulant therapy.

Basic Principles of Manual Therapy

There are a number of approaches to manual therapy, including those developed by physicians[24-26] and by physical therapists.[2,3,27-29] Regardless of the approach, there are several principles that should be considered as the patient is examined and treated. Maitland stresses the importance of communicating with the patient to fully "understand what the patient is enduring."[2] Of course, in the case of animals, the examiner must communicate with two constituents—the dog and the owner. Body postures, pain patterns, and respiratory patterns should be closely monitored in the patient.

Physiologic and Accessory Motion

Physiologic motion is the normal active motion that is available at any synovial joint. Another way to describe physiologic motion is the motion that occurs in the cardinal planes. Examples include flexion, extension, abduction, and internal rotation. Accessory motions are movements that cannot be performed actively, but can be performed passively. Examples are distraction, glides, spins, and rotations of a joint. Accessory motions must be present for full physiologic motion to be present.

Concave and Convex Relationship of Joints

All synovial joints have a convex-concave relationship. When the examiner is passively moving a joint, caution should be made to move the joint in a manner similar to how it moves when the joint is being actively moved by the dog. Osteokinematics is defined by how the bone is being moved through space (i.e., flexion, abduction). Arthrokinematics is defined by how the joint surfaces are moving as the bone is being moved (i.e., rolling, sliding, and spinning). When the joint surface is convex with respect to the other concave side of the joint, the articular surface moves in the opposite direction of the shaft of the bone. When the shoulder joint is being flexed (as in the swing phase of gait) by moving the humerus on the scapula, the convex surface of the proximal humerus is sliding and spinning on the concave glenoid of the scapula. When the joint surface is concave, the articular surface moves in the same direction of the shaft of the bone. When the distal radius is being moved on the stationary carpal bones (as in the stance portion of gait), the concave surface of the radius is rolling and sliding on the convex proximal row of carpal bones. Manual therapists strive to move joint surfaces physiologically to avoid injuries such as joint subluxations and sprains.

Relationship of Pain and Range of Motion

Maitland states, "During examination and assessment, pain should never be considered without relation to range nor range without relation to pain."[2] Depending on the nature of the injury or disease, joints are affected by the presence of pain and stiffness. If pain is the primary problem, it may limit motion, as in hip dysplasia. When the pain diminishes, the ROM may improve. If the primary problem is joint dysfunction or stiffness, pain will be most evident at or near the end of available ROM. As the available ROM increases, pain will be less of a factor. In those cases in which both pain and loss of motion occur simultaneously, the examiner must decide which is the primary problem. These relationships are the basis of deciding which grade of passive movement and other therapies should be used to treat the problem.

Table 26-1 Joint End Feel Characteristics

Name	Description	Example
Bony or hard	Bone approximates bone, resulting in an abrupt, hard stop.	Loss of flexion with end-stage elbow osteoarthritis; loss of stifle motion as a result of quadriceps contracture following fracture of the distal femoral physis
Soft tissue approximation	Motion is stopped by compression of soft tissues. Abnormal if occurs too early in the range as the result of edema.	Normal stifle flexion
Capsular or firm	A firm but slightly yielding stop occurs as a result of tension in the joint capsule or ligaments. Abnormal if occurs too early in the ROM.	Normal carpal extension
Springy block	A rebound is felt at the limit of motion—motion stops and then rebounds. Abnormal; may indicate joint effusion or a joint mouse.	Entrapped torn medial meniscus.
Empty	No end point is felt because the patient stops the motion because of pain; no resistance felt. Abnormal; indicates presence of sharp pain.	Sciatic nerve entrapment as a result of a migrating intramedullary pin; fracture of the lateral humeral condyle

ROM, Range of motion.

If pain occurs before the examiner feels resistance, the primary problem is pain; if the pain occurs when resistance is felt, or after, the primary problem is limitation of motion with joint involvement. In addition, if active and passive motions are limited or painful in the same direction, the lesion is in the noncontractile tissues. For example, if the cranial joint capsule of the stifle is tight, flexion of the stifle is limited with active and passive flexion of the joint. Conversely, if active and passive motions are limited or painful in opposite directions, the lesion is in the contractile tissues—muscle and associated tendon. For example, if the biceps tendon is affected, active flexion of the elbow and passive extension of the elbow are painful with a possible reduction in ROM. These presentations affect which grade and type of mobilization should be used to treat the problem.

The dog exhibits a *capsular pattern* if the entire capsule of the joint is inflamed or involved. Cyriax[24] described a capsular pattern as a joint-specific pattern of loss of motion with arthritis. These have not been described for dogs, but limitations in movement accompany certain disorders. An example is that hip extension and hip abduction are typically the most limited motions in a dog with hip dysplasia.

End feel is the sensation imparted to the examiner when the end of passive ROM is encountered. The passive motion must be performed with a relatively quick movement and the end of range is bumped briskly. Cyriax[24] described the quality of normal and abnormal end feels, which are defined in Table 26-1.

Assessment

The therapist participates in three activities while managing a patient problem: examination, treatment, and assessment. Although each is important to the provision of a complete and quality program, assessment is the keystone. Assessment involves "open-mindedness, mental agility and mental discipline, linked with a logical and methodical process of assessing cause and effect."[2] The therapist is thinking and assessing to make certain that the right task is being performed and that the task is being performed correctly. The therapist must continually assess the decision-making process during examination and treatment procedures. Assessment is implemented at three points of the patient interaction. The first assessment is implemented during the initial examination of the patient, making certain that clinical signs are examined and evaluated to reach the best diagnosis and formulate an appropriate plan of care. The second assessment occurs during the treatment session. The therapist assesses the appropriateness of the treatment techniques while the patient response is evaluated. The third assessment is an analytic process to consider the entire treatment program to determine whether the patient is progressing as a result of the treatment and to determine the prognosis for resolution of the current problem.

The Clinical Environment

The clinical setting should be safe for both the patient and the therapist so that the desired outcome of the examination and treatment session will more likely be achieved. First, a quiet area that is free of distractions for the dog facilitates relaxation and cooperation. The area should also be safe for the dog and therapist. It may be necessary to muzzle the dog initially, especially with a fearful or apprehensive dog. Second, the therapist should make certain he or she is using proper body mechanics to prevent undue stress on the spine and joints of both the dog and the therapist. Third, the therapist should approach the dog from a position where the dog can see the therapist, and he or she should touch the dog with firm, but not aggressive hands. Touch the dog at a place that is not painful and gradually move toward the involved area. Finally, the affected part should be cradled or held with a firm but gentle supporting grip to encourage relaxation and trust by the dog. If at any time the dog or therapist is not relaxed, the activity should be discontinued and further assessment and correction of the situation should be pursued.

Evaluation

The basic components of the initial examination for musculoskeletal dysfunction are observation, history of the present episode, relevant past history, active and passive ROM, functional tasks, strength assessment, and palpation. After the interview and examination are complete, all data should be evaluated to determine a rehabilitation diagnosis and a plan of care. The primary problem should be assessed to determine if it is primarily a painful (acute) or stiffness problem. After these decisions are made, only then should joint mobilizations be considered as a part of the plan of care.

Treatment with Mobilization

The effectiveness of the manual therapy treatment program is affected by the environment and the skills of the therapist. Efforts must be made to facilitate relaxation of the dog to avoid excessive pain and stress to the animal. The therapist's grip should be firm enough to support the limb, but not excessive. Good body mechanics and positioning by the therapist will help to make the movements controlled and in the desired pattern.

Grades

Joint mobilization should be planned with a specific grade of mobilization in mind. Grades are assigned to the mobilizations depending on the range through which the mobilization is applied and the point in the range where it is applied. Figure 26-1 shows a modification of Maitland's graded mobilizations.[2]

R represents resistance. *R1* is the point in passive ROM at which the therapist senses resistance from a stretch on the noncontractile structures of a joint. *R2* is the resistance felt at the end of available passive ROM.

Grade I

Grade I mobilizations are small amplitude movements performed with three to four oscillations per second. There should be no pain during the oscillations; this is a very relaxing treatment technique for the dog. The therapist should be working in the range opposite of that which causes pain and avoid end range (R2). For example, if pain occurs during extension, then a grade I mobilization is performed in flexion, away from any area of the range of motion that could be painful.

Grade II

Grade II mobilizations are large-amplitude movements that are performed with three to four oscillations per second. A grade II mobilization should not cause pain during the oscillations. The mobilization should be in the range opposite that of the pain and the therapist should avoid either end range (R2).

Grade III

Grade III mobilizations are large-amplitude movements performed with three to four oscillations per second at the end of range of the restricted motion (bumping R2 but then moving off of any tightness). This amplitude of motion may cause slight discomfort to the patient.

Grade IV

A grade IV mobilization is a small-amplitude movement that is performed with three to four oscillations per second between R1 and R2. This amplitude of motion may cause slight discomfort for the patient. A grade IV joint mobilization is indicated when a grade III is too painful or not tolerated by the patient. Even though the grade IV may be more vigorous, it is sometimes better tolerated because the joint motion is very small and it does not completely progress to R2.

How to Choose the Appropriate Mobilization Grade

When ROM is decreased due to **pain:**

- If the pain is treated, then ROM should increase.
- Grade I and II mobilizations should be performed.
- Grade I and II mobilizations should be performed in the pain-free range for **30 seconds** or longer. Function should be assessed after mobilization to determine whether any change has been achieved.

When ROM is decreased due to **stiffness:**

- Grade III and IV mobilizations should be used.
- Grade III and IV mobilizations should be performed in the direction of the stiffness for 60 seconds if possible.

Function should be assessed after mobilization to determine whether any change has been achieved.

If both **pain and stiffness** are present, the therapist must decide what the primary problem is. Does the pain limit ROM or does the stiffness cause the pain? The sequence of pain and resistance can contribute to the treatment plan. For example:

- If **pain occurs before resistance,** use techniques to control the pain before progressing to more aggressive treatment.
- If **pain occurs with resistance,** mobilizations may be used with caution. However, it is customary to treat the pain and then the stiffness.
- If **pain occurs after the resistance,** vigorous mobilization may be used to treat the stiffness followed by techniques for pain.

There are two types of mobilizations:

- **Physiologic mobilizations** are done in the same pattern of movement that is produced by voluntary muscle contraction.
- **Accessory mobilizations** are performed in a pattern of movement that must be done passively and cannot be produced by voluntary muscle contraction. These should be done in the midrange of the joint motion (open pack position) because this allows for more joint play or motion.

There are additional guidelines to help the therapist decide which type of mobilization to perform.

- If pain is being treated and **physiologic mobilization** grades I and II cause pain, then **accessory** grades I and II should be performed.
- If 50% of physiologic active ROM is not available secondary to pain, then use **accessory** grades I and II to treat the pain (50/50 rule).
- If treating stiffness (Grades III and IV), the therapist should perform 60 seconds of an **accessory** mobilization followed by 60 seconds of a **physiologic** mobilization and then assess the effect. During any mobilization treatment, an assessment should be made after each set of oscillations, whether for pain or stiffness, to determine the effect of the mobilization. This sequence should be performed three times.

Oscillation versus Sustained Stretch

Mobilization for joint restrictions should be oscillations. Mobilization for muscle, tendon, and skin restrictions should be sustained. Repetitive low-intensity stretches are beneficial to stimulate tissue elongation. Contracted tissues respond positively to sustained stretch.[30]

Open versus Closed Kinetic Chain

Dutton defines open kinetic chain activities as when the "involved end segment of an extremity [is] moving freely through space, resulting in isolated movement of a joint."[23] Examples of an open kinetic chain activity in a dog include when a paw is swinging forward in the gait cycle or when the dog is moving a forelimb while in lateral recumbency. On the other hand, closed kinetic chain activity occurs when the distal segment of an extremity is in contact with a surface and the limb is weight bearing.[28] Examples of a closed kinetic chain activity include the stance phase of gait. It is of value to note whether a limb is being exercised in the open versus closed kinetic chain. Closed kinetic chain activities facilitate proprioception, muscles working in groups, and coordination of joint function. On the other hand, it is easier to achieve isolated joint motion in the open kinetic chain.

Dogs perform a large array of movements to maintain function. The restoration of normal movement plays a significant role in the treatment of the dog. Joint mobilization offers one method of obtaining the motion required for the restoration of function through manual therapy skills.

Prior to beginning mobilization on the dog, it is essential that the therapist be completely familiar with the bony landmarks associated with the joint. The joint should be evaluated to be certain that pathologic joint luxation or subluxation is not present. Hair may need to be pulled out of the way or clipped to expose the joint. Active and passive ROM should be functionally assessed and measured by goniometry prior to beginning the treatment. The assessment of passive and active ROM is an important aspect of the treatment process. The comparison of ROM before and after treatment demonstrates the effectiveness of the treatment.

The initial treatment of a dog's joint depends largely on the nature of the problem, in particular, pain or stiffness. If the primary problem is pain, a relaxation technique, such as light massage, may be used to calm the dog and to gain trust. If the animal is not in pain, but has stiffness, the joint or joints to be mobilized should be prepared for treatment with a warming modality or light exercise. Moist heat, massage, or therapeutic ultrasound may be used prior to treatment. Active exercise, including aquatic therapy, may also be used. The joint mobilization treatment should then be followed with passive and active ROM accentuating the motion addressed. Therapeutic exercises prescribed should also address the motion. Ice may then be applied after the session to help prevent soreness from the mobilization and other activities.

Forelimb injuries are common in dogs. Elbow dysplasia and resultant osteoarthritis of the elbow occurs quite frequently in dogs. In agility and jumping events, problems in the shoulder, elbow, and carpus are common secondary to the stresses placed on the forelimbs. Functional ROM at the carpus and elbow is essential to perform activities. For example, full elbow ROM is required for normal function. ROM demands in working and athletic dogs are greater than in pet dogs. For example, full shoulder extension is

required for dogs participating in agility activities such as jumping.

The hindlimb is responsible for generating propulsion of the dog. Decreased ROM may result in a loss of power and function, especially loss of joint extension. Decreased joint motion in the hindlimb is common with hip dysplasia, cruciate disease, muscle injuries, and osteoarthritis.

The Shoulder Joint

The shoulder, or glenohumeral joint, is a ball-and-socket joint and follows the convex on concave rule of motion (Box 26-1). Impairment of shoulder ROM may be seen after a fracture of the humerus, bicipital tenosynovitis, osteochondritis dissecans, infraspinatus contracture, scapular fracture, surgical repair of shoulder instability, and other conditions. Compensations in the shoulder complex may also be seen as a result of hindlimb or spinal problems such as intervertebral disk disease, canine hip dysplasia, cruciate disease, or neurologic problems. It is believed that dogs increase the amount of weight placed on their forelimbs to compensate for a problem affecting the spine or hindlimb. Over time, problems may develop in the forelimbs secondary to the increased forces placed on the joint.

Lateral Distraction of the Glenohumeral Joint (Figure 26-2)

Dog position: The dog is in lateral recumbency with the shoulder to be treated facing the therapist.

Therapist position: The therapist is ventral to the dog's shoulder, facing the shoulder to be mobilized.

Hand position: The stabilizing hand is placed on the proximal scapula. The web space and fingers of the mobilizing hand are placed under the axilla, as close to the proximal humerus as possible.

Mobilization: A lateral distraction is applied with the mobilizing hand while the stabilizing hand maintains the position of the scapula. The distraction is held for up to 15 seconds and then released. This may be repeated up to 10 times. This is a sustained mobilization.

Objective: Stretch the capsule, relax the surrounding tissues, and prepare the joint for additional mobilizations.

This technique can be performed as a grade II mobilization to diminish pain in the shoulder joint. The mobilizing hand should be oscillated at a rate of two to three repetitions per second. The head of the humerus should be moved laterally only a minimal amount, making certain that no joint resistance is felt by the therapist and no pain is experienced by the dog. The oscillations are maintained for approximately 30 seconds, at which time the dog should be assessed to determine whether there has been any change in function. The technique may be repeated up to three times if the dog's pain improves.

In addition to a lateral distraction, a grade III oscillation can be performed to improve joint restriction. In this case, pain is not a major factor and should only be present when the joint is moved to the point of joint motion limitation. To perform a grade III mobilization, the head of the humerus is oscillated to the point of end feel of the capsule, where the bone cannot be moved any further and the therapist feels a distinct stop in the available motion. These oscillations are maintained for up to 60 seconds, assuming the dog remains relaxed and allows this motion to occur. This mobilization can be followed with a physiologic motion to improve shoulder flexion, extension, or rotation. For example, the dog's shoulder should be passively moved through physiologic, or the normal range, of flexion. This may be followed with a movement of physiologic extension and then internal and external rotation. This technique is contraindicated if medial shoulder instability is present at the shoulder. A lateral distraction may cause pain and increase instability.

Box 26-1 Brief Overview of the Mechanics of the Shoulder Joint

- As the glenohumeral joint extends, the humeral head moves in a caudal direction. Therefore to increase extension, a caudal glide or mobilizing force should be applied.
- As the glenohumeral joint flexes, the humeral head moves in a cranial direction. Therefore to increase flexion, a cranial glide or mobilizing force should be applied.
- As the glenohumeral joint abducts, the humeral head moves in a medial and ventral direction. Therefore to increase abduction, a medial and ventral glide or mobilizing force should be applied.
- As the glenohumeral joint adducts, the humeral head moves in a lateral and dorsal direction. Therefore to increase adduction, a lateral and dorsal glide or mobilizing force should be applied.

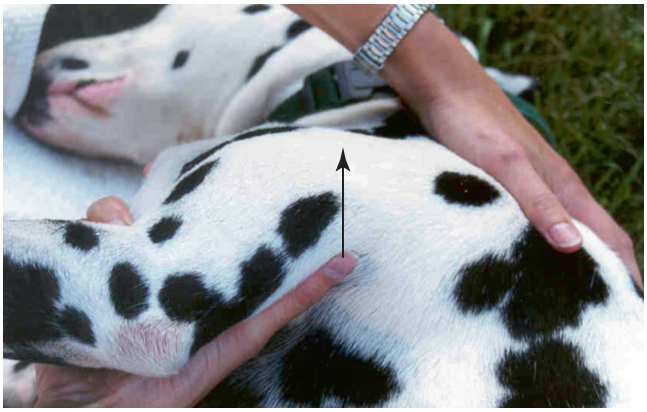


Figure 26-2 Lateral distraction of the glenohumeral joint. The arrow indicates the direction of the mobilization.

Caudal Mobilization: Accessory Glide (Figure 26-3)

Dog position: The dog is in lateral recumbency with the shoulder to be mobilized facing toward the therapist.

Therapist position: The therapist is cranioventral to the shoulder, facing the shoulder.

Hand position: The stabilizing hand is placed over the scapula; the dog's forelimb may rest on the forearm of the stabilizing hand. The thenar eminence or the web space of the mobilizing hand is placed over the greater tubercle of the humerus.

Mobilization: A gentle lateral distraction should be performed prior to the caudal glide to facilitate movement. A caudal glide is applied to move the humerus in relation to the scapula. This glide should be sustained 3 to 5 seconds.

Objective: Increase shoulder extension.

A grade III accessory mobilization may be performed from the same grips by adding a oscillating movements with the mobilizing hand. The caudal oscillations are performed with the joint held in the loose packed position, and the oscillations should bump against the end of the available joint motion. The oscillations are performed at a rate of three or four repetitions per second and are continued for up to 60 seconds. The caudal glide is typically followed with a grade III physiologic shoulder extension mobilization, oscillating for an additional 60 seconds.

Physiologic Shoulder Extension

To perform a grade III physiologic shoulder extension mobilization, the mobilizing hand and the stabilizing hand should exchange positions so that the therapist's mobilizing hand moves the humerus cranially into shoulder extension while the stabilizing hand holds the scapula stable.

Hand position: The mobilizing hand should grip the midshaft of the humerus.

Mobilization: With the scapula stabilized, the humerus is pushed into extension to the end of available ROM, bumping end feel of the joint. The humerus is moved back to near midposition of the joint. An oscillatory movement is performed by moving the humerus to end feel and back to midposition at a rate of three or four repetitions per second. The end feel should be felt by the therapist with each oscillation. The mobilization is performed for up to 60 seconds.

The dog's shoulder extension should be assessed for improvement in ROM. If motion is improved, the grade III accessory mobilization and the grade III physiologic mobilization can be repeated up to three times in one treatment session.

Cranial Mobilization: Accessory Glide (Figure 26-4)

Dog position: The dog is in lateral recumbency with the shoulder to be mobilized facing toward the therapist.

Therapist position: The therapist is on the ventral side of the dog, caudal to the forelimb.

Hand position: The stabilizing hand is placed over the scapula; the dog's forelimb may rest on the forearm of the stabilizing hand. The thenar eminence or web space of the mobilizing hand is placed on the caudal portion of the humerus as close to the joint as possible.

Mobilization: A cranial glide is applied to the humerus.

Objective: Increase shoulder flexion.

The cranial glide is typically followed with a grade III physiologic shoulder flexion mobilization, oscillating for an additional 60 seconds.



Figure 26-3 Caudal mobilization of the humerus to increase shoulder extension. The arrow indicates the direction of the mobilization.



Figure 26-4 Cranial mobilization of the humerus to increase shoulder flexion. The arrow indicates the direction of mobilization.

Physiologic Shoulder Flexion

To perform a grade III physiologic shoulder flexion mobilization, the mobilizing hand and the stabilizing hand should exchange positions so that the therapist's mobilizing hand moves the humerus caudally into shoulder flexion while the stabilizing hand holds the scapula stable.

Hand position: The mobilizing hand should grip the midshaft of the humerus.

Mobilization: With the scapula stabilized, the humerus is pushed into flexion to the end of available ROM, bumping end feel of the joint. The humerus is moved back to near midposition of the joint. An oscillatory movement is performed by moving the humerus to end feel and back to midposition at a rate of three or four repetitions per second. The end feel should be felt by the therapist with each oscillation. The mobilization is performed for up to 60 seconds.

The dog's shoulder flexion should be assessed for improvement in ROM. If motion is improved, the grade III accessory mobilization and the grade III physiologic mobilization can be repeated up to three times in one treatment session.

Other motions about the shoulder joint in the dog, including rotation, abduction, and circumduction, are important, but less crucial to the overall function of the dog. Both physiologic and accessory mobilizations to improve these motions can be performed, but are not included in this chapter.

The Elbow Joint

The elbow is a joint that is frequently problematic, with stiffness and pain secondary to degenerative joint disease. It is complex and consists of three joints—the humeroradial joint, the humeroulnar joint, and the radioulnar joint.[29] Clinically, stiffness in the elbow appears to respond well to distraction of the elbow joint. Pain and stiffness of the elbow may respond well to physiologic flexion and extension mobilizations. Full ROM of the elbow joint is necessary for proper function of the forelimb. Full elbow extension is necessary in the dog's normal gait cycle and a significant amount of flexion is necessary to engage in functional activities such as stair climbing, stepping over obstacles, and jumping.[31,32]

The proximal portion of the radius is positioned laterally at the elbow, and the distal portion of the radius is positioned medially at the carpus. The articulation between the radius and ulna allows approximately 45 degrees of rotational movement of the antebrachium. The articulation between the humerus and ulna provides the hinge motion responsible for elbow flexion and extension. The proximal ulna is positioned medially at the elbow. The trochlear notch is a deep, half moon–shaped concavity that faces cranially and articulates with the convex trochlea of the humerus. Therefore in an open kinetic chain, the concave ulna moves on the convex humerus.



Figure 26-5 Humeroulnar distraction for the elbow. The arrow indicates the direction of mobilization.

Humeroulnar Distraction (Figure 26-5)

Dog position: The dog is in lateral recumbency or in a sitting position with the elbow flexed to 90 degrees, and the radioulnar joint in slight supination. Approximately 45 degrees of flexion allows the anconeal process to move more freely in relation to the humerus without engaging the olecranon fossa.

Therapist position: The therapist is on the ventral side of the dog, facing the elbow to be mobilized.

Hand position: The stabilizing hand is located over the distal humerus. The mobilizing hand cradles the dog's forearm to maintain flexion and slight supination while grasping the proximal radius and ulna.

Mobilization: A longitudinal distraction is applied and maintained. In addition, the elbow may be moved into flexion as the distraction occurs.

Objective: Decrease joint stiffness for elbow flexion or extension.

Grade III physiologic elbow flexion or extension mobilizations may be performed following humeroulnar distraction, oscillating for an additional 60 seconds. Elbow ROM should be reassessed after the mobilization. If there is an improvement, three additional sets of the mobilization may be performed in a treatment session.

Physiologic Elbow Extension

Dog position: Lateral recumbency is preferred, although the mobilization may be performed while the dog is in a sitting position.

Therapist position: The therapist is ventral, facing the cranial aspect of the elbow to be mobilized.

Hand position: The stabilizing hand is wrapped around the distal portion of the humerus, placing the thenar and hypothenar eminences on the caudal aspect of the humerus, taking care to avoid the olecranon. The index

finger, third finger, and thumb of the mobilizing hand gently grasp the proximal radius and ulna.

Mobilization: To perform a grade III physiologic elbow extension mobilization, the therapist's mobilizing hand pushes the radius and ulna caudally into elbow extension while the stabilizing hand holds the distal humerus. With the humerus stabilized, the radius and ulna are pushed into extension to the end of available ROM, bumping the end feel of the joint. The radius and ulna are moved back to near midposition of the joint. An oscillatory movement is performed by moving the radius and ulna to end feel and back to midposition at a rate of three or four repetitions per second. End feel should be felt by the therapist with each oscillation. The mobilization is performed for up to 60 seconds.

Objective: Increase elbow extension.

Elbow ROM should be reassessed after the mobilization. If there is an improvement, three additional sets of the mobilization may be performed in a treatment session.

Physiologic Elbow Flexion

Dog position: Lateral recumbency is preferred, although the mobilization may be performed while the dog is in a sitting position.

Therapist position: The therapist is ventral to the elbow and faces the cranial aspect of the elbow to be mobilized.

Hand position: The stabilizing hand is placed on the cranial aspect of the distal humerus. The web space of the mobilizing hand is placed over the proximal radius and ulna.

Mobilization: To perform a grade III physiologic elbow flexion mobilization, the therapist's mobilizing hand pushes the radius and ulna cranially into elbow flexion while the stabilizing hand holds the distal humerus. With the humerus stabilized, the radius and ulna are pushed into flexion to the end of available ROM, bumping end feel of the joint. The radius and ulna are moved back to near midposition of the joint. An oscillatory movement is performed by moving the radius and ulna to end feel and back to midposition at a rate of three or four repetitions per second. End feel should be felt by the therapist with each oscillation. The mobilization is performed for up to 60 seconds.

Objective: To increase elbow flexion.

Elbow ROM should be reassessed after the mobilization. If there is an improvement, three additional sets of the mobilization may be performed in a treatment session.

The Carpus

Despite its intricacy, the carpus functions primarily as a hinge joint, permitting flexion and extension.[29] The main movement occurs at the antebrachiocarpal joint (Box 26-2), which consists of the distal radius and ulna, and the radial and ulnar carpal bones. The distal radius and ulna offer a concave surface for the convex radial and ulnar carpal bones to move within.

Box 26-2 Overview of Kinematics of the Antebrachiocarpal Joint

- As the antebrachiocarpal joint extends in an open kinetic chain, the convex articular surface of the radial and ulnar carpal bones moves in a caudal direction on the concave articular surface of the distal radius and ulna. Therefore to increase carpal extension, a caudal glide of the radial and ulnar carpal bones on the distal radius and ulna should be applied.
- As the joint extends in a closed kinetic chain, the concave surface of the radius and ulna moves in a cranial direction on the proximal row of carpals.
- As the antebrachiocarpal joint flexes in an open kinetic chain, the convex articular surface of the radial and ulnar carpal bones moves in a cranial direction on the concave articular surface of the distal radius and ulna. Therefore to increase carpal flexion, a cranial glide should be applied.
- As the joint flexes in a closed kinetic chain, the concave radius and ulna moves in a caudal direction on the convex surface of the proximal row of carpals



Figure 26-6 A caudal accessory glide of the antebrachiocarpal joint. The arrow indicates the direction of mobilization.

Caudal Glide of the Antebrachiocarpal Joint: Accessory Glide (Figure 26-6)

Dog position: The dog is either in lateral recumbency or in a sitting or sternal position.

Therapist position: The therapist is ventral, facing the carpus to be mobilized.

Hand position: The stabilizing hand is placed over the caudal surface of the distal radius and ulna, just proximal to the joint. The mobilizing hand is placed on the dorsal portion of the radial and ulnar carpal bones.

Mobilization: A caudal mobilization is applied by directing the radial and ulnar carpal bones in a palmar direction while stabilizing the distal radius and ulna. This glide should be held for 3 to 5 seconds.

Objective: Increase carpal extension.

A grade III accessory mobilization may be performed with the same hand position by adding an oscillation with the mobilizing hand. The caudal glide mobilizations are performed with the carpus held in a loose packed position, or slight flexion, and the caudal glide oscillations should bump against the end of the available motion. The oscillations are performed at a rate of three or four repetitions per second and are continued for up to 60 seconds. The accessory glide is typically followed by a grade III physiologic carpal extension mobilization for up to 60 seconds.

Physiologic Carpal Extension Mobilization

To perform a grade III physiologic carpal extension mobilization, the mobilizing hand is placed over the palmar portion of the radial and ulnar carpal bones and the stabilizing hand is placed on the cranial surface of the distal radius. The mobilizing hand pushes the distal radial and ulnar carpal bones dorsally into carpal extension. The distal radius and ulna are stabilized while the mobilizing hand moves the distal radial and ulnar carpal bones to the end of the available ROM and then back to the mid position of the joint. This oscillation between the available end range and the mid range should be performed at a rate of three to four oscillations per second for up to 60 seconds.

As always, the ROM should be assessed for an improvement. Further mobilizations may be repeated up to three times in one treatment session. Functional activities may then be prescribed to the dog to assist in maintaining the improved ROM, such as walking and wheelbarrowing, to encourage carpal extension while in a weight-bearing position.

Cranial Glide of the Antebrachiocarpal Joint (Figure 26-7)

Dog position: The dog is either in lateral recumbency or in a sitting or sternal position.

Therapist position: The therapist is ventral, facing the carpus to be mobilized.

Hand position: The stabilizing hand is placed over the cranial aspect of the distal radius and ulna. The mobilizing hand is placed on the palmar portion of the radial and ulnar carpal bones, just distal to the accessory carpal pad.

Mobilization: A cranial glide mobilization is applied by directing the radial and carpal bones dorsally while stabilizing the distal radius and ulna.

Objective: Increase carpal flexion.

A grade III accessory mobilization may be performed using the same hand positions by adding an oscillation of the mobilizing hand. The cranial glide mobilizations are performed with the joint held in a slight amount of flexion, or the loose packed position. The oscillations should bump against the end of the available motion. The oscillations are performed at a rate of three to four repetitions per second, and are continued for up to 60 seconds. The cranial glide is then typically followed with a grade III physiologic carpal flexion mobilization for an additional 60 seconds.



Figure 26-7 Cranial antebrachiocarpal glide. The arrow indicates the direction of mobilization.

Physiologic Carpal Flexion Mobilization

To perform a physiologic carpal flexion mobilization, the mobilizing hand is placed on the dorsal aspect of the radial and ulnar carpal bones and the stabilizing hand is placed on the palmar surface of the distal radius and ulna. With the distal radius and ulna stabilized, the mobilizing hand moves the radial and ulna carpal bones in a palmar direction into flexion. The radial and ulnar carpal bones are moved from the end feel of the joint back to the midposition of the joint at a rate of three or four repetitions per second for up to 60 seconds.

Carpal flexion should be assessed after the mobilization for an improvement. If the motion is improved, the grade III accessory and physiologic mobilizations may be repeated up to three times in one treatment session. Activities to be performed after mobilizations to maintain motion include high stepping over objects to encourage active flexion, or walking in water, sand, snow, or tall grass.

The Hip Joint

The hip or the coxofemoral joint is a ball-and-socket joint (Box 26-3). The convex-on-concave mobilization principles apply to this joint, similar to other joints.

Caudal Glide: Accessory Glide (Figure 26-8)

Dog position: The dog is in lateral recumbency with hip in slight abduction and slight flexion.

Box 26-3 Brief Overview of the Mechanics of the Hip Joint

- As the hip moves into flexion, the femoral head moves caudally with respect to the acetabulum. Therefore to increase hip flexion, a caudal glide is applied to the femoral head.
- As the hip moves into extension, the femoral head moves cranially with respect to the acetabulum. Therefore to increase hip extension, a cranial glide is applied to the femoral head.
- As the hip moves into abduction, the femoral head moves medially and ventrally in a rolling manner with respect to the acetabulum. Therefore to increase hip abduction, a medial and ventral glide is applied to the femoral head.
- As the hip moves into adduction, the femoral head moves laterally and dorsally in a rolling manner with respect to the acetabulum. Therefore to increase adduction, a lateral and dorsal glide is applied to the femoral head.



Figure 26-8 Caudal glide to increase hip flexion. The arrow indicates the direction of mobilization.

Therapist position: The therapist faces the hip to be mobilized on the ventral surface.

Hand position: The stabilizing hand is placed over the sacrum. The forearm of the mobilizing hand may be placed under the hindlimb to assist in guiding the limb. The web space of the mobilizing hand is placed at the cranial portion of the proximal femur, as close to the joint line as possible.

Mobilization: A caudal glide is applied with the mobilizing hand. The forearm may assist the hindlimb into flexion as the guide is applied.

Objective: Increase hip flexion.

A grade III accessory mobilization may be performed from the same described hand position by adding an oscillation of the mobilizing hand. The caudal glide oscillations are performed with the joint held in slight flexion and slight abduction, or the loose packed position. The oscillations should bump against the end of the available motion at a rate of three or four repetitions per second and be continued for up to 60 seconds. The caudal glide is typically followed by a grade III physiologic hip flexion mobilization for an additional 60 seconds.



Figure 26-9 Physiologic hip flexion. The arrow indicates the direction of mobilization.

Physiologic Hip Flexion (Figure 26-9)

To perform a grade III physiologic hip flexion mobilization, the mobilizing hand should be moved to the caudal portion of the proximal femur, just below the ischial tuberosity. The stabilizing hand should remain on the sacrum and cradle the distal portion of the hindlimb if possible. With the pelvis stabilized, the femur is moved to the end of the available ROM and then back to near midposition of the joint. An oscillatory movement is performed by moving the femur to end feel and back to midposition at a rate of two to three repetitions per second for up to 60 seconds. With the physiologic mobilization, it is important that the therapist move the limb with his or her body instead of with arm movements only because this is a larger joint and requires proper body mechanics to avoid injury to the therapist.

Hip flexion should then be assessed after the mobilization to determine if there has been an improvement. If the motion is improved, the grade III accessory mobilization and the grade III physiologic mobilization may be repeated up to three times in one treatment session. Active movement after treatment includes high stepping, swimming, or walking in deep water to encourage active hip flexion.



Figure 26-10 Cranial mobilization to increase hip extension. The arrow indicates the direction of mobilization.



Figure 26-11 Physiologic hip extension. The arrow indicates the direction of mobilization.

Cranial Glide: Accessory Glide (Figure 26-10)

Dog position: The dog is in lateral recumbency with hip in slight abduction and slight flexion.

Therapist position: The therapist is caudal and ventral to the dog, facing the hip to be mobilized.

Hand position: One hand may be placed at the distal femur to assist with control or placed at the sacroiliac region. Because this is a large joint to mobilize with a long lever arm, it is often easiest to place the hand not performing the mobilization at the distal femur. The pelvis is stabilized by the dog's weight in a lateral recumbent position. The forearm of the stabilizing hand may be used to assist the positioning of the hindlimb. The web space of the mobilizing hand is placed under the ischial tuberosity, on the proximal and caudal aspect of the femur.

Mobilization: A cranial mobilization is applied by directing the proximal femur cranially, with a slight dorsal angulation.

Objective: Increase hip extension.

A grade III accessory mobilization may be performed with the same hand positions as described previously. The cranial glide oscillation is performed with the hip in a loose packed position, or in slight flexion and abduction. The oscillations should bump up against the end of the available motion. The oscillations are performed at a rate of three to four repetitions per second and are continued for up to 60 seconds. The cranial glide of the femur is typically followed by a grade III physiologic hip extension mobilization for an additional 60 seconds.

Physiologic Hip Extension (Figure 26-11)

To perform the physiologic mobilization for hip extension, the mobilizing hand should be placed on the cranial and most proximal portion of the femur and the stabilizing hand should remain on the sacrum. The mobilizing hand applies a caudal force to the proximal femur to encourage hip extension. With the pelvis stabilized, the proximal femur is pushed into the available amount of extension, bumping the end feel of the joint. The femur is then moved back to the near midposition of the joint. Oscillatory movements are performed by moving the femur to the end feel and back to midposition at a rate of three to four repetitions per second for 60 seconds.

Hip extension should then be assessed after the mobilization for an improvement in ROM. If the motion is improved, the grade III accessory and physiologic mobilization should be repeated up to three times in one treatment session. Active hip extension should also be encouraged through resisted walking by having the dog pull against a leash or climbing stairs.

The Stifle Joint

The femoral condyles are a convex surface that articulate with the concave tibial plateau.[29] The stifle is a ginglymus joint, or a hinge joint, and flexion and extension are the primary motions at the joint (Box 26-4). The concave tibia moves on the convex femur and is therefore following the concave on convex rule. Therefore with stifle extension, the tibia glides cranially on the femur. With stifle flexion, the tibia glides caudally on the femur.

Restoration of both stifle flexion and extension is essential to normal ambulation and function in the dog. The dog requires full stifle flexion to obtain a normal sitting posture and full stifle extension for proper ambulation.

Femorotibial Cranial Glide (Figure 26-12)

Dog position: The dog is in lateral recumbency with the stifle in a resting position.

Therapist position: The therapist faces the ventral aspect of the dog and the stifle to be mobilized.

Box 26-4 Overview of Kinematics of the Stifle Joint

- As the stifle joint flexes in the open kinetic chain, the concave plateau of the tibia moves in a caudal direction on the convex femoral condyles. Therefore to increase stifle flexion, a cranial glide mobilization of the proximal tibia on the femur should be applied.
- As the stifle flexes in the closed kinetic chain, the convex femoral condyles roll caudally on the concave tibial plateaus.
- As the stifle joint extends in the open kinetic chain, the concave plateau of the tibia moves in a cranial direction on the convex femoral condyles. Therefore to increase stifle extension, a caudal glide mobilization of the proximal tibia should be applied.
- As the stifle extends in the closed kinetic chain, the convex femoral condyles roll cranially and glide caudally on the concave tibial plateaus.



Figure 26-12 Cranial glide to increase stifle flexion. The arrow indicates the direction of mobilization.

Hand position: The stabilizing hand is on the cranial surface of the distal femur. The mobilizing hand is placed on the caudal surface of the proximal tibia.

Mobilization: A cranial glide is applied to the tibia while the distal femur is stabilized.

Objective: Increase stifle joint flexion.

A grade III accessory mobilization may be performed from the same described hand position by adding an oscillation of the mobilizing hand. The cranial oscillations are performed with the joint held in slight flexion or the loose packed position. The oscillations should bump against the end of the available motion at a rate of three to four repetitions per second for up to 60 seconds. The cranial glide is typically followed by a grade III physiologic stifle flexion mobilization for an additional 60 seconds.

Because there is virtually no cranial or caudal glide motion in the normal stifle joint with intact cranial and caudal cruciate ligaments, the value of cranial glide and accessory joint mobilizations in the stable joint are uncertain.



Figure 26-13 Physiologic stifle flexion. The arrow indicates the direction of mobilization.

Physiologic Stifle Flexion (Figure 26-13)

To perform a grade III physiologic stifle flexion mobilization, the hand positions may remain the same or the mobilizing hand may be placed on the cranial aspect of the distal tibia while the stabilizing hand is placed on the caudal aspect of the distal femur. With the femur stabilized, the tibia is moved to the end of the available range of flexion and then back to near midposition of the joint. An oscillatory movement is performed by moving the tibia to end feel and back to midposition at a rate of three or four repetitions per second for up to 60 seconds.

Stifle flexion should then be assessed after the mobilization to ascertain if there has been an improvement. If the motion is improved, the grade III accessory mobilization and the grade III physiologic mobilization may be repeated up to three times in one treatment session. Active movement after treatment includes high stepping, sit-to-stand exercises, and swimming to encourage active stifle flexion.

Femorotibial Caudal Glide (Figure 26-14)

Dog position: The dog is in lateral recumbency with the stifle in a resting position.

Therapist position: The therapist faces the caudal aspect of the stifle to be mobilized.

Hand position: The stabilizing hand is placed on the caudal aspect of the distal femur. The mobilizing hand is placed on the cranial aspect of the proximal tibia.



Figure 26-14 Caudal glide to increase stifle extension. The arrow indicates the direction of mobilization.

Mobilization: A caudal glide is performed by the mobilizing hand while the distal femur is stabilized.
Objective: Increase stifle joint extension.

A grade III accessory mobilization may be performed from the same described hand position by adding an oscillation of the mobilizing hand. The caudal glide oscillations are performed with the joint held in slight flexion or the loose packed position. The oscillations should bump against the end of the available motion at a rate of three or four repetitions per second for up to 60 seconds. The caudal glide is typically followed by a grade III physiologic stifle extension mobilization for an additional 60 seconds.

Because there is virtually no cranial or caudal glide motion in the normal stifle joint with intact cranial and caudal cruciate ligaments, the value of caudal glide and accessory joint mobilizations in the stable joint are uncertain.

Physiologic Stifle Extension (Figure 26-15)

To perform a grade III physiologic stifle extension mobilization, the hand positions may remain the same, or the mobilizing hand may be placed on the caudal aspect of the distal tibia while the stabilizing hand is placed on the cranial aspect of the distal femur. With the femur stabilized, the tibia is moved to the end of the available motion of extension and then back to near midposition of the joint. An oscillatory movement is performed by moving the tibia to end feel and back to mid-position at a rate of three to four repetitions per second for up to 60 seconds.

Stifle extension should then be assessed after the mobilization to ascertain if there has been an improvement. If the motion is improved, the grade III accessory mobilization and the grade III physiologic mobilization may be repeated up to three times in one treatment session.



Figure 26-15 Physiologic stifle extension. The arrow indicates the direction of mobilization.

The Spine

The spinal region of the dog is complex in that it allows a significant amount of movement as it absorbs and distributes the forces from the four limbs. Each area of the spine (cervical, thoracic, lumbar, and coccygeal) possesses varying amounts of motion. The cervical region permits enough movement so that the dog may be able to touch any part of its body with its nose except between the scapulas. Observation of a running dog demonstrates the amount of thoracic and lumbar flexion and extension that may be present.[29]

Movements of the spine include extension, flexion, side flexion, and rotation. The motions of each component of the spine contribute to the total movement of the entire spine. The movement at the spinal level takes place between two vertebrae. Some regions of the spine demonstrate more mobility than other areas. For example, the thoracolumbar and the lumbosacral regions are two of the most mobile areas of the spine. These are also the areas in which there are a significant number of issues.

Mobilization techniques of the canine spine must be approached with extreme caution and knowledge of canine anatomy, medical conditions of the nervous system, the diagnosis, and the anatomy of the individual breed of dog. For example, the angulation of the thoracic spine differs according to the breed of the dog and the topline.[29,33] A Labrador retriever has a level topline. This differs from the arched topline of a greyhound or a whippet.[29,34] The differences in angulation of toplines alters the direction of the mobilization.

Because specialized training and clinical experience are necessary to safely perform spinal mobilizations, a limited number of basic mobilization techniques for the thoracic and lumbar regions are described. The cervical region is very complex and there are many contraindications associated with cervical mobilizations. It is essential that mobilization not be attempted without a clear comprehension of cervical anatomy, the nature of the dog's problem, and the contraindications of the region. Cervical ROM may become reduced secondary to arthritic conditions of the cervical spine, after surgery for a cervical disc, or conditions related to overuse of the forelimb muscles. It is common to see restrictions in the adjacent vertebrae after a ventral slot procedure. Changes in functional characteristics may result in decreased cervical extension while walking, lifting the head up from drinking or eating from a bowl on the floor, or flexing the head and neck as the dog ascends stairs. It is important to wait until after the surgical site has healed and is stable prior to performing any manual therapy techniques.

Spinal mobilizations are applied in the dog when there is loss of motion related to pain or dysfunction. For example, a decrease in passive thoracic dorsal to ventral movements produces decreased thoracic extension. This results in a relative lack of active thoracic extension—the dog will not be able to "dance" backward or jump up onto a piece of furniture. Of course, the spinal area must be identified as the problem and other areas of concern should be ruled out. For example, the practitioner should assess if hip disease or lumbar disease is restricting extension.

Thoracic Region

The dorsal spinous processes of the thoracic spine are very narrow and long. Some dogs object to direct pressure over the spinous process. The lower thoracic region is one of the more common areas where problems secondary to increased mobility occur. Intervertebral disc disease, compensatory problems from canine hip dysplasia, and arthritic changes are common in this area. As described earlier, dogs place more weight on the forelimbs with hip and stifle disease. From a clinical point of view, the thoracic area often exhibits restrictions as a result of these compensations. Dogs experiencing signs of hip dysplasia have decreased hip extension. Changes in the motion of the spinal column occur to compensate for the reduced extension.

There are thirteen thoracic vertebrae in the dog. The eleventh vertebra is called the *anticlinal vertebrae*.[35] It is a transition point along the thoracic vertebrae. The spinous processes cranial to T11 point caudally, and the spinous processes caudal to T11 point cranially. Therefore when performing mobilizations to this area, it is essential to understand the topline of the dog as well as the inclination of the particular vertebral spinous process.



Figure 26-16 Dorsal to ventral glide to thoracic spine. The arrow indicates the direction of mobilization.

Thoracic Central Dorsal to Ventral Glides (Figure 26-16)

Dog position: The dog is standing or in a sitting position with the spine in neutral, with no rotation or side flexion.

Therapist position: The therapist should stand or kneel at the dog's side, with the shoulders directly over the dog. Kneeling may be necessary if the dog is on the floor or a mat.

Hand position: The thumb of the mobilizing hand is placed over the spinous process of the vertebra to be mobilized. The remaining part of the mobilizing hand may be draped over the region. The stabilizing hand may be placed under the ventral position of the thoracic region. This hand helps to detect any changes in tension of the abdominal region in response to pain or discomfort as well as provide light counterforce stabilization. The therapist's elbows should be slightly flexed to facilitate a smooth oscillation of the vertebra.

Mobilization: Depending on the angulation of the thoracic region of the dog, a dorsal-to-ventral glide is applied at an angle from approximately 90 to 75 degrees. Oscillations for a grade II mobilization should be performed very gently so that the therapist senses only minimal resistance from joint structures and the dog does not experience any pain. Grade II mobilizations should be performed for up to 30 seconds. Oscillations for grade III mobilizations should be performed so that the therapist senses the end feel of the segment. This grade of mobilization should be performed for up to 60 seconds. After each mobilization, the dog should be assessed to determine whether there has been a functional change. Changes that may occur include an increase in functional mobility, such as with side flexion of the spine while the dog lies down or reaches for a treat near the base of the tail. Additional changes in thoracic mobility include the ability to ascend stairs with improved



Figure 26-17 Dorsal to ventral glide to lumbar spine. The arrow indicates the direction of mobilization.

extension. Pain and muscle spasms should also be diminished.

Lumbar Region

The seven lumbar vertebrae possess blunt spinous processes and the facet angles are positioned at an approximate 90-degree angle to the floor when the dog is in a standing position. Therefore mobilizations will mostly be performed at a 90-degree angle or perpendicular to the floor.

The areas of greatest mobility in the lumbar spine include the thoracolumbar region and the lumbosacral region.[29,30] This is also an area where intervertebral disk injuries are commonly seen.[36]

Lumbar Dorsal to Ventral Glide (Figure 26-17)

Dog position: The dog stands with the spine in a neutral position.

Therapist position: The therapist stands or kneels directly over the area to be mobilized. The therapist's shoulders should be directly over the area to be mobilized.

Hand position: The dominant thumb should be placed directly over the spinous process to be mobilized and then reinforced by the nondominant thumb. The remainder of the hand should be draped over the dog. If the therapist feels comfortable with performing the mobilization with one hand, the nondominant hand may be placed under the dog's abdomen to ascertain any signs of pain or discomfort. The shoulders should be directly over the area and the elbows and wrists slightly flexed to provide proper body mechanics.

Mobilization: A dorsal to ventral glide is applied at approximately 90 degrees or perpendicular to the skin surface. Oscillations for a grade II mobilization should be performed very gently so that the therapist senses only minimal resistance from joint structures and the dog does not experience any pain. Grade II mobilizations should be performed for up to 30 seconds. Oscillations for grade III mobilizations should be performed so that the therapist senses the end feel for the segment. This grade of mobilization should be performed for up to 60 seconds. After each mobilization, the dog should be assessed to determine whether there has been a functional change. If an increase in ROM and function has been made, the mobilization may be repeated two more times. Functional improvements include an improved ability to ascend stairs, transition from a lying position to a sitting position, and possibly jumping. A decrease in pain and muscle spasms should be felt, as well as increased comfort during palpation. Often, dogs contract their abdominal muscles when pressure is placed on a painful intervertebral segment.

Acknowledgments

Photographs by Shauna Miner and Brenda Walker. Thank you to Ann and Champion Majessa Easy Rider "Tommy" Fischer, Latte, Edsel, Cheryl, Max Brienza, and Fred for their photographs.

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27 Massage

Amanda Sutton and Dana Whitlock

Biologic Basis

Massage has many definitions and is employed by many practitioners of various backgrounds. The working definition used in this chapter is that massage is the manipulation of the soft tissues of the body. The use of massage has been well received in the human field and, despite a paucity of research, it has continued to be developed and explored.

The word *massage* is derived from the Arabic word *mass,* which means "to press." Many ancient civilizations developed a system of massage, which was used for various medical conditions. Just Lucas Championnies (1843-1913) used it in the management of fractures in the United States, and it was advanced by Mary McMillan, director of physical therapy at Harvard Medical School. Dr. JB Mennell described physical treatment by movement, manipulation, and massage, which was first published in 1917. There has been variable interest in massage by medical establishments in the twentieth century, but interest in complementary medicine has grown in the United States. The American Massage Therapy Association was founded in 1943 and had grown to more than 58,000 members by 2009.

Massage is now incorporated along with the medical management of human patients for pain relief and cases of decreased mobility. It is used as a prevention measure and in the management of competitive athletes. It is also incorporated into most chiropractic, physical therapy, and osteopathic manual therapy approaches.

Biomechanics of Connective Tissue

Massage must be directed toward a specific purpose and should be aimed at promoting physical and psychological change.

The main constituent of connective tissue is collagen. Its function is to resist axial tension, and it exhibits a stress-strain behavior (Box 27-1). Microscopically, collagen fibers are arranged in bundles and have a crimped appearance. Type I collagen is in the dermis and fascia and gives support and resistance to tension.

The basal lamina of epithelial tissue contains type IV collagen, which supports the epithelial cells. Hydrogen cross-links are formed between chains and between molecules, giving stability at fibril levels, assisting in the formation of collagen fibers. They allow the tissue to function under mechanical stress.

The orientation of the fibers depends on the stresses to which the fibers are subjected; connective tissue must be pliable, yet very strong. A stress-strain curve demonstrates the behavior of biologic materials. When a longitudinal stress is applied to collagen, the tissue responds by elongation, which occurs in the toe region of the curve (Figure 27-1). Elongation occurs as a result of straightening of the crimped fibers, and probably also as a result of some interfibrillar sliding and shear of ground substance, which flows between the collagen fibers.

Massage produces most of its effects in the toe region of the stress-strain curve. Care should always be taken to not overstretch the tissues and to avoid damage to the internal structure of connective tissue fibers. Therefore the end feel of the tissues must be understood to avoid damage to connective tissues.

Permanent deformation is termed *plastic deformation,* which occurs with microfailure and disruption of collagen cross-links. The undamaged fibers absorb a greater proportion of the load as a new length is established, which reflects the balance between elastic recoil and the resistance of the water and glycosaminoglycans to compression.

Biologic materials also have viscosity, a property of fluids of resistance to flow, and elasticity, a property of solids. Together, these result in the viscoelastic properties of tissues. The response of tissues to loading depends on how quickly the load is applied or removed. The faster the repetitive loading and unloading, the stiffer the material behaves. With rapid loading, friction results and tissue temperature rises. Energy is dissipated as heat when the tissue is returned to its original length. This is referred to as *hysteresis.*

This is relevant in the application of massage because it is important to be sensitive to the stiffness of tissues. If a low constant or repetitive load is maintained over a long period, the elongation of tissues that occurs is by creep. Conversely, if the tissue length is held constant, elongation occurs by relaxation. Massage produces low, repetitive loads over the medium term and may cause a nonpermanent creep response. Rapid loading of tissues during

Box 27-1 Characteristics of Stress-Strain Behavior of Collagen

Stress and strain are linear up to the yield point. Elongation along this portion of the curve will not be permanent.

Elastic limit is the point to which maximum stress may be applied without permanent deformation.

Increased loading requires greater force to increase elongation of collagen because the tissue becomes stiffer.

Microfailure begins to occur in some of the fiber bundles in the linear region. However, the tissue retains its external appearance of continuity.

The end of the linear region is known as the *yield point of the tissue.* At this point, elongation of collagen occurs with a minimal increase in load. Beyond this point, major fiber bundle failure occurs, the external appearance changes, and the smooth outline is lost.

When maximum load point is reached (P_{max}), complete rupture occurs.

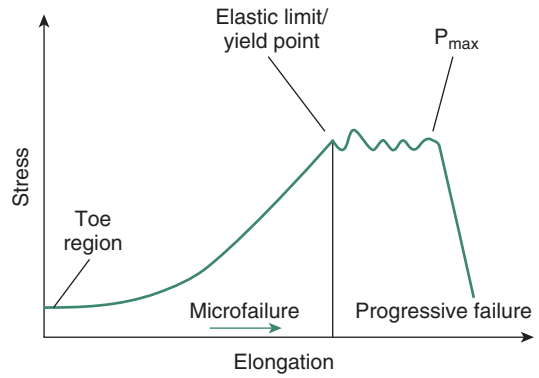


Figure 27-1 Typical stress-strain curve for connective tissue. Stress, Force applied per unit area; strain, proportional elongation; P_{max}, maximum load point.

massage is undesirable because of increased tissue stiffness as a result of their viscoelastic properties. Also, dry tissue results in less compliance and elongates less readily. These are important facts for deciding the rate of massage application.

Lymphatics

In humans, one tenth of the tissue fluid is removed by the lymphatics. Understanding lymphatic anatomy, physiology, and function is necessary for successful massage. The rate of flow is determined by the interstitial fluid pressure and lymphatic pump activity.

Increased lymphatic flow is caused by the following:

- Increased capillary pressure
- Increased plasma osmotic pressure
- Increased interstitial fluid pressure
- Increased capillary permeability

Massage has been shown to increase lymph flow rate by 22-fold, lymph colloid concentration by 47 times, and leukocyte counts by 8.5 times.[1] It is believed that when tissues and fluid are compressed against the outer surfaces of the larger lymph vessels, lymph flow is impeded. This results in an increase in interstitial fluid pressure slightly above atmospheric pressure. Massaging may increase interstitial fluid pressure and help to move fluid into the lymphatic system. By massaging in a distal to proximal direction, it is proposed that fluid is moved from the extremity toward the central body core. This supports the practice of applying massage in a distal to proximal direction. Other controlled studies show that massage does not improve lymph drainage in certain situations, such as cancer. A study of women with breast cancer–related lymphedema evaluated the effect of compression garments with or without manual lymph drainage (massage). It was concluded that compression garments improved lymphedema, but there was no additional benefit of massage in relieving limb edema in women with breast cancer.[2]

Circulatory Effects

The pressure of the massage itself increases pressure within the tissues. Pressure gradients are created between the tissue spaces and vessels. As the hands are moved, changes in tissue pressure occur, creating fluctuating pressure differences between one area of tissue and another. It is believed that fluid moves constantly from tissues to vessels and back again, as it flows from areas of high pressure to areas of low pressure.

During an actual massage session, various massage manipulations are employed that are complex and involve a combination of squeezing, stretching, pulling, and traction forces, with movement occurring in different directions and in different tissue planes. Therefore there will be complex repetitive pressure changes occurring in varying directions and at different tissue depths. This is likely to have an effect on fluid interchange, whereby fluid is pushed from the tissue spaces into the vessels, toward the lymph nodes and heart, and new fluid is pushed or drawn into the spaces. Massage is believed to replenish the fluid in the spaces, producing a flushing effect, which brings in additional nutrients. There is evidence in humans that chemical irritants in the tissues (e.g., substance P, prostaglandins) and waste products of metabolism may decrease pain threshold by sensitizing free nerve endings. Replenishing tissue fluid and removing inflammatory products may reduce this effect, preventing or reducing some types of chronic pain. By removing metabolites and chemicals from muscles and "flushing" the muscle tissue with new circulation, muscle soreness following exercise may be reduced. However, controlled studies have not been able to demonstrate that massage reduces metabolites following exercise.[3,4] Studies involving Doppler ultrasound have failed to demonstrate an effect of massage on muscle blood flow.[5,6]

Tissue Movement

As the hands move along the superficial tissues, pressure to the tissues increases, and layers of tissue are moved. A light glide causes movement of the epidermis. If friction is maintained between the therapist's hand and the patient's skin, the epidermis moves with the hand and is gently stretched. There will also be some movement of the dermis because of the traction between the two layers. More pressure with friction, although still very light, results in traction between the dermis and the subcutaneous tissues. The end-feel of the stroke is when this traction reaches its limit and all layers are stretched.

Massage involves the interaction of energy between the person applying the massage and the dog, and the effects of touch to induce relaxation, communication, and a sense of general well-being. It may also produce movement of the tissues in subsequent layers as a result of traction of the tissue interfaces. Sensory and autonomic nerves may be stimulated, inducing changes in the nervous and circulatory systems, and movement may be affected in abnormal tissue, such as scar tissue or layers of adherent tissues. Reed and Held[7] found no consistent systemic effect on autonomic nervous system parameters either acutely or cumulatively when comparing connective tissue massage and a placebo treatment (sham ultrasound) in healthy middle-aged and geriatric humans.

Therapeutic Effects

Massage is generally enjoyed by small animal patients and helps to relieve distress, anxiety, and discomfort. There are many postulated effects of massage reported in the literature. Unfortunately, many of these have not been substantiated by systematic scientific investigations.

Stroking young animals results in a reduction in the animal's physiologic response to stress, demonstrated by a decreased output of adrenocorticotropic hormone.[8] Young animals that are handled also show greater development of the cortex and the subcortex of the brain. They learn faster and have a more advanced stage of neural development than nonhandled animals.[9] Resistance to infection later in life may also be beneficially influenced by cutaneous stimulation experienced by the infant animal.[10]

If a hand is placed over the surface of a dog's skin, heat is felt between the two surfaces. If the hand is placed on the skin and held still, this heat increases. Rubbing over the surface of the skin causes friction, and this results in an even greater increase in heat. Heat is a form of energy, and some schools of massage, particularly those grounded in Eastern practices, use the energy field that exists around the body. This is sometimes referred to as an *electromagnetic radiation* around the entire body. It is felt as heat and can be felt a small distance from the body. Some also believe that other forms of energy fields run through the body, along specific pathways known as *meridians*. These are used in acupuncture and reflex therapy treatments.

Most of the research on the effects of massage has been done in humans. Most studies have evaluated healthy individuals, which may be misleading because physiologic compensatory mechanisms are extremely efficient in healthy tissues and any alterations in local blood flow may be compensated for by autoregulatory processes. Most of the research studies have also been performed on small groups and often without control groups. One study by Hansen and Kirstensen[11] gave little detail concerning the massage itself, such as the amount of pressure applied. Although the research supported the concept that massage increases blood flow in muscles, the lack of information regarding the type of massage and length of treatment make interpretation of the results difficult.

Changes in blood constituents following massage have provided information regarding the mechanism of action of massage.[12] Ernst[13] found that a standard 20-minute massage treatment reduced the hematocrit and blood and plasma viscosity. It is believed that the fluid immediately surrounding poorly perfused vessels has low viscosity because of decreased cellularity and that the vasodilatation caused by massage aids in the recruitment of dormant vessels. It is desirable to increase local circulation to promote healing. The mechanical effect of the massage helps to remove low-viscosity tissue fluid into the circulation. The changes in blood detected within the vessels in this study support the belief that massage produces a flushing and mechanical effect on the circulation. In a more recent study, Hinds and colleagues[6] showed no difference in femoral arterial blood flow, quadriceps muscle temperature, blood lactate levels, heart rate, or blood pressure when comparing post quadriceps exercise and massage to exercise and rest. They did show that skin blood flow and skin temperature were both significantly elevated in the massage group. They hypothesized that this potential diversion of blood flow away from the muscle could have a deleterious effect to recovering muscle tissue. They did not demonstrate a decrease in muscle blood flow, however, so this hypothesis is not supported by their work.

In a Cochrane database review, Furlan and colleagues[14] critiqued 13 randomized trials and concluded that massage might be beneficial in the treatment of low back pain in acute and chronic cases, especially when combined with exercise and education.

Effects on Muscle

Massage may also affect skeletal muscles. Muscles have a natural resting tone that may be increased in postural muscles by external influences, such as cold or stress. This occurs because of the interaction of the muscle spindle and the central nervous system. Stretching a muscle may stimulate the muscle spindle and cause a reflex muscle contraction while reflex inhibition of the antagonist occurs.

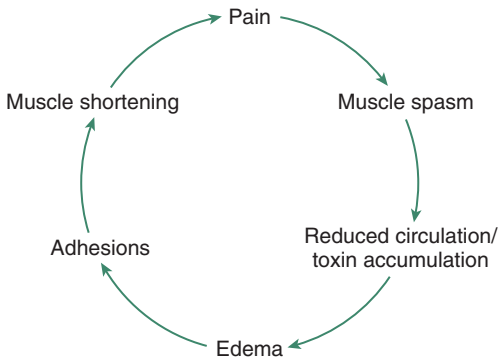


Figure 27-2 Self-perpetuating muscle spasm cycle.

Massage may add an external stimulus to sensory organs and either increases muscle tone by stimulation or reduces it, probably by facilitating an accommodation of the spindle, causing it to reset at a lower threshold of excitability.

It is believed that the circulatory effect of massage may reduce muscle soreness and therefore aid clinical signs associated with muscle injury or postexercise recovery. Danneskiold-Samsoe and colleagues[15] studied the effects of massage for shoulder and back pain in humans. Blood samples were evaluated for myoglobin concentrations. There was a statistically significant effect, with peak myoglobin concentrations occurring within 3 hours of the massage. Myoglobin concentrations gradually decreased; after seven treatments, there was no difference. In a systematic review of the literature published in 1998, Ernst[16] concluded that most studies evaluating the effect of massage on delayed-onset muscle soreness were methodologically flawed, findings were inconsistent between studies, and, although there appeared to be some potential for a positive effect, the evidence was not convincing and warranted further controlled investigation.

Jacobs[17] advocated the use of massage for the relief of spasm of unknown cause but which resulted in a self-perpetuating muscle spasm cycle (Figure 27-2). Massage is also said to reduce tone in muscles that are tense or in spasm and to reduce the soreness and tenderness of exercised muscles. It is also used to prepare muscles for exercise. Hernandez-Reif and colleagues[18] found that children with cerebral palsy who received 30 minutes of massage twice weekly for 12 weeks had decreased muscle spasticity of the arms, decreased arm muscle and overall tone, increased hip extension range of motion (ROM), and improved fine and gross motor function when compared with children with cerebral palsy who received 30 minutes of reading twice weekly.

Jones and Round[19] discussed the process by which an analgesic substance, possibly potassium, is released when muscles maintain excessive tone or static contraction for prolonged periods. Eccentric exercise of muscles, which generates high forces in muscles, produces delayed-onset soreness and a feeling of stiffness. Jones and Round[19] explained this by identifying swelling in muscles and inflammation of the connective tissue. Massage may help reduce or prevent the initial tenderness. It is also believed that massage may help to prevent subsequent postexercise pain. Regular massages may make the muscles softer and more pliable, and the stretching manipulations may increase extensibility and strength in the connective tissues.[15] Willems and colleagues[20] demonstrated decreased muscle soreness in portions of the quadriceps muscle (vastus lateralis and rectus femoris), but not in the vastus medialis after massage following eccentric exercise–induced muscle soreness. They also demonstrated improved vertical jumping performance in the massaged leg 48 hours after massage when compared with the nonmassaged leg. Mancinelli and colleagues[21] showed significant effects of massage versus control in vertical jump displacement, perceived muscle soreness, and algometer muscle pain readings, but not timed shuttle run in women collegiate athletes.

Pain experienced after exercising with the muscles in a lengthened position may also be due to inflammation of the connective tissue rather than muscle damage. Massage may assist by increasing pliability in the connective tissue around the muscle fasciculi. Massage assists mobility between the interfaces within the muscle and also stretches any fibrous adhesions or scarring in the tissue.

The effects of massage on the Hoffman (H) reflex, which indicates the excitability in α-motoneurons or anterior horn cells, has also generated interest. Goldberg and colleagues[22] studied the effects of massage on healthy people and patients with spinal cord injuries. They applied one-handed petrissage to deep and superficial tissues. There was a 49% reduction in H reflex activity with deep massage, and a 39% reduction with light massage. From this study, it was concluded that stimulating cutaneous mechanoreceptors and pressure receptors reduces H reflex activity. Sullivan and colleagues[23] also demonstrated a reduction in α-motoneuron excitability of massaged muscles.

Effects on Pain and Sensation

The effects of massage on connective tissues, which helps to reduce muscle spasm and soft tissue discomfort, have already been discussed. However, Ueda and colleagues[24] demonstrated that postsurgical massage may facilitate the regression of sensory analgesia following an epidural block.

Carreck[25] evaluated the effect of massage on levels of pain perception in humans. She found that pain threshold was raised to significantly higher levels in the massage group and this work suggests that massage may be used to help manage pain and reduce treatment soreness.

Massage may cause traction at tissue interfaces. Horizontal plexi lie at interfaces in the tissues and gentle pulling on these vessels may stimulate the accompanying

sympathetic nerves, which supply the mechanoreceptors. These receptors may be distorted by the massage, possibly lowering mechanoreceptor sensitivity and reducing pain and tenderness. If delayed-onset pain in the muscle is caused by connective tissue inflammation, the flushing effect in the surrounding fluids and removal of inflammatory mediators may increase the speed at which the inflammation resolves.

Effects of Massage on Connective Tissue

Following injury or disease, inflammation causes increased vascular permeability. The resulting edema contains fibrinogen, which may result in formation of fibrous tissue. Adhesions form within the tissues, binding tissue interfaces. These adhesions prevent normal sliding of tissues on each other and also reduce the ability of the fibers to spread apart. *Massage* is described as being able to stretch tissues that have become short, tight, or adhered. Petrissage massage stretches and pulls the tissues in various directions and mobilizes the adjacent connective tissue fibrils. The molecular cross-bridges between fibrils may be influenced and plastic changes may occur; their length may be maintained or increased because of elongation or creep. Mobility may also be increased at the fibrous cross-bridges between fibers and areas of adhesions by stretching connections between the fibers. This facilitates spreading and sliding at tissue interfaces, and longitudinal elongation helps to increase flexibility.

Following surgery, scars may form with adhesions of various tissue layers to the underlying tissues. Massage helps mobilize and soften adhesions by elongation, creep, and increased fluid exchange of tissues. Collagen fibers align themselves along lines of stress. Techniques that stretch the fibers and adhesions in different directions eventually restore their mobility, promoting remodeling along the lines of normal stresses.

Following injury, it is important to maintain local flexibility of the involved structures to assist overall suppleness and to reduce the possibility of reinjury resulting from decreased extensibility of normal and scar tissues. Massage should be used to promote the functional recovery of structures and prevent reinjury by maintaining mobility at the interfaces, both inside and outside the affected structures. Massage should be combined with controlled longitudinal stretching exercises.

One technique to mobilize scar tissue is deep-friction massage.[26] It is applied at 90 degrees to the fibers and delivers a stretch and separation between individual fibers. Deep friction is believed to create movement and induce reactive hyperemia,[27] stretch cross-bridges between fibers, and stretch adhesions within the structure. Fibrous scarring in tendons and their interfaces may become more pliable with massage. Although one researcher has suggested that deep friction application may result in some analgesia to the area,[28] there is little evidence to support this claim. Although many theoretical effects of massage have been put forth, there is little high-quality research evidence to support these claims. A Cochrane review evaluating the effect of deep transverse friction massage for treating tendinitis found no evidence of benefit with its use and concluded that there was the need for a high-quality randomized study using validated outcome measures to further evaluate its usefulness.[29]

How Massage Works

Massage may have immediate benefits to a patient, but it usually takes regular treatments to bring about significant improvements in a particular condition. Initially, massage may provide relief from pain, reduce tension, and help to sedate the nervous system. When pain has subsided, attempts to correct the underlying cause of the problem may begin. Healing is stimulated by both mechanical and reflex actions.

The mechanical effect of massage to the soft tissues may help to relieve tense muscles through relaxation, decreased pain, and increased mobility, and it may improve the circulation and lymphatic flow. The body, mind, and emotions all interact with the nervous system. It is possible that touching the skin sends messages, including reflex actions as the involuntary reaction of one part of the body to the stimulation of another part, to reduce the amount of stress hormones, which may lower blood pressure, slow breathing, improve digestion, and generate a sense of well-being. It is believed that massage also helps to release endorphins, the body's natural pain relievers, and to activate neuropeptides, the intercellular messages within the nervous system. These link the skin to the nervous system and the entire body, including the immune system. These actions as a result of massage are thought to have tremendous benefits.

Indications for Massage

Mechanical

If a dog has a chronic musculoskeletal problem, it may develop secondary problems, which lead to postural and gait adaptations. In time, these may also become primary problems in themselves as a result of changes in the soft tissues, which lead to unprotected joints, possible damage to the joints, and a painful response.

Surgical

If a dog has had surgery, massage may help to maintain mobility. If the dog must be confined for a period of rest or is on restricted exercise, it helps to maintain tone and muscle condition. Massage also helps psychologically by maintaining contact between the dog and the owner, and it may ease pain and discomfort. After resolution of the

surgical condition, massage is indicated to maintain flexibility of all joints and soft tissues, and to prevent further loss of function.

Disease

For dogs suffering a permanent low-grade condition, such as osteoarthritis, massage before exercise helps to encourage blood flow, delivery of nutrients, and maintenance of soft tissue extensibility. For those that suffer chronic imbalances, which have caused structural changes of the soft tissues, massage helps to relax and ease discomfort and may have a significant effect on tension and muscular effort.

Massage for the Competitive Dog

Proper massage applied to competitive dogs may help to establish or reestablish full tissue function. Massage may be employed during the training period for treatment and rehabilitation, and to enhance athletic performance. It may also help to prepare the dog for competition or exercise, and to reduce stress and aid relaxation. After hard exercise or competition, it aids recovery.

At an amateur level, training may sometimes be somewhat neglected, and massage can play a significant role in maintaining tissue pliability, in hopes of preventing injury. At the other end of the scale, top canine athletes are more likely to be overtrained, and massage can assist in recovery from fatigue. In this case, 2 hours should be allowed to elapse after competition before massage is started for maximum effects. Care must be exercised to not massage too deep and induce mechanical trauma, which may be detrimental to fatigued muscles.

Massage for Performance

To perform properly, the body must be able to adapt to the extra stresses placed on it. Requirements for excellence are power, agility, and coordination of the dog, and to attain this level of performance, the dog must cope with activity at the extremes of joint movement and muscle length. This places heavy demands on the strength and the endurance of the muscle, and the ability of the joints to move freely in all directions. The dog also needs freely mobile connective tissues. Inevitably, excess or repetitive stresses, or overwork, may increase the likelihood of other injuries, such as muscle strain. In all cases, this results in replacement of connective tissue with scar tissue, resulting in loss of power, flexibility, and movement in the tissues. Moreover, if the tissue is not rehabilitated before normal full activity is resumed, then it may never fully recover and return to normal limits. This in turn may overload the surrounding tissues, causing fatigue and hypertrophy of muscle groups.

Massage can be used to stretch and free this inelastic scar tissue and return tissue mobility to normal. It also helps to remodel and promote resorption of the scar and to reestablish full function of the tissues.

Fatigue

When a dog trains very hard, it may not fully recover between sessions, which may lead to general muscle soreness and increased likelihood of injury. Massage is used to decrease muscle tone and tension, thereby reducing muscular discomfort and promoting relaxation. There is also evidence to suggest that it helps recovery from fatigue, as compared with rest alone. It would therefore be very appropriate to use massage after competition, training, traveling, or any particularly stressful situation.

Muscle Soreness

Dogs may suffer soreness in their muscles as a result of extreme exertion and adverse environmental conditions. Endurance dogs galloping over uneven terrain in wet weather conditions or in extreme heat and humidity are examples of extreme conditions. This form of soreness normally begins after completion of the event, and sometimes not until the next day. Less fit animals may also suffer discomfort during such exercises, but as a result of metabolic insufficiency. Well-conditioned muscles generally do not suffer from this problem because they have a well-functioning capillary bed and adequate oxygenation of the blood supply. Obviously, a dog will be at a disadvantage if it has not had an opportunity to fully recover from the previous competitive efforts. An inadequate rest period may result in very severe discomfort caused by connective tissue inflammation, tenderness, and stiffness. Massage may delay the onset of soreness and reduce it.

Experience and some studies suggest that the ideal time to massage is 1 to 3 hours after exercise to enhance athletic performance and for the best therapeutic benefit. Also, for certain muscles, massage may enhance tissue lengthening more than stretching alone.

There are also contraindications to massage (Box 27-2).

Planning a Massage

When planning a massage for a competitive animal, the stage of training, the fitness of the animal, and the purpose of the massage must be considered. The various purposes of massage include relaxation, toning, precompetition warm up, intercompetition treatment, aiding muscle soreness 1 to 3 hours after competition, improving healing, relief of acute or chronic injury and fatigue, muscle soreness, and preventing adhesion formation. A thorough patient evaluation is necessary to assess areas that might benefit the most from proper massage techniques.

Patient Evaluation

During the initial observation period, care should be taken to ensure that the dog is standing on a flat, even surface in an area with adequate lighting.

Box 27-2 Contraindications to Massage

Do not massage if the dog is suffering from the following conditions:

- Shock (lowers blood pressure)
- Fever
- Acute inflammation (do not address area directly, but it would be acceptable to work elsewhere on the body)
- Skin problems (ringworm)
- Infectious diseases
- Acute stages of viral disease (herpes)



Figure 27-3 Caudal view of patient.

Caudal View

The patient is evaluated from a caudal view while standing. From this angle, the general posture is evaluated, looking for any skeletal abnormalities (e.g., scoliosis), areas of atrophy or hypertrophy, natural foot placement, weight-bearing tendencies of the limbs, head carriage, and the general appearance of the hindquarters and tail. The patient in Figure 27-3 has arthritis. Note that the dog has a clamped tail and decreased weight bearing on the left hindlimb, with the limb held in slight flexion.

Cranial View

From the cranial view it is important to note the symmetry of the head, eyes, ears, and paws, head carriage, forelimb weight-bearing tendencies, and the natural position of the forelimbs (e.g., neutral, rotation, adduction, abduction). Note in Figure 27-4 the deviation of the head to the left with slight rotation to the left and external rotation of the forelimbs. Also, the right paw is more splayed than the left.



Figure 27-4 Cranial view of patient.



Figure 27-5 Lateral view of patient.

Lateral View

It is important to observe both sides of the patient. Important points to note are the general posture, appearance of the muscles, head carriage, tail position, and limb joint angles. In the dog in Figure 27-5, note the roached back and clamped tail. Also note that the limb angles cannot be truly assessed because the dog is not standing squarely and symmetrically.

Gait Assessment

Walk: Caudal View

At this stage it is important to assess any hindlimb lameness. The ability of the dog to walk in a straight line should be assessed, along with the fluency of hindlimb movement



Figure 27-6 Gait assessment at a walk—caudal view.



Figure 27-7 Gait assessment at a walk—cranial view.

(i.e., does the dog move forward in a straight line, or does it circumduct, abduct, adduct, and so forth). It is important that the dog handler moves in a straight line and is educated in handling of dogs for evaluation purposes. Observations should be made of the lumbosacral rhythm, or the rise and fall of the tuber coxae and tuber sacrale, and the presence of any protective muscle splinting that may be occurring. In the dog in Figure 27-6, the tail is held low, the hindlimb toe-off phase initiates in an adducted position, the dog appears to move toward the left, and the dog appears to be dragging the left hind toe.

Walk: Cranial View

The cranial view should evaluate forelimb lameness and fluency of movement along with natural head carriage and foot placement. The nose, head, and neck deviate to the left, and foot placement appears close in the forelimbs in the dog depicted in Figure 27-7.

Trot: Caudal View

Similar to assessing walking from a caudal position, it is also important to note any hindlimb lameness, symmetry, fluency of movement, protective splinting of muscle groups (e.g., hamstrings or gluteals), tail carriage, and limb carriage and placement at a trot (Figure 27-8).

Trot: Cranial View

As with observation at a walk, observe the dog for any lameness, head and neck carriage, forelimb carriage and foot placement, and symmetry and fluency of movement. In Figure 27-9, note the low head carriage and the adducted position of the right forelimb.



Figure 27-8 Gait assessment at a trot—caudal view.

Side View

Both sides of the animal should be assessed. From this angle it is important to note the equality of the hindlimb and forelimb stride lengths, the ability of all limbs to retract and protract, head and tail carriage, protective muscle splinting, and general muscle interaction. In Figure 27-10, note the clamped appearance of the tail and possible guarding of the epaxial muscles.



Figure 27-9 Gait assessment at a trot—cranial view.



Figure 27-10 Gait assessment—side view.

Turning

It is important to evaluate the dog while turning in small circles in both directions, noting whether the bend follows through from nose to tail, whether the limbs cross over, and whether any areas look restricted. It is also important to recognize whether the dog actually understands what is being asked of it. In Figure 27-11 note the reluctance of the dog to perform the task: the dog appears to be braced from neck to tail, and the dog is also not crossing his legs.

Palpation

The dog's skeletal structure and muscle tone should be assessed after evaluation of the dog's gait. This is performed by palpating along the dog, from the wings of the atlas, along the spine and limbs, to the tail (Figure 27-12). Areas of irritability, spasm, atrophy, hypertrophy, and asymmetry are noted, along with any skeletal abnormalities.



Figure 27-11 Turning short in small circles.



Figure 27-12 Palpation of the dog.



Figure 27-13 Palpation of dorsal spinous processes.

By palpating on either side of the dorsal spinous processes with the thumbs, sensitive areas may be assessed (Figure 27-13). The surrounding areas are also palpated for any evidence of any other sensitive areas in the region, such as the withers and triceps region (Figure 27-14). Limb musculature is also assessed independently (e.g., shoulder



Figure 27-14 Palpation of the scapular and triceps region.



Figure 27-15 Palpation of the tuber sacrale and tuber coxae.

girdle and pelvic girdle musculature), noting whether any tone or spasm are present. Bony landmarks are palpated bilaterally, noting the symmetry of position and surrounding musculature, for example, the tuber sacrale and tuber coxae (Figure 27-15).

Passive Movements of the Cervical Spine

Flexion

While standing above the dog, place one hand over the cervical spine to enable assessment of the muscles and joints as they move through a ROM, then place the other hand over the dog's nose and guide the head down toward the forelimbs. Note the quality and range of movement and end feel, as well as the feel of the muscles and joints under the palpating hand. Although it is difficult to appreciate in the photograph, the dog in Figure 27-16 has restriction in flexion.

Extension

Support the dog's head under its chin with one hand and palpate the cervical spine with the other. Using the



Figure 27-16 Flexion of the cervical spine.



Figure 27-17 Extension of the cervical spine.

supporting hand, guide the head dorsally into extension. Note the quality and range of movement, the end feel, and the characteristics of the muscles and joints under the palpating hand. The dog in Figure 27-17 appears to resist this maneuver.

Side Flexion

While placing one hand over one side of the dog's nose, use the other hand to stabilize the dog while palpating the cervical spine. First guide the dog's head to one direction, and then after changing positions of the hands, guide the head in the other direction. Note the range and quality of movement, end feel, and so forth for both sides. In Figure 27-18, left-side flexion appears restricted, and increased tone is palpated by the evaluator in the cervical musculature.



Figure 27-18 Side flexion of the cervical spine.



Figure 27-19 Flexion of a forelimb.

Forelimb

Flexion

While ensuring that the dog is standing supported with the head pointing forward, flex the carpus and elbow. Note the range of movement, quality of movement, end feel, and scapular elevation. The dog in Figure 27-19 has good flexion in these joints.

Protraction

After evaluating flexion, continue to guide the limb forward into full protraction. Again, note the range and quality of movement and the end feel. Any areas of increased muscle tone should be identified. Moving the limb across the midline into adduction tests the latissimus dorsi muscle. In this dog, protraction of the limb appears to be adequate (Figure 27-20).



Figure 27-20 Protraction of a forelimb.



Figure 27-21 Retraction of a forelimb.

Retraction

From the protracted position, guide the limb back into retraction, ensuring that the dog's neck is not tilted from midline. The range, quality, and end feel of movement are noted. The dog in Figure 27-21 appears to have restricted movement, possibly because of increased tone in the brachiocephalicus muscle, and the dog does not appear to be comfortable.

Cervical Spine Side Flexion with Forelimb Retraction

Support the dog between your legs and with one hand, flex the neck to the side. Hold this position, and use the other hand to gently retract the opposite forelimb to assess the range of movement (Figure 27-22).



Figure 27-22 Side flexion of the cervical spine with retraction of the opposite forelimb.



Figure 27-23 Adduction of a forelimb.

Adduction

From the neutral position, guide the limb across the chest toward the opposite leg. Note the range, quality, and end feel of the movement (Figure 27-23).

Hindlimb

Flexion

With the dog supported, flex the hindlimb, obtaining full flexion of the hock and stifle. Assess the range and quality of movement, end feel, and the muscles. The dog in Figure 27-24 appears to have decreased limb flexion. Also note the dog's stance in the other limbs and overall level of comfort.

Protraction

With one hand palpating the hindquarters and caudal thigh muscles, allow the other hand to guide the limb forward into protraction. Assess the range and quality of movement, end feel, and muscles. The dog in Figure 27-25 appears to have full protraction, but may also have compensatory flexion of the lumbosacral area.



Figure 27-24 Flexion of a hindlimb.



Figure 27-25 Protraction of a hindlimb.

Retraction

With the dog in a standing position, use one hand to support the limb and guide it into retraction. Use the other hand to palpate the quadriceps and caudal thigh muscles. Again, note the range and quality of movement, end feel, and any fasciculation of the muscle. The dog in Figure 27-26 has very restricted retraction.

Note: All of the passive limb movements described previously can also be performed with the dog in lateral recumbency.

Tail

Tail movements may also be assessed, noting range of movement, quality of movement, and end feel. Elevation, depression, side flexion, and rotation movement of the tail can all be assessed (Figure 27-27).

Practical Application of Massage

Body Position and Comfort of the Clinician

Performing a massage can be quite tiring initially because you are concentrating on a new task and your body is



Figure 27-26 Retraction of a hindlimb.



Figure 27-27 Assessment of tail motion.

learning new movements. If the clinician is not careful, this may lead to tension and fatigue. To prevent this from occurring, try to work in a systematic way. Also, be careful to maintain proper body posture and relax while learning the moves to help minimize these risks. Using your larger muscles such as the shoulder muscles instead of your hands helps to minimize fatigue. It is important that the dog feel confident in you, too. Dogs appear to be very good at picking up tension and any lack of confidence.

Maintaining good posture means greater comfort, and the clinician will not fatigue as quickly. As a result, the massage will be more enjoyable for the clinician, resulting in greater relaxation for both parties. Altogether, the massage will be more effective, and a better result will be achieved.

Identification of Massage Areas and Application of Massage

When massaging, you may work on the entire dog, or selected areas. If the dog has a diagnosed condition, local massage may be appropriate on a regular basis. General massage helps with treatment of compensatory conditions and biomechanical changes.

Massage may be performed to isolated areas or to the entire limb and body. It may be used in conjunction with other therapies and veterinary treatments. It is also useful to be applied by the owner and may be successfully taught to the owner by the therapist.

Massage Techniques

Massage has many benefits for dogs, and there are many techniques that may be employed. Massage involves the use of a series of movements using the hands, which have an effect on the tissues. It is important to comfortably position the dog in lateral recumbency. It is also important to position yourself comfortably. The person performing the massage should be beside the dog on the floor, or place the dog on a raised surface, which may prevent you from stooping over or compromising your posture. It is best to start at one end, such as the head, and work along the dog in a sequenced manner (e.g., head, neck, shoulders, forelimbs, back, hindquarters, and tail). The following are techniques that may be used at any stage and over any muscle group.

Stroking

Stroking is a good technique to start the treatment (Box 27-3). When the dog is positioned, run your hand over the dog from neck to tail and down the limbs with medium pressure. This technique aids relaxation of the dog and gives further opportunity to assess the tissues while the dog is not bearing weight. Note the muscle tone and any swelling, masses, or temperature differences between body areas (Figure 27-28).

Effleurage

Effleurage is often performed to begin a massage, and is useful for relaxation, and to decrease swelling (Box 27-4). Beginning at a distal area, such as the paws, the hands move proximally, using medium pressure. This helps to move any toxins in the body toward the lymph nodes and aids drainage (Figure 27-29).

Petrissage

Petrissage is a type of massage with applied pressures which are deep and compress the underlying tissues. Kneading, wringing, skin rolling and pick-up-and-squeeze are petrissage movements.

Compressions

When performing effleurage, an increase in muscle tone in certain muscles may be identified. The triceps and deltoideus muscles are commonly affected. Compressions to the affected muscles may be easily administered and are often

Box 27-3 Stroking

Stroking is often used initially in a massage and on most areas. Some consider this a form of effleurage.

Uses

Relax the dog.
Introduce touch.
Sedate (slow stroking) the dog.
Stimulate (brisk stroking) the dog.
Decrease muscle tone (slow).

When to Use

At the beginning or end of massage
When only limited time is available, on a daily basis
To calm and relax in a stressful situation
Any time

Application

Stroking is usually applied from the proximal limb toward the distal limb.
Begin at the top of the area to be worked.
Place the entire hand in contact with the skin.
Maintain a gentle but firm pressure.



Figure 27-28 Stroking.

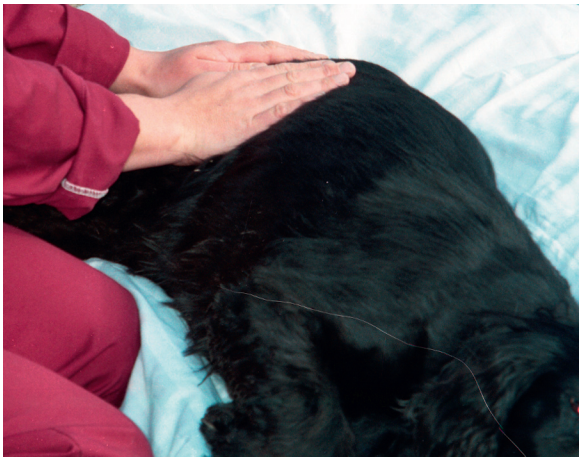


Figure 27-29 Effleurage.

Box 27-4 Effleurage

Effleurage is often performed initially in a massage on most areas of the body, particularly on the limbs and when swelling is present. It is the most common type of massage used, and often it is used in an evaluative manner to assess for tissue tightness, presence of muscle spasm, and so forth.

Uses

Increase venous and lymphatic return.
Aid removal of chemical irritants.
Improve mobility between tissues.
Stretch muscle fibers.
Decrease muscle tone.
Note: If used deeply, it may be used to increase muscle tone.

When to Use

At the start and end of a session, and often in between various techniques
Should be performed toward the lymph nodes
May be performed using one or both hands, fingers or thumbs

Application

Begin at the distal part to be effleuraged.
Make contact with the skin and apply even pressure to sink into the superficial tissues.
Make a sweeping movement to the top of the area, molding to the contours and maintaining the same depth of pressure throughout the stroke.
Finish the stroke over the lymph node, then remove the hands and reposition at the start of the next stroke. If the stroke is not to the lymph nodes, continue to the nearest body part.
Bring the hands toward you as you work, using the heel of your hand.
Watch that the fingers and palm of the hand do not lose contact with the patient.
Maintain an even depth of pressure while the hands mold to the body.
Overlapping strokes are used, continuing until the entire body region is covered.
Note: In a small area, use only one hand, finger or thumb.

very effective (Box 27-5). The palm or heel of the hand is placed over the muscle and pressure is applied. This pressure is maintained for 15 seconds before moving on to another area. This maneuver should be repeated several times. By restricting blood flow and then releasing pressure, the circulation increases, and this is thought to help to decrease tone (Figure 27-30).

Holding and Placing

An area of increased tone or spasm may be relaxed by placing a hand over the area. This technique traps heat in the area, increasing the local circulation and promoting local relaxation (Figure 27-31).

Percussion (Clapping, Hacking, Pounding)

If there is a general area of increased tone, such as in the caudal thigh or gluteal muscles, clapping or hacking

Box 27-5 Compression and Wringing

Compressions can be used all over and are very effective on large muscles. Wringing is often very relaxing and has a great sedative effect.

Uses

Aid venous and lymphatic return.
Help with removal of chemical irritants.
Increase the mobility and length of fibrous tissue.
Restore mobility between tissue surfaces.
Aid tissue fluid mobility.
Increase extensibility and strength of connective tissue.
May also trigger skin reflexes and others linked to it.

Features

These techniques compress the soft tissues, then they squeeze or roll them (wringing), working to the tissue end.

When to Use

Use on superficial tissue, ligaments, or muscles to soften up large areas.

Application of Compressions

Use one hand or both, or pads, fingers or thumbs.
Begin at the proximal end of the tissue and move distally.
Contact the skin and compress the tissue.
Skin is moved on the underlying tissue; there is no glide.
The hands or digits or back of fist are used in a circular motion; this will initiate a slight stretch behind, and skin wrinkling ahead.
If using right and left hand, the right moves clockwise and the left counterclockwise.
Pressure is applied to deeper tissues when tissues are compressed. It is a similar movement to that used to open a childproof medicine bottle: press down initially, twisting as you do so, then release.
To go from one completed compression to the next, slide the hands across.

Application of Wringing

Wringing is applied using two hands.

- One brings the tissue toward you, the other moves it away. Hold for 2-5 seconds, then slowly release.
- The hand then moves to take up position on the adjacent tissue.

techniques may be employed (Box 27-6). This technique may also be used in areas of muscle weakness.

Clapping is performed by using a cupped hand. Gently but firmly, a clap is applied to the area (Figure 27-32). As the hands fall onto the muscle mass, a hollow sound should be heard. Hacking is performed by alternately using open hands, with the ulnar border of the hands falling vertically on the muscle mass (Figure 27-33). In both cases the techniques are performed with light pressure initially, progressing to medium pressure. Both clapping and hacking improve general circulation in the areas treated, promoting relaxation.



Figure 27-30 Compressions applied to tissues.



Figure 27-31 Placing technique over tissue.



Figure 27-32 Clapping of tissue.

Trigger Point Therapy

Often small areas of spasm may be felt within a muscle belly. Such nodules may be treated using trigger point therapy. The nodules are located and ischemic compression is applied using one or two fingers. The compression is held for approximately 20 seconds and released for 10

Box 27-6 Percussion

Percussion consists of several techniques, the three most important being hacking, pounding, and clapping.

Uses

Stimulate local circulation.
Provoke muscle and tendon reflexes.
Provoke a general stimulatory effect.
Stimulate muscle tone.
Assistance in clearing airways (coupage).

Features

Light percussion has an effect on the superficial tissues; heavier application affects deeper layers and must not be used over organs.

Application of Clapping (see Figure 27-32)

Hold the fingers and thumbs closed together to form a relaxed, cup shape. The elbows should be flexed and the arms abducted. The arms should be alternately flexed and extended so that the borders of the hands and fingers stroke the skin. The strokes are rapid, light, and brisk. Air is trapped between the hands and the skin, producing a hollow sound as contact is made. The entire treatment area should be covered.

Application of Hacking (see Figure 27-33)

Hold the arms away from your sides, and flex the elbows approximately 90 degrees, with the wrists fully stretched backward (extended) and fingers relaxed. The shoulders, where possible, should be over the area.

The border of the little finger and hand strike the skin alternately, lightly and rapidly. The movement comes from the wrist, and it involves a full side-to-side movement of the wrist in this position.

When doing very light hacking, only the fingers need strike the skin.

Application of Pounding

The arms should be held out, with elbows flexed to near 90 degrees. The wrists are extended with fingers flexed loosely into fists. The hips should be flexed to enable the shoulders to be over the area being treated. The medial borders of the fifth fingers strike the skin alternately and rapidly. The movement is at the radioulnar joint, which pronates and supinates.

seconds before compression is reapplied. Generally three or four repetitions may be required (Figure 27-34).

Deep Transverse Friction

Scar tissue forms within muscles as a result of injury. Deep transverse frictions may be applied to help reduce the scarring (Box 27-7). These techniques were developed by Cyriax.[26] Using the index and middle fingers at a 90-degree angle to the muscle fibers, pressure is applied perpendicular to the direction of the fibers. This is repeated 10 times and performed in sets of 3 to 10. It is a good practice to complete this technique with some gentle massage and stretching to relax the dog, as deep transverse friction may be uncomfortable (Figure 27-35).



Figure 27-33 Hacking of tissue.



Figure 27-34 Compression of trigger point.

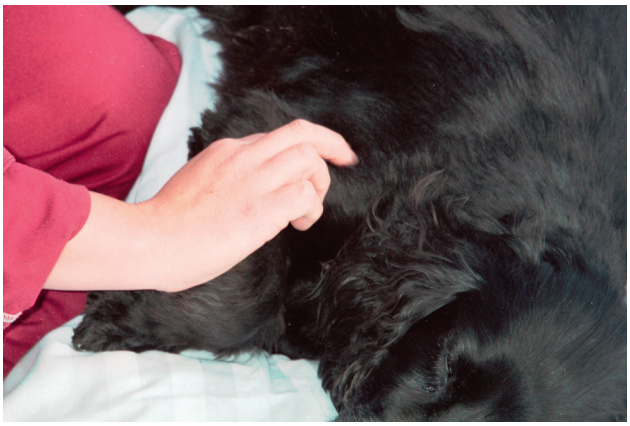


Figure 27-35 Deep transverse friction massage.

Passive Movements

To conclude the treatment session, when all the muscles are warm and relaxed, passive movements similar to those done in the weight-bearing position to evaluate the patient

Box 27-7 Friction Massage

Traditional Friction Massage

Uses

Stimulate local circulation.
Aid in removal of chemical irritants.
Restore mobility between tissue interfaces.
Restore mobility to specific anatomic structures.

Features

Performed with the tip of the thumb or fingers making small rotary movements, the pressure being appropriate to the desired tissue interface. Lubricants are not used as there is no glide on the skin.

Application of the Friction

The tip of the thumb or fingers should be placed on the skin, over the structure to be treated. The tissues are compressed to the depth of the structures being treated. Small rotary movements are performed on the structures while maintaining constant pressure, but there is no glide on the skin. The superficial tissues are moved on the underlying structures.

Deep Transverse Friction

Uses

Stimulate local circulation.
Promote reactive hyperemia.
Restore mobility between tissue interfaces.
Restore mobility to specific anatomic structures.

Features

Performed with the tip of the thumb or fingers making small rotary movements, the pressure being appropriate to the desired tissue interface. Lubricants are not used as there is no glide on the skin.

Application of Friction (see Figure 27-35)

The tip of the thumb or fingers should be placed on the skin, over the structure to be treated. The tissues are compressed to the depth of the structures being treated. Small rotary movements are performed on the structures while maintaining constant pressure, but there is no glide on the skin. The superficial tissues are moved on the underlying structures.

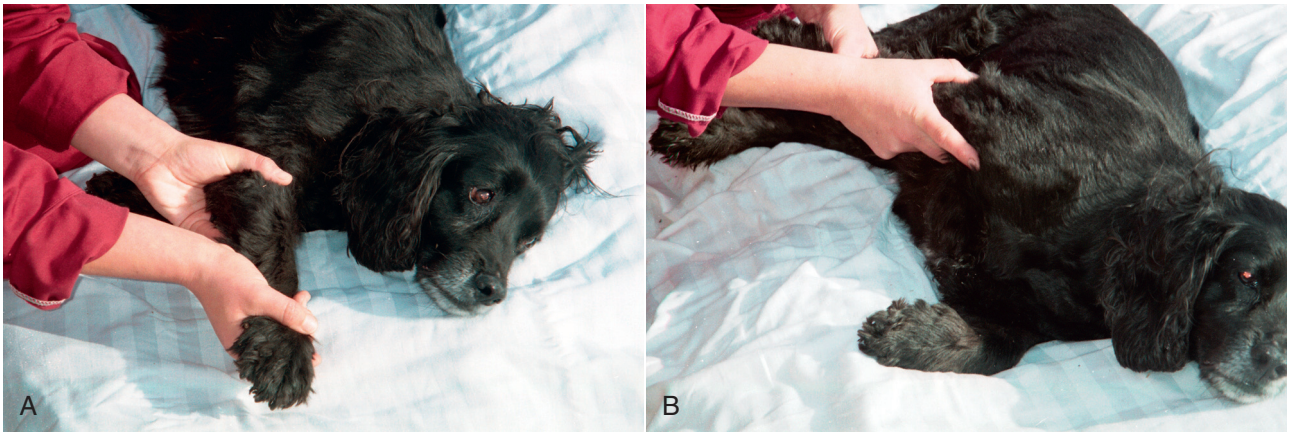


Figure 27-36 **A,** Forelimb mobilization with limb fully protracted. **B,** Forelimb mobilization with limb fully retracted.

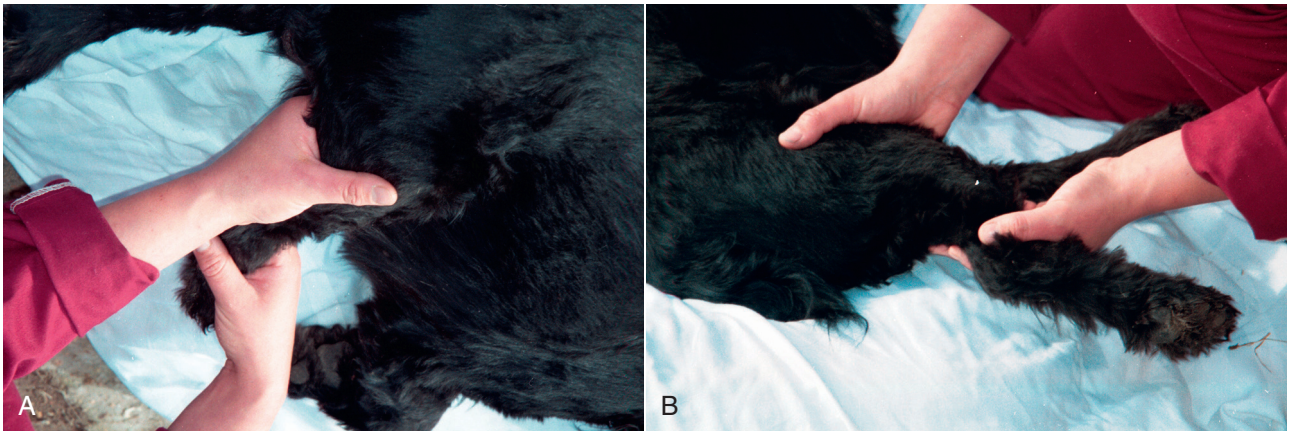


Figure 27-37 **A,** Hindlimb mobilization with limb fully protracted. **B,** Hindlimb mobilization with limb fully retracted.

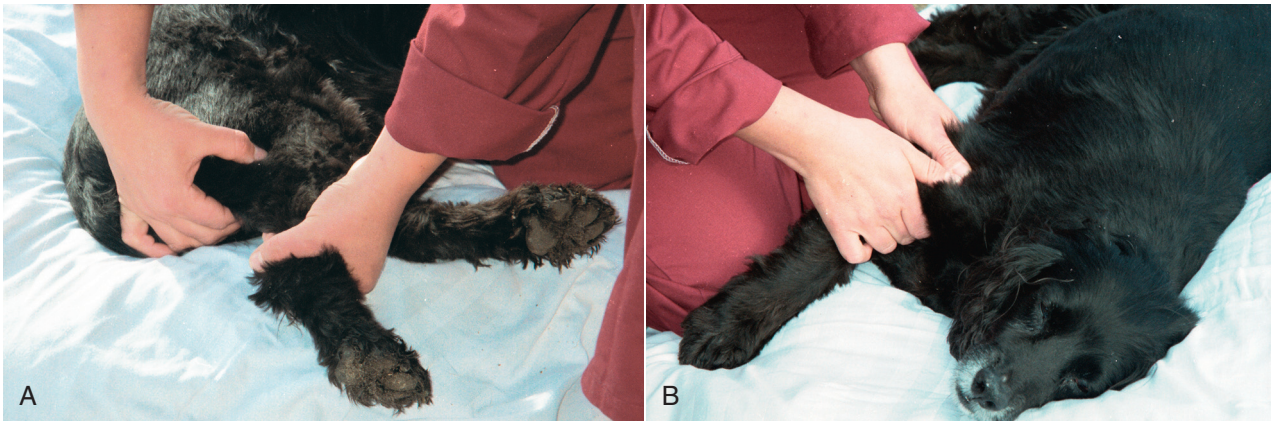


Figure 27-38 **A,** Isolating muscle spasm while placing rear limb through a range of motion. **B,** Isolating muscle spasm while placing forelimb through a range of motion.

are performed. Again it is best to work in a sequence, such as from forelimbs to hindlimbs.

Forelimb and Scapular Mobilizations

Place one hand over the scapula and support the forelimb with the other. The scapula is mobilized from elevation to protraction, depression, and retraction. The limb is fully protracted and retracted (Figure 27-36).

Hindlimb

Similar movements are performed on the hindlimb. With one hand supporting the hindlimb and the other resting over the hip, the leg is elevated and depressed before it is protracted and retracted (Figure 27-37).

In some cases when passive movements are performed, areas of spasm may remain within the muscles, restricting full available range of movement. In these situations it is often useful to combine passive movements with trigger point therapy. The isolated muscle spasm is held by the therapist's finger and thumb while the other hand guides the limb through a ROM (Figure 27-38). If restriction occurs again, the position should be held for a minimum of 15 seconds, then released before repeating. Following treatment, the dog should be very relaxed. The dog should be turned over and the massage should be repeated on the other side.

Alternative Methods

Several methods may be used as an alternative or adjunct to massage, including mechanical massagers, grooming the dog with a massager, and using commercially available massage lubricant.

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28 Exercises for Proprioception and Balance

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Many conditions affect proprioception of small animals. The most obvious of these are neurologic conditions. However, recent evidence suggests that musculoskeletal conditions and injuries also affect proprioception, especially of joints.

Exercise enhances balance and proprioception,[1] and a well-designed balance training program can improve balance in normal individuals.[2]

Balance is the ability to adjust equilibrium at a stance, or during locomotion to adjust to a change in direction or ground surfaces, whereas proprioception is the perception of stimuli produced by the body in relation to movement and changes in body position. Proprioceptive training includes activities that may be performed at low or high speed and require an awareness of limb position in space. Example of activities include walking in circles or a figure eight and walking across obstacles of various shape, height, and spacing. Balance exercises are exercises requiring rapid responses to changes in a surface, such as walking on a trampoline, or standing on a balance or wobble board. Other balance and agility exercises include cavaletti rails, exercise balls, rapid changes of direction while trotting and galloping, ball playing, tug of war, dancing, and wheelbarrowing. This chapter discusses current knowledge regarding proprioception and balance and exercises to enhance patient function.

The Science of Proprioception

The proprioceptive system is responsible for detecting changes in the position of the trunk, limbs, and head. The system is distributed widely throughout all of the spinal nerves, and general proprioception receptors are located in the muscles, tendons, and joints deep to the surface of the body. The receptors respond to changes in the length and position of the structures where they are located, giving the normal individual a precise feeling of where their body parts are located. The postural control system consists of three functional components, including biomechanical, motor coordination, and sensory organization components.

Joint Proprioception

Ruffini and Pacinian receptors have been identified in canine cruciate ligaments and are innervated by axons penetrating from the peripheral synovium surrounding the ligament.[3] The proximal third of the ligament contains a greater number of receptors.[4] The cranial cruciate ligament is capable of sending afferent input to the central nervous system, and it is important to the biomechanical and proprioceptive functions of the stifle joint, including characteristics of movements and position-related stretches of these ligaments.[5] The effects of the gamma-muscle-spindle system in the muscles around the knee are so potent that stretching of the cruciate ligaments at relatively moderate loads may induce major changes in responses of the muscle spindle afferents.[5] These changes may result in changes in the muscular stiffness around the knee joint, helping to affect functional stability of the knee joint.

Mechanoreceptors have been studied in torn and reconstructed anterior cruciate ligaments (ACL) in animals and humans.[6] In humans with untreated ACL lesions, normal mechanoreceptors remain in the ligament 3 months after injury. The number then gradually decreases, and by the ninth month, few free nerve endings are present.

The correlation between the number of mechanoreceptors in ACL remnants and joint position sense just before an ACL reconstruction in human patients has been studied.[7] A positive correlation between the number of mechanoreceptors and accuracy of the joint position sense was found, suggesting that proprioceptive function of the ACL is related to the number of mechanoreceptors. Therefore consideration should be given to preserving cruciate ligament remnants during stabilization surgery if the ligament is relatively normal. However, a study in cats that evaluated joint capsule afferent neuron stimulation before and after transection of the cranial cruciate ligament suggested that capsule afferents continue to behave normally after ligament transection, despite the increased joint laxity.[8]

Proprioceptive ability declines with aging in humans.[9] A randomized clinical trial evaluated the effects of an 8-week balance program on proprioception compared with only a fall-prevention program in healthy older adults (mean age = 73.9).[10] Each group was assessed before and after the study, and a control group also underwent follow-up testing 8 weeks after intervention. Additionally, this study compared the intervention group to a younger, healthy population who were only assessed once for comparison. Interventions included typical balance and proprioceptive exercises such as activities on foam, foam balance beams, wobble board activities, and others. Subjects underwent 1-hour sessions three times per week, for

a total of 8 weeks. The balance group showed significant improvement within the group in velocity discrimination from before and after intervention. In addition, there were significant differences after intervention between both treatment groups. In fact, the balance group showed no significant differences in velocity values after training when compared with younger adults. However, no other differences in joint position sense or threshold to perception of movement were encountered. Because velocity, regulated mainly by muscle spindles, is considered vital to postural stability, this study suggests that proprioceptive training could decrease the risk for falls in older adult humans. However, these results were not maintained long term, suggesting that continued training is necessary.[10]

Proprioception is further impaired in older adult patients with knee osteoarthritis (OA), which may contribute to additional functional impairment.[1,9,11-13] On the other hand, the pain of osteoarthritis does not seem to have good association with disability and joint proprioception.[14] Although patients with arthritis have decreased joint position awareness, physical therapy may improve joint position sense and proprioception through participation in a carefully constructed sensorimotor rehabilitation program in which pain, effusion, and fatigue are minimized.

In healthy individuals, muscle receptors are the primary determinants of joint position sense, and capsular receptors have a secondary role. Studies have been performed in humans to test position sense of the knee joint before and after fatigue.[15,16] With fatigue, there appears to be a change in mechanism of appreciation of joint position, possibly because of an increased role of capsular receptors regarding joint position sensitivity with muscle fatigue–induced laxity.

Appreciation of joint position was also found to be significantly more sensitive after warming up by jogging and stretching for 4 minutes, demonstrating adequate warm-up prior to activity may be beneficial.[17] Conversely, to test whether cryotherapy affects position sense of the knee, a cooling pad was applied to knees of humans.[18] Cooling for 15 minutes resulted in increased knee joint stiffness and decreased sensitivity of joint position sense. However, another study indicated that cryotherapy of the shoulder does not affect joint position awareness.[19] It may be possible that differences exist in different joints, with more superficially located joints affected more by cryotherapy. These findings should be considered in therapeutic programs that involve exercise immediately after applying cryotherapy.

Long-term effusions and the nature of the inflammatory fluid may be responsible for the seeming loss of proprioception observed in some clinical conditions with chronic joint effusion. However, infusion of saline to simulate acute joint effusion apparently does not change proprioceptive ability.[20] An elastic bandage placed on the knee of humans had a positive effect on joint proprioception, and may be recommended in those patients with joint proprioception problems.[21,22] Patella taping techniques improved joint position sense in patients with patellofemoral pain syndrome that had poor proprioception to begin with, but did not help those with good proprioception.[23]

Neurologic Conditions

Proprioception is commonly affected by neurologic conditions in animals and people. Proprioception and rehabilitation have been studied in people following a stroke. There are many approaches to physical therapy following stroke injury in people. One method of treatment, known as supported treadmill training, appears to have beneficial effects on proprioceptive function in people after a stroke.[24,25] A 4-week period of body weight–supported treadmill training with up to 40% of the patient's body weight supported resulted in significantly improved clinical outcomes, including functional balance, motor recovery, overground walking speed, and overground walking endurance.[26] The effects lasted for at least 3 months after training. The subjects with greater gait impairments benefited the most from training with body-weight support.

Another study evaluated the delayed effects of a balance training program compared with no training on patients following a hemiplegic stroke.[27] Dynamic balance function of patients in the visual feedback training group had significant improvements when compared with the control group. Activities of daily living also had significant improvements at 6 months of follow up in the trained group. A study of older humans indicated that a short individualized exercise program improved functional balance. This improvement was maintained for at least 1 month, but there was no effect 1 year after training ceased, suggesting that continued training may be necessary.

Strength training should also be considered as part of the rehabilitation program for patients. In one study, chronic human stroke patients underwent a progressive resistance strength training program to evaluate changes in muscle strength, gait, and balance.[28] Subjects participated in a 12-week resistance training program. Lower limb strength improved 68% on the affected side. Repeated chair stand time improved 21%, motor performance improved 9%, and static and dynamic balance improved 12%. Other studies have confirmed the benefits of strength training in human stroke patients.[29] These results suggest that strength training should be considered even in patients with chronic deficits.

Proprioception and Activity

In the past decade, research in humans has suggested that stretching prior to activity may reduce acute proprioception, which might lead to injury. A recent study showed significant differences in detecting knee movement after undergoing a hold-relax proprioceptive neuromuscular facilitation stretch routine on the hamstrings and quadriceps compared with a control group.[30] The treated group had a significantly slower reaction time from pretest to posttest, whereas the control group did not. It is important

to note that this study suggests stretching in general may promote decreased proprioception in certain joints immediately after stretching. This could hold implications for dogs in the late phases of rehabilitation, especially for sporting dogs, in which stretching prior to training could alter coordination and motor function, considering that proprioception is essential for skilled movements.

Proprioceptive training may help reduce injuries in athletes. In a recent randomized controlled trial of highly competitive basketball players, a treated group (n = 81), who received a proprioceptive training regimen along with normal workouts, was compared with athletes undergoing a normal training routine (n = 91).[31] Proprioceptive training was performed once a week and consisted of six stations, each performed twice each for 45 seconds, followed by 30 seconds of rest as they switched stations. All exercises were clearly illustrated and showed proper progression. Over the course of an entire season, 21 injuries occurred in the control group compared with 7 in the treated group. The treated group also showed a more stable single limb stance in the frontal plane and overall sway compared with the control group. Although this study was performed on humans, it suggests that proprioceptive training may prevent injuries, and this might also be applicable to animal rehabilitation.

Finally, in a recent review to help guide clinical decisions on prevention of ACL injury with the use of proprioceptive and balance training, the authors concluded that there is a lack of solid evidence to determine if this type of training has beneficial effects. However, this was mainly a result of poor experimental design of the studies (such as poor description of training regimens and high drop-out rates), which at least determined no detrimental effects of proprioceptive training.[32] Others have agreed, stating that many articles combine proprioceptive exercises with plyometrics, agility, and sport-specific exercises, making the specific effects of each individual treatment difficult to ascertain.[33]

Proprioceptive and Balance Training

When an animal is able to safely stand (independently or with assistance), activities to improve balance may begin. Balance may be assessed using a scoring system based on similar evaluation instruments in people (Table 28-1). Static balance is the animal's ability to maintain balance while standing, while dynamic balance is the ability to maintain balance while the body is moving. Static balance training is generally performed with the animal in a standing position, either independently or with support. The following exercises may be performed to challenge the animal's balance. These exercises should be conducted on a nonslip surface to reduce the risk of falling.

Body Slings

Patients with weakness, limited motor function or poor proprioception may benefit from placement in a body sling or harness to provide support for standing (Figure 28-1). An advantage of a sling is that the patient is able to maintain a position with the limbs placed under the body in a standing position, which may provide an opportunity for limb strengthening and early proprioceptive training. Frequent episodes of supported standing also help relieve pressure on bony prominences and may reduce the chances of decubital ulcer formation. Excessive congestion of the lungs that may result from spending long periods in lateral recumbency may be prevented with early implementation of standing in a sling. A body harness might be used for patients with tetraparesis, but must be adjusted properly so that respiration is not compromised and the limbs are not compressed in the openings of the harness. Proper padding is also important

Table 28-1 Balance Score for Small Animals

BALANCE ITEM SCORE (0-5)	
1. Sitting squarely and unsupported	_____
2. Change of position: sitting to standing	_____
3. Change of position: standing to sitting	_____
4. Position from lateral recumbency to sternal position	_____
5. Standing unsupported	_____
6. Standing with feet together	_____
7. Standing with one leg raised	_____
8. Turning 360 degrees	_____
9. Stair climbing	_____
10. Balancing while standing and turning head	_____
TOTAL (0-50):	_____

Score each item from 0 to 5,
0 – unable to perform the activity
1 – barely performs the activity with assistance
2 – performs activity to some extent with difficulty, with or without assistance
3 – performs activity reasonably well with mild difficulty and no assistance
4 – performs activity well with minimal difficulty and no assistance
5 – performs the activity normally

INTERPRETATION
0-15: Generally needs full support
16-40: Generally can walk with assistance
41-50: Generally independent activity

Based on the following in people:
Berg K, Wood-Dauphinee S, Williams JI, Maki, B: Measuring balance in the elderly: Validation of an instrument. *Can J Pub Health* July/August supplement 2:S7-S11, 1992.
Berg K, Wood-Dauphinee S, Williams JI, Gayton D: Measuring balance in the elderly: preliminary development of an instrument. *Physiother Can* 41:304-311, 1989.
Provided by the Internet Stroke Center: www.strokecenter.org



Figure 28-1 **A,** Patients with poor balance or proprioception may benefit from placement in a body sling or harness to provide support for standing. **B,** An elastic band may also be used for support and to provide a form of rhythmic stabilization while standing or walking.



Figure 28-2 Rubber bungee cords may be used to secure a harness to the supporting frame.



Figure 28-3 For weight shifting of the rear limbs, the therapist is positioned behind the patient, and the hands are placed on either side of the pelvis for support.

for comfort. The skin should be inspected after each session in the sling or harness to identify areas of potential skin irritation or breakdown. Rubber bungee cords may be used to secure a harness to the supporting frame (Figure 28-2). This may aid in the facilitation of weight bearing and movement during the sessions. The elasticity of the cord permits some gentle up-and-down movement that encourages the animal to safely bear partial weight on its limbs. It also may allow some attempts to move forward, backward, and side to side. A gentle bouncing motion may be manually introduced by the therapist to initiate rhythmic stabilization of muscle groups throughout the trunk and limbs.

Weight Shifting

The therapist may begin gentle weight-shifting activities by supporting the animal on either side, and then gently pushing on one side, then pushing back the other way in a slow, rhythmic fashion. For example, for weight shifting of the rear limbs, the therapist is positioned behind the patient, and the hands are placed on either side of the pelvis (Figure 28-3). The animal is slowly pushed to one side, while supporting the patient with the other hand. The animal is then slowly pushed back the other way. As balance improves, the speed of the unbalancing movements may increase. Additional challenges may be added



Figure 28-4 A supporting towel is moved back and forth to shift the dog's weight ("shoe buff" maneuver).



Figure 28-5 Perturbation exercises are performed to disturb the animal's balance by gently pushing the animal at the hips or shoulder without supporting the opposite side.

by slowly moving a supporting towel or sling back and forth to shift the dog's weight ("shoe buff" maneuver) (Figure 28-4).

As balance improves, the handler may perform *perturbation exercises* to disturb the animal's balance by gently pushing the animal at the hips or shoulder without supporting the opposite side (Figure 28-5). The goal is to disturb its balance just enough so the animal can recover, being careful not to push with a force that may cause the animal to fall. Generally, pushing the animal to the more affected side challenges the animal sufficiently to allow the activity to have the desired effect.



Figure 28-6 A loaded weight shift is performed by placing force over the dorsal aspect of the dog's body while shifting the weight to the affected side.

In situations of reduced weight bearing on one side, a *loaded weight shift* may be performed (Figure 28-6). Dogs recovering from joint surgery or other conditions that have reduced weight bearing may benefit from this exercise to encourage some weight bearing, early muscle strengthening, and joint position awareness. For this exercise the therapist's forearm that is on the side of the injury is placed across the animal's dorsal pelvis (hindlimbs) or shoulders (forelimbs) and gentle downward pressure is applied. With the other hand, the weight is gently shifted toward the affected side.

Some dogs become conditioned to having the therapist push on one side to shift the weight to the other side, and shift their weight back toward the therapist to prevent being pushed toward the affected side. In this case, a *rebound weight shift* may be effective. For this maneuver, the therapist gently pushes the animal toward the affected side as usual. When the animal shifts its weight to resist the movement, the therapist suddenly releases pressure, and simultaneously pushes gently toward the unaffected side (Figure 28-7). This results in a sudden unbalancing; the animal initially shifts its weight toward the unaffected side, but to keep from falling, it immediately shifts its weight back toward the affected side.

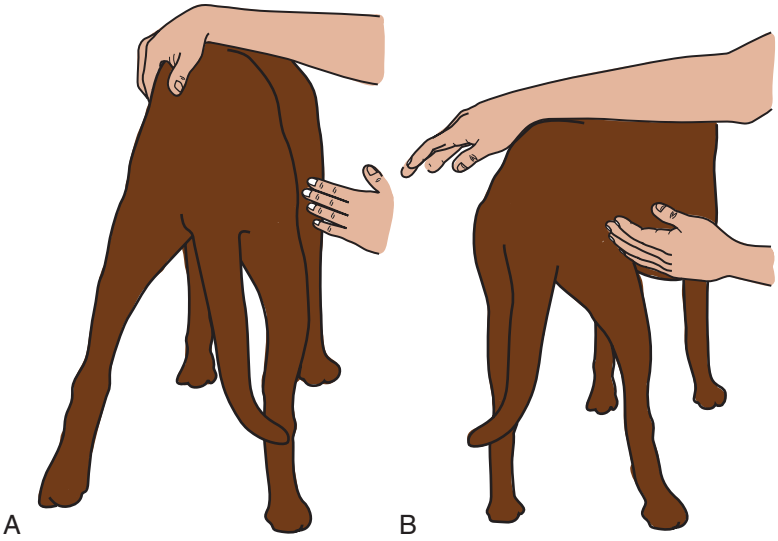


Figure 28-7 Rebound weight shift to encourage an animal to bear weight on the affected limb. The dog's weight is initially shifted toward the normal (*right*) limb. Pressure is suddenly released, resulting in a shift in weight toward the affected (*left*) limb to maintain balance.

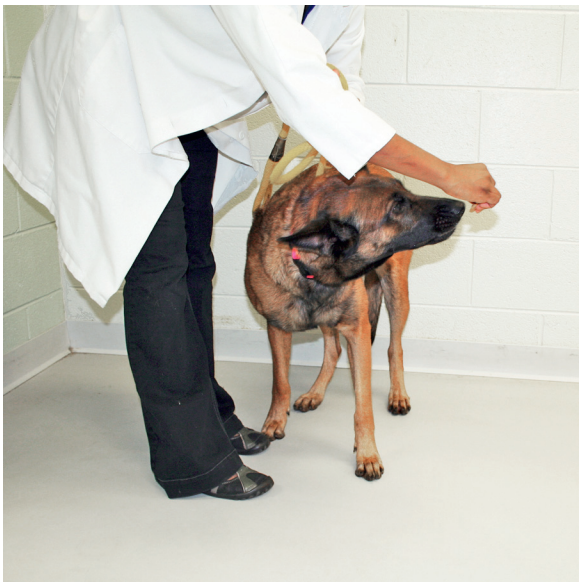


Figure 28-8 The therapist encourages weight shifting to help develop improved proprioception by moving a treat side to side.



Figure 28-9 The therapist is ready to provide support to a patient in the event of a loss of balance.

While the animal is standing, a treat or ball may be used to encourage weight shifting. The dog will follow the treat up and down and side to side (Figure 28-8). Start with small movements and progress to larger, more challenging movements. The movement of the head causes the dog's center of gravity (COG) to shift. As the COG shifts, the dog must shift its weight to maintain its balance. To maintain the unassisted standing position, the animal is required to use strength, coordination, and balance. If the dog is motivated by ball play, a more challenging form of this exercise is close-distance ball tossing from above and the sides. During early attempts at these exercises it may be necessary to have someone available for standby assistance as the dog may be overchallenged, lose its balance, and fall (Figure 28-9). As the patient improves, close-distance ball tossing may be performed with the patient on a trampoline or air mattress.

Manual unloading of a limb during stance is another challenge to balance. Lifting and holding a single limb off the ground while the dog is standing cause a shift in the animal's COG (Figure 28-10). The animal shifts its body weight and COG to maintain the standing position. During a session, the handler may lift each leg separately to see where the animal is the weakest and focus on that area in successive sessions. If the animal is unable or unwilling to perform this exercise, it will not shift its weight properly,



Figure 28-10 Shifting an animal's center of gravity by lifting a limb off the ground and forcing the patient to redistribute its weight.



Figure 28-11 Dynamic balance and proprioception may be challenged while gently shifting the dog's weight while walking. Caution should be used to avoid too much force so the patient does not fall.

but instead will bear the weight on the handler's hand or collapse to the ground. A technique to avoid bearing all of the weight on the handler is to slowly abduct the raised limb, which allows transfer of weight from the handler to the desired limb to avoid falling.

Weight shifts may also be performed during walking to provide challenges to dynamic stability. As the animal is walked in a straight line, the handler gently bumps or pushes the animal to one side to challenge the dog to maintain its balance (Figure 28-11). Caution should be used to tailor the force of the push to the animal's stage of recovery to avoid falls and injury. As the patient improves, dynamic weight shifting may be performed while the animal is walked in circles, figure eights, or while pole weaving (Figure 28-12).



Figure 28-12 Dynamic weight shifting may also be performed while the animal is walked in circles.

After the animal has reasonable balance and proprioception while standing and walking, more challenging balance and proprioception exercises may be initiated.

Balance Board

A board placed over a fulcrum, such as a length of pipe, may be used to rock the dog forward and backward or side to side (Figure 28-13). Movement of the surface the dog is standing on provides additional challenge to the patient. In most cases, one person helps to support the dog while standing on the board, while another person slowly rocks the board back and forth. Initially, the movements are slow and rhythmic. As the patient improves, the motions may be faster and less predictable to challenge the animal to react quicker.

Wobble Board or Quadruped Balance Platform

A wobble board or human biomechanical ankle platform system board may be used to help the animal practice proprioceptive training on just the forelimbs or the hindlimbs (Figure 28-14). If the goal is to challenge the animal using all four limbs, a balance board large enough to accommodate all four limbs may be used (Figure 28-15). It is important to provide support to allow the animal to shift its weight and exercise its proprioception and balance. Again, the initial movements are slow and rhythmic, but as the patient improves, the motions may be faster and less predictable to further challenge the animal.

Exercise Balls and Rolls

Therapeutic exercise balls and rolls designed for human use may be employed to improve balance, coordination, and strength. The forelimbs may be placed on the ball and the patient supported by the handler, requiring the dog to maintain static balance of the caudal trunk and rear limbs

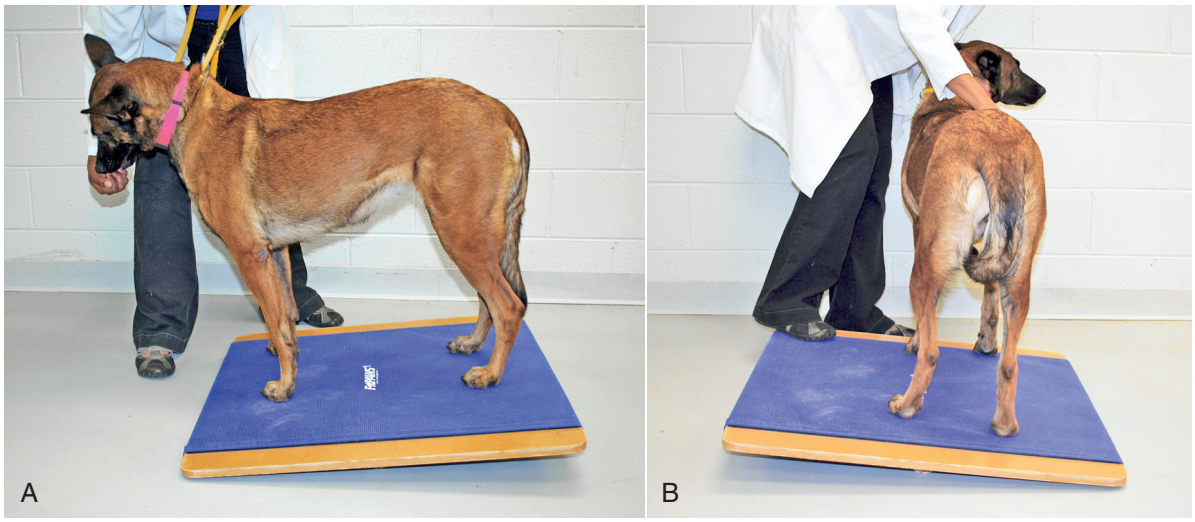


Figure 28-13 A balance board may be used to provide balance and proprioceptive training from the forelimbs to the hindlimbs (*A*) or side to side (*B*).



Figure 28-14 Use of a wobble board or human biomechanical ankle platform system board under a patient's hindlimbs to assist with balance and proprioceptive training. Similar training may be applied to just the forelimbs, or small dogs may stand with all four limbs on the board.



Figure 28-15 Using a balance platform to help quadruped animals develop improved balance and proprioception.

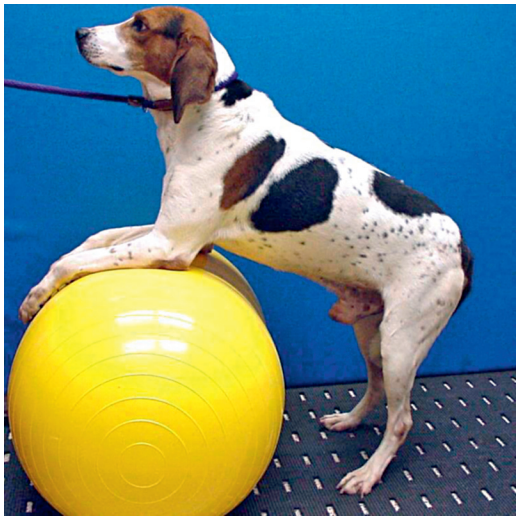


Figure 28-16 An exercise roll may be used to challenge balance and proprioceptive function. The forelimbs may be placed on the ball and the patient supported by the handler, requiring the dog to maintain static balance of the caudal trunk and rear limbs.

(Figure 28-16). Dynamic balance may also be challenged as the ball or roll is slowly moved forward, backward, and side to side, challenging the rear legs to maintain balance while movement occurs. To address the cranial trunk and forelimbs, the rear limbs are placed over the ball as the forelimbs are asked to balance the body weight during both stance and gentle movements (Figure 28-17).

Further challenges may be instituted by having the dog stand on a therapy ball or roll (Figure 28-18). This is extremely challenging and uses multiple core stabilizing muscles. Therefore the sessions should be relatively short to avoid fatigue and possible injury. One person stabilizes the ball, while one or two people, depending on the size of the dog, support the patient. Further challenges are



Figure 28-17 To address the cranial trunk and forelimbs, the rear limbs are placed over the exercise roll as the forelimbs are asked to balance the body weight during both stance and gentle movements.



Figure 28-18 A very challenging balance and proprioceptive exercise is to have the animal balance on an exercise roll with all four limbs. Support must be provided by the therapist to avoid possible injury.

provided by slowly rocking the therapy ball back and forth or side to side.

Functional Training Exercises for Balance and Proprioception

Standing or walking on foam rubber, mattresses, air mattresses, or trampolines alters the texture of the ground and challenges the animal's functional walking proprioceptive ability (Figure 28-19). Animals may also be walked over different surfaces, such as grass, concrete, a mulched path, a curb, sand, or other surfaces.

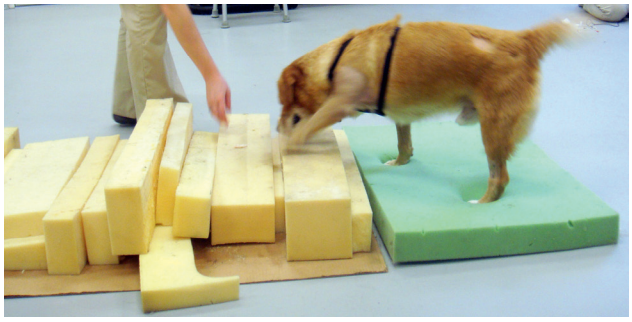


Figure 28-19 Standing or walking on foam rubber, mattresses, air mattresses, or trampolines alters the texture of the ground and challenges the animal's functional balance and proprioceptive ability.



Figure 28-20 Walking through vertical weave poles helps develop coordination and balance while learning to turn and pivot.

Animals must also develop balance and proprioception while performing activities of daily living, including turning, walking over obstacles, and negotiating stairs. Initially, dogs may be walked in large circles, going in both directions. The radius of the circle may be reduced as the animal improves, and patients may then progress to walking in a figure-eight pattern. Animals may also benefit by walking through vertical weave poles (Figure 28-20). In addition to helping improve general proprioception, patients also begin to learn how to contract their limb muscles to help stabilize joints while slowly pivoting and turning.

Some dogs must also perform certain activities in their home environments, such as negotiating stairs or walking over obstacles. Therefore walking up and down stairs may be an important activity to challenge proprioception in some patients. If possible, dogs may begin training on stairs with a gradual rise, walking slowly, and then progress to steeper stairs. Dogs may also walk over cavaletti rails to begin to judge how high to lift their legs to step over an obstacle. The height of the rails may vary between different strides, or the rails may be crossed at different



Figure 28-21 Walking over cavalletti rails helps dogs learn how to negotiate obstacles and walk over them by lifting their limbs to the appropriate height.



Figure 28-22 Walking over cavalletti rails while negotiating vertical weave poles provides additional challenges to balance and proprioception.

angles to provide additional challenges (Figure 28-21). Finally, dogs may negotiate vertical weave poles while stepping over cavaletti rails to provide additional challenges (Figure 28-22).

Summary

A rehabilitation program for a patient with proprioceptive and balance deficits does not need to be elaborate or costly. Consideration should be given to patient, owner, and therapist needs. Although the willingness of the owners to help with rehabilitation and the education level of the staff influence the rehabilitation program the most, some rehabilitation is better than none at all.

Therapeutic exercises are undoubtedly the most important aspects of rehabilitation. They may (and should) be incorporated with other modalities to enhance recovery, but using exercises that help improve proprioception and balance are among the earliest aspects of canine rehabilitation. In addition, most of these exercises help improve muscle strength, joint mobility, and limb use. Although a variety of techniques have been described, the ingenuity of the rehabilitation team, including the owner, will allow the development of other exercises that are specific to a patient's recovery. The keys to a successful therapeutic exercise program are to have site- and condition-specific exercises whenever possible, to use a variety of exercises and techniques to keep the therapy team and patient from becoming bored, and to allow the animal to appropriately progress to the next phases of rehabilitation.

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29 Therapeutic Exercises: Early Limb Use Exercises

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Therapeutic exercise is perhaps the most valuable modalities used in canine physical rehabilitation. Therapeutic exercise is an important method to assist an animal's return to the best function possible. In addition, the equipment needed is relatively inexpensive, and similar principles apply to a variety of individuals and conditions. Therapeutic exercise programs designed for the home environment also provide an opportunity for owners to become actively involved in their pet's rehabilitation.

Some of the common goals of early limb use exercises are to improve active pain-free range of motion; muscle mass and muscle strength to allow weight bearing, balance, and performance of activities of daily living; lameness; aerobic capacity; and weight loss and to help prevent further injury.[1-4]

When designing a therapeutic exercise program, several factors must be considered. A problem list is developed based on an initial evaluation of the rehabilitation patient, realistic outcome goals are established, and a treatment plan is formulated to address the identified problems. Therapeutic exercise is a significant component of the treatment plan. Appropriate exercises are those that can be performed safely and effectively and accomplish the therapeutic goals. When prescribing therapeutic exercise, the therapist should understand the diagnosis, identify the structure or structures involved, and recognize the stage of tissue recovery with the resultant functional limitations. It is important to remember that the force placed on the body with exercise should not exceed the strength of healing tissues.

The focus on patients that have serious afflictions with limited to no weight bearing on the affected limbs centers on early limb use and is the topic of this chapter. As patients are able to bear weight and begin to use their limbs, the emphasis shifts to improving proprioception and balance for patients with neurologic conditions or those recovering from joint injury or surgery, as described in Chapter 28. Finally, as patients improve with an approach to maximal expected function, the emphasis should be on strength, speed, and joint motion exercises, which are the focus of Chapter 30.

Treatment considerations and the choice of exercises vary with each stage of tissue repair and endurance. As the animal improves clinically and tissue healing progresses, the exercise plan should be altered to match the animal's progress and appropriately challenge the involved tissues. The intensity of an exercise may be increased or reduced by changing the duration of time that an animal performs an exercise, the frequency with which an exercise is performed, and the rate or speed at which a particular exercise is performed (Box 29-1). Any of these may be altered to fine-tune an exercise prescription to achieve the expected outcome goals. In general, the frequency of the activity is increased first if the patient is severely affected. For example, a realistic initial goal for a morbidly obese, deconditioned patient with degenerative joint disease may be to increase the amount of time the dog is able to comfortably tolerate walking by increasing the frequency of leash walks from twice daily to three times daily and finally to four times daily. This strategy helps to improve endurance and promote weight reduction, while limiting fatigue or muscle soreness. Increasing the length or duration of activity is usually the next phase of activity. For example, the dog may progress from walking 5 minutes four times per day to 6 minutes four times per day the next week. Increasing the speed at which the dog walks may not be a realistic initial goal for this animal at this point. A contrasting example may be an athletic animal recovering from injury that must be challenged to improve speed and endurance to meet the goal to return to performance activity. This type of patient may progress from jogging 20 minutes per session to playing ball in an enclosed run. It is important for the therapist to have an understanding of exercise intensity and what is appropriate for each patient during rehabilitation treatment. Routine reevaluation of the patient is recommended to assess the adaptations that are occurring with the rehabilitation treatment plan and to determine the appropriate rate of progression.

Common activities to encourage early limb use include standing exercises, controlled leash activities, ground treadmill activity, taping a bottle or syringe cap to the bottom of an unaffected foot to encourage weight bearing, slinging the contralateral good limb, and using balance balls or rolls. The exercise plan should target the affected muscles and other structures. The type of injury or repair must be considered when formulating a treatment plan. For example, the postoperative treatment of hip dysplasia

Box 29-1 Therapeutic Exercise Strategies

Exercise is generally increased as the patient and healing tissues allow:

1. Increase frequency of activity (e.g., from twice daily to three times daily, and then to four times daily).
2. Increase the duration of activity (e.g., from 5 minutes to 6 minutes).
3. Increase the speed or intensity of activity (e.g., increasing from a walk to a slow jog, and then from a slow jog to a medium trot).

In general, it is safe to increase the amount of activity 10-15% per week, as long as there is no increase in pain, decreases in function, etc.

differs depending on whether a femoral head and neck excision or a total hip replacement was performed. With this knowledge and understanding, appropriate decisions can be made regarding therapeutic exercise choices.

Therapeutic exercise routines should be monitored at regular intervals by a trained individual familiar with the patient and the exercise techniques. Inappropriate exercise or improper technique may result in inappropriate stresses, further injury, or exacerbation of an existing condition. Certain exercises may not be safe for the strength, flexibility, or endurance level of the animal performing the exercise, or an exercise may not be the correct one to accomplish the intended goal.

Standing Exercises

Assisted Standing Exercises

Patients with severe injuries or debilitating conditions may not be able to stand and support their own body weight. A period of standing, either completely or partially assisted, may strengthen the patient, aid in proprioceptive training, improve circulation and respiration, provide an opportunity to eliminate, and enhance the patient's psychological well-being. The purposes of assisted standing exercises are to encourage neuromuscular function, reeducate muscles, develop strength and stamina of supporting postural muscles, and enhance proprioception.

Maximal assisted standing is appropriate for patients that are unable to support their body weight as a result of paralysis, paresis, pain, trauma, postoperative precautions, or general debilitation. Maximal assistance may be defined as support of 75% to 100% of the patient's body weight by the handler to maintain a standing posture. Animals with multiple orthopedic injuries (especially pelvic fractures following repair or after a period of healing), neurologic conditions, or severe debilitation are excellent candidates for assisted standing exercises (Figure 29-1). Assisted standing exercises are among the first therapeutic exercises prescribed for severely afflicted animals that are



Figure 29-1 Support while standing is beneficial for patients with limited ability to stand and bear body weight. A sling may be used to provide support for maximal assisted standing of a patient.

unable to independently rise from a recumbent position or support their own body weight, and may begin in patients that are receiving adequate pain control following injuries that are stable. Animals with unstable injuries or in pain resist standing and may further injure themselves or the therapist while struggling.

It is appropriate to begin assisted standing exercises in patients with adequate muscle tone and some ability of the limbs to initiate motion and resist collapse of the body (adequate upper motor neuron function). Patients with lower motor neuron signs have little or no muscle tone and flaccidity of the limbs. Although patients with lower motor neuron conditions will likely benefit from standing exercises, it is unlikely that they will be able to fully support their weight, and some additional assistance will be required.

Depending on animal size and weight, the handler can manually lift or use an assistive device such as a pelvic sling, abdominal sling, or thoracic sling to assist the animal from a recumbent to a supported standing position. To perform standing exercises, a sling is placed under the cranial thorax, caudal abdomen, or both for conditions affecting the forelimbs, rear limbs, or both, respectively. After the animal is in a supported standing position, the handler or a second person manually places the animal's limbs in a normal stance position, with the feet positioned squarely on the ground. Even distribution of the animal's weight on the ground allows for greater stability and may provide position sense, which provides proprioceptive input and facilitates the animal's awareness of its joints.

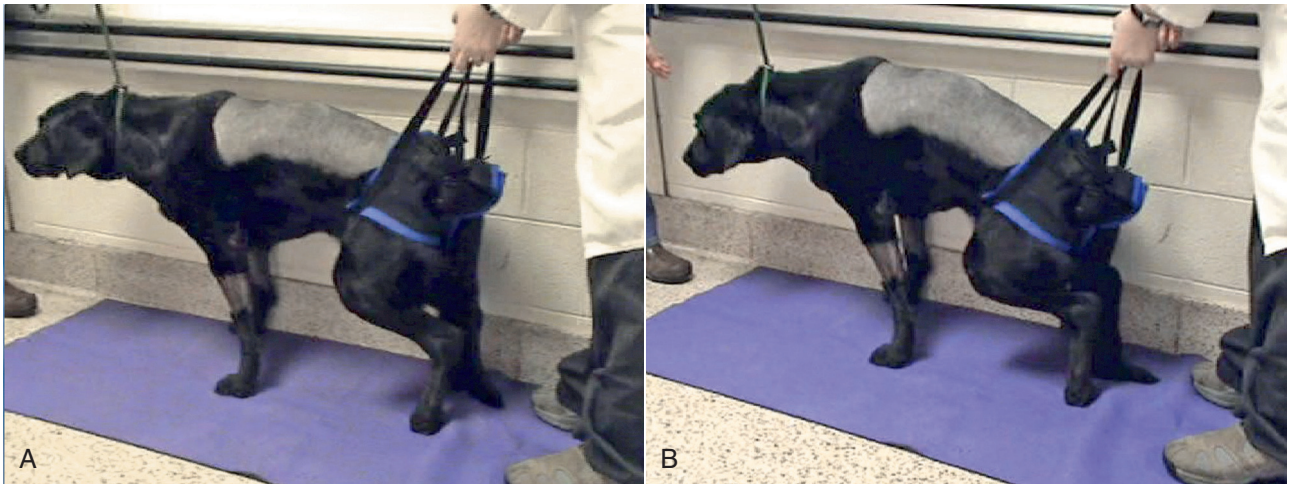


Figure 29-2 Tetraparesis or other conditions causing weakness in multiple limbs. **A,** Use of a sling to help provide support for standing. **B,** As the patient begins to fatigue and attempts to sit, the therapist lifts the dog back into a standing position.

Additional limb awareness can be encouraged via proprioceptive neurosensory facilitation techniques such as tickling the paw or distal segments, ascending circular touching, or back brushing the hair of the limbs.

The animal should be encouraged to support its body weight as it is able, and the therapist gives only the necessary assistance to maintain the standing position (Figure 29-2, *A*). While supporting the animal, the handler slowly releases tension on the assistive device, allowing the animal to continue weight bearing as much as it is able. Alternatively, it may be easier for the therapist to support the patient manually with a hand placed on the ventral pelvis to maintain the rear limbs in a normal position, preventing inappropriate crossing or a base narrow stance. Because the animal is encouraged to accept and support a portion of its weight, strength, balance, coordination, and proprioception are challenged. If the dog begins to collapse, the sling is gently pulled up to assist the patient back into the standing position and the exercise is repeated (Figure 29-2, *B*). It is important to place the limbs in a square standing position to give neuromuscular feedback and to also allow the animal to stand in a stable fashion.

During a treatment session, the animal may need periods of rest. Although the duration of each session should match the animal's tolerance, an initial starting goal is 5 to 15 repetitions two to three times daily, gradually increasing to standing 5 minutes per session. Individual patients progress at different rates. The therapist can keep track of and document the amount of time the animal is able to stand and how much assistance is needed.

Active Assisted Standing

As the animal becomes stronger and regains neuromuscular function, maximal assistance from the handler is not required. The progression from maximal assisted standing to active assisted standing occurs when the animal is able to actively support some of its weight, requiring only partial assistance from the handler (less than 75% of body weight). The animal should be encouraged to support as much of its weight as possible. The handler provides only the additional assistance required to maintain a standing position or prevent loss of balance. The amount of assistance required may be documented by recording the actual weight the handler is required to support for the animal to maintain a standing position, as well as the length of time the animal can maintain a standing position. The animal may require periodic rests during a treatment session. As the animal's strength and endurance improve, the duration of each session may be increased, the number of rest periods decreased, and the amount of assistance decreased.

Standby Assisted Standing

The next progression in standing activities is standby assisted standing. At this point of the recovery, the animal has the strength and motor control necessary to support itself against gravity in a standing position. However, it may still experience ataxia or weakness and have an occasional loss of balance, requiring standby assistance. The therapist should be behind the animal or at the animal's side, ready to guard against a fall caused by a loss of balance (Figure 29-3). The therapist does not assist the dog unless support is needed to prevent a fall. As strength improves, the patient will be more willing to advance the limbs if motor function is present. In cases of hindlimb paresis, the therapist may also challenge the patient further by lifting a forelimb to encourage weight bearing on the hindlimbs. It is also beneficial to lift a forelimb in dogs with unilateral hindlimb weakness to shift the weight to the hindlimbs.



Figure 29-3 The therapist is ready to provide support to a patient in the event of a loss of balance. The therapist does not assist the dog unless support is needed to prevent a fall.

Aquatic Standing

Standing in water may be a valuable activity for large patients with generalized weakness, lower motor neuron disease, or other conditions resulting in inability to stand and support weight. The buoyancy of the water often results in a patient's willingness to try to stand and bear some weight; often these patients will not even try to stand on dry ground. Supportive devices, such as life vests, slings, or harnesses may be used to provide further assistance and support. The reader is referred to Chapter 31 on aquatic therapy for further details.

Aids for Assisted Standing

Body Slings

Nonambulatory patients may benefit from placement in a body sling or harness to provide support for standing (Figure 29-4). It may be easier to use a lift device in conjunction with a sling to help support larger patients (Figure 29-5). An advantage of a sling is that the patient is able to maintain a body position with the limbs placed under the body in a standing position with partial or complete support of body weight, which provides an opportunity for limb strengthening and early proprioceptive training. Frequent episodes of supported standing also help to relieve pressure on bony prominences and may reduce the chances of decubital ulcer formation and pulmonary complications. Excessive congestion of the lungs that may result from spending long periods in lateral recumbency may be prevented with early implementation of standing in a sling. The body sling must be adjusted properly so that respiration is not compromised and the limbs are not compressed in the openings of the sling. Proper padding is also important for comfort. The skin should be inspected after each session in the sling to identify areas of potential skin irritation or breakdown.



Figure 29-4 A sling that may be used to assist standing. Standing helps the patient develop stamina and endurance with minimal assistance required from a therapist.



Figure 29-5 A lift device may be used in conjunction with a sling to help support larger patients.

Rubber bungee cords may be used to secure the sling to the supporting frame. This may aid in the facilitation of weight bearing and movement during the sling sessions. The elasticity of the cord permits some gentle up-and-down movement that encourages the animal to safely bear partial weight on its limbs. It also may allow some attempts to move forward, backward, and side to side. A gentle bouncing motion may be manually introduced by the therapist to initiate rhythmic stabilization of muscle groups throughout the trunk and limbs.



Figure 29-6 A variety of exercise rolls exist so that the appropriate size may be picked for nearly any size patient.



Figure 29-7 It is sometimes helpful to have very small dogs straddle a foam roll or roll of paper towels. In this photo, a dog is straddling a foam roll to facilitate neuromuscular electrical stimulation.

Exercise Rolls

A properly fitted inflatable exercise roll (physioroll, peanut) or a bolster may also be used to actively assist the animal with weight bearing, balance, coordination, strength, and weight shifting. The advantage of this form of assisted standing is that patients may stand independently, stand for longer periods, and avoid the problems inherent with long-term standing in a sling, such as compression of the upper limbs as they exit through the holes of a sling. A variety of exercise rolls exist so that the appropriate size may be picked for nearly any size patient (Figure 29-6). In very small dogs, it is sometimes helpful to have them straddle a foam roll or roll of paper towels (Figure 29-7). Using the roll as an assistive device also relieves stress on the handlers. Unless the animal is small, this technique may require two people to properly assist the patient into position.

Patients with limited ability to bear their own weight may be placed over an exercise roll such that the body straddles the roll with weight supported by the roll. The animal is placed in a standing position over an appropriately sized exercise roll (the roll provides greater lateral support and stability than a round exercise ball). The size should allow the animal to touch the ground with all four feet (Figure 29-8). If the roll is too tall, it may be deflated to meet the animal's height. Rolls that are slightly deflated are softer, more stable, and easier to work with than those that are fully inflated and very firm. The softer roll conforms to the animal's body better and is more comfortable. After the animal is secure on the roll, one person stabilizes the front of the animal and another, if needed, stabilizes the rear.

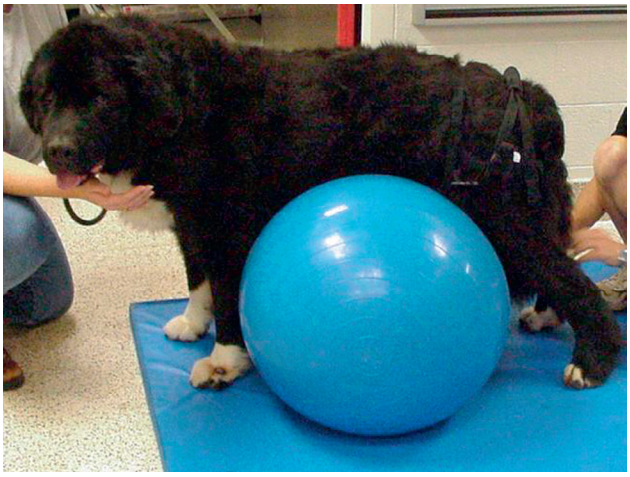


Figure 29-8 Patients with limited ability to bear their own weight may be placed over an exercise roll such that the body straddles the roll with weight supported by the roll. The animal is placed in a standing position over an appropriately sized exercise roll that may be used to provide additional challenges or increase weight bearing on the front or rear limbs or to assist standing (the roll provides greater lateral support and stability than a round exercise ball). The size should allow the animal to touch the ground with all four feet.

While the animal is supported in a standing position with paws on the ground, the handler can generate a very gentle up-and-down bouncing motion through the patient and the inflated roll. This provides proprioceptive input and may stimulate contraction of the supporting limb muscles. Dynamic balance and weight bearing may also be challenged in the thoracic or pelvic limbs as the therapists shift the roll forward and backward. As the animal becomes stronger, these same techniques may be performed at faster speeds to provide greater challenges to neuromuscular function and balance. It is recommended to perform standing over a roll during mealtimes, to allow the patient to focus on food and not on the therapist. This also provides positive reinforcement and relaxation for anxious patients and tends to decrease the extensor rigidity for brief periods in patients with upper motor neuron signs.

Carts

Mobility carts and their associated slings may be used as assistive devices for active assisted activities. Several manufacturers offer supportive carts for dogs, cats, and other animals. These carts are designed to assist with ambulation



Figure 29-9 A four-wheeled cart may be used for assisted ambulation in patients with tetraparesis.

of animals that are unable to support adequate weight for ambulation. Common indications for cart use are paralysis, paresis, severe arthritis, trauma in multiple limbs, and generalized weakness affecting one or more limbs. Carts allow the animal to have some degree of independence and reduce the lifting and support needed from a caregiver. Two-wheeled carts may be used for patients with active forelimb function, and four-wheeled carts are appropriate for patients with all limbs affected (Figure 29-9).

It is important that the cart be properly sized and fitted to ensure comfort and adequate function. Most manufacturers provide specific instructions regarding proper measurements to provide a custom fit. The therapist can assist with the measuring and fitting process. In addition, the cart should have adequate padding for comfort and to prevent chafing or breakdown of skin, but it should not interfere with the use of functional limbs.

If the animal has some functional use of the limbs, the devices should be adjusted to the patient's height so the feet may be placed (either manually by the therapist or independently by the animal) in a normal weight-bearing position on the ground. The patient should not dangle above the ground in the cart because this may compromise circulation and place undue pressure on soft tissues that are in contact with the supporting structures of the cart. Patients with poor motor or proprioceptive function may benefit from foot protection (rubber booties or infant socks) to prevent trauma to the dorsum of the paws. Patients with no motor function will likely need to have their feet maintained in a non–weight-bearing position.

Dynamic Ambulation Activities

Assisted Ambulation and Gait Training

If a dog is unable to walk independently, an assistive device may be used to support the animal as needed. Gait training



Figure 29-10 Commercial canine treadmills are available for rehabilitation.

should begin with the use of a fabric or elastic band sling, harness, or canine cart. The handler should assess the animal's needs before choosing the appropriate device. Encourage the dog to move slowly, allowing time for the dog to advance the limbs as independently as possible. Allow adequate time for the feet to come in contact with the ground with each step during the stance phase of gait. It may be necessary for the handler to manually assist the dog in the sequencing and placement of the limbs as the animal relearns the walking gait. If the animal moves too quickly, it will often avoid bearing weight on a painful limb, adopting an abnormal gait, such as a three-legged lameness, hopping, or dragging of a limb. The emphasis is on weight bearing with each and every step, by walking slowly. It is important for each person handling the dog to be consistent and not allow the dog to move too quickly. For food-motivated dogs, choosing a highly desirable treat that is only used when encouraging movement can help positively associate movement or the active attempts at movement during a therapy session. In some cases, dogs may be stimulated to ambulate to a greater degree if other animals are present and a game of "assisted chasing" ensues.

Treadmill Walking

Walking on a treadmill is very useful in rehabilitation. Most dogs trained to a leash readily take to treadmill walking in one or two sessions. A variety of treadmills are available for use, including commercial canine treadmills (Figure 29-10). A number of models available for human use may be modified for canine use by adding an overhead bar with a support system to which a canine harness can attach. A harness is useful to help support the dog in case it stumbles or falls. Alternatively, a therapist may support the dog with a sling while on the treadmill. Side rails or fences placed on both sides of the treadmill are useful if a dog tends to step off to the side. Other useful features include variable speed control, a timer, and the ability to change the incline or decline of the surface.

Treadmills are useful for patterning gait and encouraging initial weight bearing following surgery. When the dog stands with the foot carried near the ground, it will generally begin to bear weight when it is walked on a treadmill. It is important that the treadmill not face toward a wall; rather, it should face toward a hallway or the middle of a room to encourage unimpeded walking. Having one person standing in front of the dog with words of encouragement or treats and one person straddling the dog behind is helpful in the early training stages to keep the dog walking straight.

Walking on a treadmill for the first time is a new and sometimes awkward experience. Animals are not used to the ground moving under them, so when they walk on a moving belt, proprioception, coordination, and balance are challenged. The ground moving underneath the dog often encourages a dog that is non–weight bearing on a limb as a result of an orthopedic condition to begin using the limb. In many instances, the animal will continue to use the limb even after the treadmill session is over. For patients with neurologic conditions, it may be necessary to support the dog with a sling or harness. The therapist may stand or squat beside a dog and manually advance a foot during the normal gait sequence to correct abnormal foot placement, such as knuckling over, and encourage gait reeducation.

Assisted active advancement of a limb during the swing phase of gait can also be performed by using an elastic band. An animal with conscious proprioceptive deficits or muscle weakness may have difficulty advancing the limb during the swing phase of gait; these patients can be assisted by using an elastic band to help advance the limb. After one end of the elastic band is attached to the foot or just above the hock, the other end is secured to the body with the direction of pull designed to assist active advancement of the limb during gait. The handler may also use the elastic band to advance the limb and manually place the foot as the animal walks on the treadmill. A rigid toe extension attached to a boot, syringe cap, or any mild irritant (such as a marble or bottle cap) placed on the bottom of the contralateral, unaffected foot may encourage weight bearing in patients that are reluctant to place weight on a limb (Figure 29-11).



Figure 29-11 A syringe cap, rigid toe extension attached to a boot, or any mild irritant (such as a marble or bottle cap) placed on the bottom of the contralateral, unaffected foot may encourage weight bearing in patients that are reluctant to place weight on a limb.

Aquatic Therapy

In addition to standing in water, patients that have some motor function may greatly benefit from walking in water or on an underwater treadmill. The buoyancy of the water, combined with ambulation, results in support while walking. Further support may be provided by using life vests, slings, harnesses, or an assistant in the water. Elastic bands may be used to help advance limbs in the water. After each aquatic use, the bands should be laid flat and allowed to thoroughly dry to prevent degradation and tearing of the elastic. Assisted swimming is also possible in patients, especially smaller patients recovering from neurologic conditions, such as intervertebral disk herniation surgery. Patients with little motor function may simply kick the limbs in an uncoordinated fashion in the beginning, but such activity results in muscle contraction and strengthening and promotes recovery of neuromuscular coordination and function. It is important to provide assistance and support and not allow vigorous swimming initially. As the patient improves, the assistance is gradually reduced.

Aids for Dynamic Ambulation

Walking Slings

Commercially available canine slings should be durable, flexible, and washable and should conform to the body. They are available for the forelimbs and hindlimbs and for trunk support. When using rear limb slings it is important that bowel and bladder function are not obstructed. Adjustable-length handles for the therapist to grip make a sling a more ergonomic choice of assistive device than a bath towel because the sling allows the handler to stand erect without bending forward while carrying the weight of the dog and reduces excessive stress to the handler's forearm, wrist, shoulders, and back. This is an important concern, especially if the handler will be assisting a large dog several times a day for any length of time. It is important for persons handling the animal to practice proper body mechanics while assisting the canine patient to avoid injuring themselves. The ideal sling length and position for the handler is to simulate carrying a suitcase or large bag, and not bending over to stress the back, while making sure the patient is able to position the paws in a weight-bearing position without dragging the toes or dorsum of the paw on the ground. The sling may be used for both orthopedic and neurologic patients in need of assisted ambulation.

Towels

A long towel may be used as a last resort to support an animal that requires assistance with standing or ambulation. Towels are readily available, inexpensive, and washable but can be problematic and are often more difficult than slings with handles for the therapist or owner, especially in patients requiring long-term assistance. When assisting the dog's hindlimbs, the handler should place the towel around the abdomen just in front of the hindlimbs in the inguinal region. Nylon straps are a highly recommended modification and may be sewn onto the ends of the towel to provide added length so the handler can maintain proper body mechanics.

A disadvantage of towels on larger dogs with urinary incontinence is that it puts pressure on the urinary bladder and may cause it to be expressed. This is less likely to happen with a properly fitted commercial pelvic sling.

Elastic Bands

It is often beneficial to "graduate" a patient from a more supportive sling into a more dynamic sling. Elastic bands used for gait patterning or resistance exercises are an excellent means to provide some support and concurrent dynamic feedback during weight bearing. There are several levels of resistance and thickness that should be matched to the patients' size and functional ability. Lighter weight bands are ideal for smaller patients even as an initial sling. Heavier weight slings can be used in medium to large dogs with moderate functional ability both as a support and a gait challenge. The elastic band in this stage can be used to pull the patient off balance to one side, while providing enough support to "catch" the patient so it doesn't fall during walking or standing exercises. Bands may also be attached to harnesses to provide resistance during gait.

Carts

Mobility carts are appropriate for temporary use as the animal regains its ability to ambulate independently, as well as for permanent use for animals that will not regain full function. After a period of acclimation for both the animal and owner, a properly fitted cart may result in a positive change in the quality of life for both. When the animal is mobile in a cart, that mobility fosters independence that may have a profound positive effect on the animal's attitude. Carts with fabric sling supports can be readily adjusted to allow preferential support or challenge to individual limbs. Additionally, elastic bands tied to the cart can be used to provide additional resistance for individual limbs or to help a weak limb advance, similar to elastic band use on land treadmill exercises.

The home environment may need to be altered to allow the animal to freely ambulate in the cart without encountering dangerous conditions. The animal's exercise area should be free of obstructions that could hinder the animal's ability to safely ambulate in the cart. Particular caution should be taken to prevent falls down stairs.

Independent Ambulation

Leash Walking

Slow leash walks are perhaps the most important exercise in the early rehabilitative period, and they are commonly performed incorrectly. Walking the animal slowly encourages the use of all limbs in a sequenced gait pattern. Walks must be slow enough to allow weight bearing; if the dog is walked too fast, the tendency is to simply hold the intended limb up in a flexed position and not bear any weight on it. Slow leash walking is indicated when the animal is reluctant to use a limb as a result of pain, weakness, or proprioceptive deficits. Slow leash walks encourage placement of each limb on the ground, increasing stance time and weight bearing.

If there are no contraindications to weight bearing, slow leash walks may begin very soon after most orthopedic procedures. Behavior modification is important. The dog should be praised when touching the limb to the ground, and not praised when the leg is held up. As the animal regains use of the affected limb and is consistently able to place the limb at a slow leash walk, the pace of the walk may be increased. Faster walks further challenge balance, coordination, proprioception, and cardiorespiratory endurance, as well as functional muscle strengthening and endurance. When an animal is weight bearing well at slow and fast speeds, an additional challenge is to focus on speed transitions while walking. Often the patient will have difficulty increasing or decreasing speeds, which requires significant muscular control and coordination in animals recovering from surgery, neurologic conditions, or significant musculoskeletal injuries. Animals recovering from neurologic conditions may be challenged by gently pushing the animal off-balance while walking, using the handler's leg or an elastic band sling, thus encouraging proprioceptive development. The therapist must be certain that the animal is not unbalanced so much that it falls, however. When appropriate, the therapist may alter the exercise treatment plan to include fast walking, slow jogging, and running on a long lead.

Treadmill Walking

Treadmills may be useful during rehabilitation programs as a continuation of assisted ambulation or for conditions in which extension of the hip or stifle is painful, such as hip dysplasia or postoperative recovery from cranial cruciate ligament surgery. Normally, patients are reluctant to perform activities such as climbing stairs, because extension of these joints is painful. Treadmill walking is less painful in some patients because the belt provides assistance with hip and stifle extension by helping to pull the rear limb caudally. There is less need for active contraction of the gluteal and quadriceps muscles for joint extension when walking on a treadmill than when walking on land. The treadmill may be angled up or down to reduce or increase the forces placed on the forelimbs or hindlimbs.



Figure 29-12 Walking or jogging the dog up inclines aids in strengthening of the quadriceps, semitendinosus, semimembranosus, and gluteal muscles with relatively low-impact activity.

The speed and length of treadmill walking may be increased as the animal recovers and gains strength and stamina. Also, varying intervals of high and low speeds within a session may be introduced.

Inclines and Declines

Walking or jogging the dog up inclines aids in strengthening of the quadriceps, semitendinosus, semimembranosus, and gluteal muscles with relatively low-impact activity (Figure 29-12). Rear limb muscle strength is required for the dog to propel itself up an incline, whereas forelimb muscle strength and eccentric contraction of both forelimb and hindlimb muscles are required for the dog to brake itself while going down a declined surface. Walking should be done slowly and on leash; otherwise the dog may only toe-touch with the limb or hop in a non–weight-bearing fashion. In addition, if the head is held up slightly while walking up an incline, the weight will be shifted caudally, requiring the animal to drive up the hill with the rear limbs and use the muscles to a greater extent. Conversely, if the head is held in a downward position, the weight will be shifted cranially, requiring the forelimb muscles to engage for braking. It is often easier to control the dog's head and body position on inclines if wearing a halter and harness rather than a neck lead.

Weight bearing while climbing promotes strengthening of the quadriceps and gluteal muscles. When the limb is in the stance phase of gait and the body is traveling forward, the stifle and hip must extend to propel the animal forward. If extension is painful, the dog's stance time on the limb may be shortened and an altered gait may result. Inclines and declines should be introduced slowly, beginning with gradual slopes. As the dog's range of motion and strength improve, the dog may be challenged by walking up longer, steeper slopes and by increasing the duration and speed of the climbing exercise.

Walking down declines is useful for strengthening the triceps muscles, and is typically more difficult for hindlimb conditions because it requires the dog to reach under the body with the hindlimbs, requiring flexion of the hock, stifle, and hip. Start with gently sloping, short declines and progress to steeper ones as the dog is able. If an incline or decline is too challenging, an easier introduction is to walk on a gently sloped hill in a serpentine pattern or on the diagonal up, down, or both until the patient is strong enough to navigate the incline and decline in a straight line. Additionally, diagonal climbing and descent can be used to alter the challenge to an affected limb depending on if the limb is uphill or downhill. For example, if the affected limb is on the downhill side while ascending diagonally, the joints and muscles perform with the limb in a more extended position. Conversely, if the affected limb is on the uphill side while ascending diagonally, the joints and muscles perform with the limb in a more flexed position.

Other Methods to Encourage Weight Bearing

In some situations, a patient may be reluctant to use a limb after injury or surgery, even though the stage of recovery is such that limb use would normally be expected. It is important to always rule out any pathologic causes of poor limb use, such as failed implants, infection, other concurrent conditions, and so on. If there is no pathologic cause of poor limb use, and pain is adequately controlled, other measures may be used to encourage limb use.

A syringe cap, coin, or marble may be placed on the bottom of the uninjured foot and held in place with adhesive tape to encourage weight bearing on the affected limb (see Figure 29-11). Also, creating limb length differences with rigid or semistiff toe extensions attached to the foot with an elastic wrap or attached to a boot may also be used for gait perturbations. In many instances, weight bearing continues on the affected limb even after the item is removed from the good foot. Some dogs will build up a tolerance to the item; therefore continuous application is not recommended. In some particularly difficult cases, a nonweight-bearing sling may be placed on the nonaffected limb to force the animal to bear weight on the affected limb. In our experience, this technique is less effective because many animals simply lie down and will not attempt to walk.

Side bending and vertical head movements to follow a toy or treat are often useful to encourage affected limb use. The patient should be positioned in an even, square stance with all four limbs and slowly asked to bend to the left and right, with the goal being to touch the nose to the stifle. For vertical movements, the dog should reach up slowly to a comfortable point and then down, with the goal being to position the head between the forelimbs. This exercise not only enhances spinal flexibility but also redistributes body weight to encourage weight bearing and position awareness of the affected limbs. Caution should be exercised in patients with nonsurgical spinal injuries or postoperative

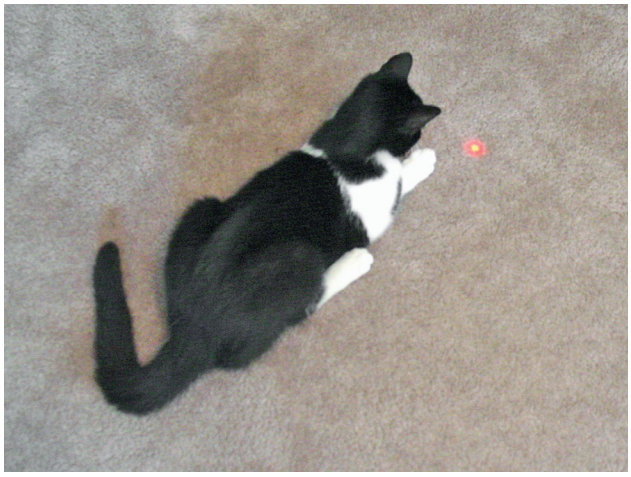


Figure 29-13 Many cats will readily chase the spot of light from a flashlight or laser pointer around a room. Care should be taken to have good footing, such as carpeting.

spinal cases, and movements should be minimal and well within the voluntary range of comfort for the patient. This technique is contraindicated in animals with an unstable spinal canal or those with unstable intervertebral disks.

Hemiwalking is sometimes useful to encourage weight bearing on a particular limb. This exercise is performed by elevating the ipsilateral fore- and rear limbs off the ground and shifting the weight to the contralateral limbs. The animal must move the contralateral fore- and rear limbs laterally to avoid losing balance and falling. Although this is generally more successful than placing a sling on a limb, some animals may still resist and simply lie down.

Many cats will readily chase the spot of light from a flashlight or laser pointer around a room. Care should be taken to have good footing, such as carpeting, and to move the light at a speed appropriate for the animal's stage of recovery (Figure 29-13). Feathers and reflective material on a string or stick are also useful as a lure in feline rehabilitation. In addition, stretching may be employed by moving the light or feather along a vertical surface, such as a wall. Some dogs will also chase a laser pointer light. Caution is advised using this technique because some animals, particularly dogs, may become obsessive-compulsive and develop chronic or permanent behavior problems when encouraging light-chasing exercises.

Many cats and dogs have been well conditioned to the sound of a can opener or other audible cues such as a door opening to go for a walk. The animal may be placed in another room and the sound made. In many instances, the animal disregards a lame leg and ambulates to the sound.

Most cats and some dogs will play with a toy such as a string or other object that they are used to "stalking" or chasing. Engaging the owner to identify some favorite playthings is important, because some animals will not react to toys in a strange environment such as a clinic. It is equally important to instruct the owner regarding the length of playtime, how to use the toy to maximize the rehabilitation process, and limits of play to reduce the chance of fatigue and injury.

Progression of Exercises

Many variables must be considered in the design of a therapeutic exercise program, including the type and severity of the condition; the stability of any surgical repairs; the number of involved limbs and joints; the size of the animal; the preexisting physical condition of the patient; the available facilities; the skill and experience of the therapist; and whether the exercises will be administered by a professional, the owner, or a combination of the owner and a professional. Unfortunately, it is impossible to have a single protocol for each specific condition. Some basic guidelines may be applied, however.

The goal of any therapeutic exercise program should be to restore the animal to as full and active use as possible. The goal may need to be periodically adjusted if the patient progresses to a greater or lesser degree than expected. The initial goal should be to have the patient bear its full weight while standing. The next step should be assisted active ambulation. Proprioceptive training should occur concurrently with active ambulation, with the goal to have unassisted active ambulation while maintaining balance. As the animal nears return to house-pet function, strategies should be developed to encourage increased weight bearing and muscle mass.

It is very important to perform the exercises correctly and to maintain a consistent level of activity daily or every other day. In most cases, several short sessions per day will be more effective than a single long session early in the recovery period. Overdoing activity on the weekends with relatively little activity during the rest of the week will likely be detrimental to the animal's recovery; regular, daily activity will avoid the "weekend warrior" syndrome.

A general rule of thumb is to increase the activity by 10-15% each week. If pain or lameness increases during or several hours after stepping up an activity, the level of activity should be reduced by 50% for 3 to 7 days. If the lameness and pain resolve, the activity may be slowly increased back to the pre–step-up activity level over 3 to 5 days, with careful observation to be certain that the activity is not increased too aggressively or rapidly. The rate of subsequent step-up activity should be half of what it originally was to avoid a recurrent episode of exacerbated lameness and pain.

It is also important to vary the routine for the animal's and the owner's sake to avoid boredom, and to allow incorporation of function-specific activities in the animal's home or work environment. During chronic management of the patient, weight control is extremely important. It is also important to continue to oversee the protocol and document improvement. Progress will occur at a slower

rate during the chronic phase of recovery, but continued rehabilitation is just as important to achieve as full a return to activity as possible.

Several medications may be appropriate for use during rehabilitation, including nonsteroidal antiinflammatory agents and chondroprotective agents. Caution should be used, however, to be certain that any analgesic medications do not mask pain and discomfort associated with the level of activity. They should facilitate rehabilitation and recovery rather than allow rehabilitation to progress faster than normal conditions and tissue healing should allow. When stepping up the level of an activity, antiinflammatory or pain medication may be withheld on the day of step-up and for the day after, to be certain that there is no additional tissue damage. If pain or lameness increases, the level of activity should be reduced 50% for 1 week; then the activity is stepped up again at a slower rate.

Summary

Beginning a rehabilitation program need not be elaborate or costly. Consideration should be given to the patient's needs, the owner's needs, and the therapist's needs. Also, although protocol development greatly depends on the available facilities and equipment, the willingness of the owners to help with rehabilitation, and the education level of the staff, some rehabilitation is better than none at all.

Therapeutic exercises are undoubtedly the most important aspects of rehabilitation. They may (and should) be incorporated with other modalities to enhance recovery, but it is the exercises that help improve muscle strength, joint mobility, limb use, proprioceptive training, and endurance to maximize function. Although a variety of techniques have been described, the ingenuity of the rehabilitation team, including the owner, will allow the development of other exercises that are specific to a patient's recovery. The keys to a successful therapeutic exercise program are to have site- and condition-specific exercises whenever possible, to use a variety of exercises and techniques to keep the therapy team and patient from becoming bored, and to allow the animal to appropriately progress so that tissues are adequately challenged for strengthening, but not so rapidly as to result in complications and tissue damage.

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30 Therapeutic Exercises: Joint Motion, Strengthening, Endurance, and Speed Exercises

Darryl L. Millis, Marti Drum, and David Levine

Therapeutic exercises to improve active joint motion through a more complete range, and to build power, strength, and speed are the final phases of rehabilitation to return the patient to as normal a function as possible. The therapist must keep in mind the degree of injury or disability in determining the likelihood for the patient to reach higher levels of function and adjust the rehabilitation program and expectations accordingly. For example, it is not expected that a 7-year-old dog with severe bilateral elbow arthritis would be able to achieve near-normal function, but there are exercises that can be performed to help achieve greater function and ability. Some of the common goals of therapeutic exercise are to improve active pain-free range of motion (ROM), muscle mass and muscle strength, performance, and conditioning. Common activities include stair climbing, sit-to-stand exercises, treadmill activity with resistance, pulling or carrying weights, "wheelbarrowing" (for forelimb activity), "dancing" (for rear limb activity), walking and trotting across cavaletti rails, jogging, running, underwater treadmill activities, swimming, playing ball, and activities using balance balls or rolls. These higher levels of therapeutic exercise are important in assisting an animal's return to the best function possible following early limb use and proprioceptive exercises. Many of the exercises that target certain aspects of performance, such as joint motion, conditioning, and strength, have some overlap (Box 30-1). In addition, the equipment needed for most exercises is relatively inexpensive, and similar principles apply to a variety of individuals and conditions. Therapeutic exercise programs designed for the home environment also provide an opportunity for owners to become actively involved in their pets' rehabilitation. In any case, it is very important to have good footing to avoid slipping and to have adequate space to perform exercises with as little distraction as possible.

It is also important to remember to use patience when working with the dog to learn new exercises. Most of the exercises in this chapter have been purposefully selected so that most dogs that are trained to leash can perform them with some basic training. However, some dogs are highly trained and the therapist should work with the owner to select exercises based on the patient's needs, as well things the dog is already trained to perform. The therapist is cautioned to use training techniques that are based on positive reinforcement. Negative reinforcement techniques may temporarily work in some instances, but because rehabilitation is an ongoing treatment, dogs will become less willing to perform an activity or to learn new ones. Many dogs will be somewhat nervous or frightened when first undergoing rehabilitation, especially if pain is involved from their injury or surgery. It is important to go slowly and gently at the beginning. Treats, favorite toys, or other rewards should be kept handy to encourage dogs when learning new exercises. The reader is referred to Chapter 4 for further tips on applying behavior techniques in the rehabilitation environment.

When designing a high-level therapeutic exercise program, several factors must be considered. A problem list is developed based on an initial evaluation of the rehabilitation patient, and a treatment plan is formulated to address the identified problems. Realistic outcome goals are then established. Appropriate exercises are those that can be performed safely and effectively and accomplish the therapeutic goals. The exercise plan should target the affected muscles, joints, and other structures. When prescribing therapeutic exercise, the therapist should understand the diagnosis, identify the structure or structures involved, and recognize the stage of tissue recovery with the resultant functional limitations. Without a good understanding of these principles, it is easy to over stress the involved tissues and potentially cause harm. The type of injury or repair must be considered when formulating a treatment plan. For example, a patient with a tibial plateau leveling osteotomy (TPLO) will require several weeks for tissue remodeling and healing of the osteotomy before engaging in high-speed activities. Similarly, a patient with a partial brachial plexus injury may initially have a difficult time performing a shaking hands exercise. But over time, the dog may recover and perform shaking hands exercise with a limb weight to achieve greater strengthening. With this knowledge and understanding, appropriate decisions can be made regarding therapeutic exercise choices.

Treatment considerations and choice of exercises vary with each stage of tissue repair and endurance. As the animal improves clinically and tissue healing progresses, the exercise plan should be altered to match the animal's progress and appropriately challenge the involved tissues. The intensity of an exercise may be increased or reduced

Box 30-1 Exercises to Improve Joint Motion, Conditioning, and Strength

Joint Motion Exercises

Incline and decline walking
Stair climbing
Dancing
Wheelbarrowing
Sit-to-stand exercises
Cavaletti rails
Underwater treadmill activities
Swimming
Tunnel and limbo walking

Conditioning Exercises

Incline and decline walking
Stair climbing
Treadmill activity
Jogging
Running
Underwater treadmill activities
Swimming
Playing ball
Sport-specific activities

Strength Exercises

Incline and decline walking
Stair climbing
Treadmill activity with resistance
Dancing with resistance
Wheelbarrowing with resistance
Running up stairs or inclines or down declines
Underwater treadmill activities with speed or resistance, floatation devices
Swimming
Pulling or carrying weights
Playing ball
Activities using balance balls or rolls (core strengthening)
Rhythmic stabilization
Lateral hopping with resistance
Sit-to-beg for core strengthening
Sit-to-stand with backpack or weights
Jumping
Limbo walking
Sport-specific activities

Feline Exercises

Proprioceptive exercises (e.g., weight-shifting activities, balance board, wobble board, and standing on a therapy ball or roll)
Walking with a harness
Ground treadmill walking with support and assistance
Treadmill walking with small leg weights or therapy bands
Playing with feathers or toys tied on the end of the string
Scratching post
Can opener
Low cavaletti rails
Chasing a flashlight beam or laser light (use caution to avoid obsessive-compulsive light chasing)
Aquatic therapy

by changing the duration of time that an animal performs an exercise, the rate of speed at which a particular exercise is performed, or the frequency (how many times per day) that it is performed.

These factors may be altered to fine-tune an exercise prescription to achieve the expected outcome goals. For example, a realistic initial goal for a morbidly obese, deconditioned patient with degenerative joint disease may be to increase the amount of time the dog is able to comfortably tolerate walking, thus improving endurance and promoting weight reduction. Increasing the speed at which the dog walks may not be a realistic initial goal for this animal. A contrasting example may be an athletic animal recovering from injury that must be challenged to improve speed and frequency to meet the goal to return to a high level of performance. It is important for the therapist to have an understanding of exercise intensity and what is appropriate for each patient during rehabilitation treatment. Routine reevaluation of the patient is recommended to assess the adaptations that are occurring with the rehabilitation treatment plan and to determine the appropriate rate of progression.

Therapeutic exercise routines should be monitored at regular intervals by a trained individual familiar with the patient and the exercise techniques. Inappropriate exercise or improper technique may result in inappropriate stresses, further injury, or exacerbation of an existing condition. Certain exercises may not be safe for the strength, flexibility, or endurance level of the animal performing the exercise, or an exercise may not be the correct one to accomplish the intended goal. The exercises described in this chapter are listed with the desired outcomes of joint motion, conditioning, or speed and strength, but there is significant overlap with some exercises, and the differences are indicated for the various outcomes. The reader is also referred to Chapter 24 on biomechanics of rehabilitation for further details on therapeutic exercises, especially as they relate to joint motion and forces.

Joint Motion Exercises

Incline Walking

Walking the dog up an inclined surface alters joint motion and aids in strengthening of the hip and stifle muscles, especially the extensors, while ascending (Figure 30-1).

Joint Motion Considerations

Climbing up an inclined surface promotes increased flexion, extension, and ROM of the hip. Stifle flexion is

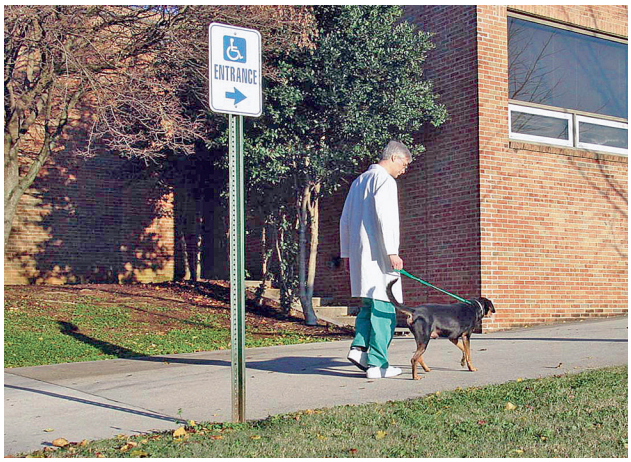


Figure 30-1 Leash walking up ramps or hills encourages muscle strengthening and cardiovascular endurance.



Figure 30-2 Walking up a ramp or hill while wearing a leg weight provides greater challenges.

decreased, however.[1] When the limb is in the stance phase of gait and the body is traveling forward, the knee and hip must extend to propel the animal forward. If extension is painful, the dog's stance time on the limb may be reduced and an altered gait may result. In addition, dogs may shorten the stride length to help control joint motion if a particular joint position causes pain. For example, a dog with hip dysplasia may shorten its stride while walking up an incline so that excessive hip extension and pain are avoided. If this occurs and a particular joint motion that is desired does not occur, it may be better to use another exercise, such as stair climbing, in which the limb must travel a discrete distance to negotiate the step rather than adjust the stride length while walking up a ramp.

Walking up an inclined ramp also results in greater ROM of the forelimb joints.[2] Shoulder extension and ROM, elbow flexion and extension, and carpal flexion are greater when walking up a ramp as compared with ascending stairs or trotting over level ground.

Exercise Considerations

Initially, walking should be done slowly and on leash; otherwise the dog may only toe-touch with the limb or hop in a non–weight-bearing fashion. Inclines should be introduced slowly, beginning with gradual slopes. As the dog's ROM, strength, and limb use improve, the dog may be challenged by walking up longer, steeper slopes and by increasing the duration, speed, and frequency of the climbing exercise. Further challenges are provided by doing inclines while wearing a weighted vest, leg weights (Figure 30-2), or pulling a small amount of weight with a harness. Ascending or descending a slope at an angle may also alter joint motion of an affected limb. If the affected limb is on the downhill side while ascending diagonally, the joints and muscles perform with the limb in a more extended position. Conversely, if the affected limb is on the uphill side while ascending diagonally, the joints and muscles perform with the limb in a more flexed position. It is particularly important to monitor gait patterns when walking downhill as many dogs with neurologic conditions may exhibit more ataxia, causing the hind end to sway to one side or the other. Also, if descending too fast, the patient may stumble and fall even if its gait appears to be normal on level ground. Varying speeds up and down inclines is another method to increase the challenge of this exercise.

Stair Climbing

Climbing stairs is useful to improve ROM, power in the rear limb extensors, coordination, and proprioception, and in many instances, is an activity of daily living that must be performed in the home environment (Figure 30-3). Quadriceps and gluteal muscle groups are strengthened as the animal pushes off, extending the stifles and hips while propelling the body up the steps.

Joint Motion Considerations

ROM of the pelvic limb joints is significantly increased during stair climbing compared with trotting on level ground.[3,4] Extension of the hip and hock joints is moderately increased with stair climbing, whereas stifle extension may be mildly increased or decreased, depending on the size of the dog relative to the size of the step. Flexion of the stifle and hock is also increased. Although hip flexion contributes little to stair ascent, the stifle and hock joints seem to contribute significantly to ascending stairs by flexing these joints to raise the limb up to the height of the step.

Regarding the forelimb joints, there is less ROM with stair ascent as compared with walking up a ramp with similar slope.[2] These differences are primarily due to changes in extension, with no significant differences in flexion of forelimb joints. Shoulder extension is less with stair climbing as compared with trotting over ground,



Figure 30-3 Stair climbing should begin on stairs with a gradual rise (*A*) and progress to steeper stairs (*B*). Care should be taken to be certain that the dog pushes symmetrically off of both rear limbs.

whereas flexion is greater with stair climbing. Elbow and carpal flexion, extension, and ROM are greater with stair ascent as compared with trotting over ground.

Stair descent results in significantly greater stifle flexion and hock flexion and extension compared with walking down a declined surface.[5] Significantly greater ROM also occurs in the coxofemoral, femorotibial, and tibiotarsal joints during stair descent.

Exercise Considerations

The dog must begin slowly climbing stairs to encourage proper use of the rear limbs, as opposed to simply carrying the limb, hopping with both rear limbs, or skipping up stairs. Encourage the dog to go slowly and deliberately, climbing the stairs in a reciprocal stepping gait. Stairs should be introduced slowly because this is a challenging exercise for both the musculoskeletal and cardiovascular systems and the animal may fatigue quickly. Initially, some dogs may require assistance from the handler. Begin with five to seven steps, and gradually increase to two to four flights of stairs once or twice daily. In severely affected dogs or smaller dogs, it is advisable to begin stair climbing on stairs with a gradual ascent and progress to steeper stairs. Another strategy for smaller dogs is to attach blocks to existing stairs to make "half stairs." As the patient improves, dogs may jog up stairs. In the motivated patient, walking backward up or down stairs can be an additional significant challenge. To introduce this exercise, use a treat or toy to encourage stepping backward up one step while the patient keeps the forelimb on the ground and then have patient come back down to level ground. The reverse is true for backing down a step. Repeat this several times until the patient is comfortable with this movement before having the patient walk backward up or down two to three steps, and advance to a full flight after the patient is comfortable and understands what is being asked. This advanced exercise is often easier to teach using a few steps versus a full flight of stairs. A three- or four-stair block can be built for this purpose.

Dancing and Wheelbarrowing

Raising the forelimbs off the ground and walking the dog forward or backward, also called *dancing,* alters motion of rear limb joints and challenges proprioception, coordination, and strength. The amount of peak force placed on the limbs is slightly greater than what would occur at a trot.[6] Therefore if the stage of recovery is not adequate to withstand trotting, then dancing should not be undertaken. On the other hand, additional strengthening of postural muscles may be obtained with dancing when it is safe to perform the exercise.

Wheelbarrowing is an exercise similar to dancing, except that the forelimbs are targeted by raising the rear limbs off the ground and walking the dog forward. This exercise encourages increased use of the forelimbs and challenges proprioception, coordination, and strength. The dog's orthopedic condition must be adequately stable to handle the stresses of these exercises. Wheelbarrowing results in peak force placed on the limb that is between walking and trotting.[6]

Joint Motion Considerations

Forward dancing results in significantly less hip flexion, total hip ROM, hock flexion, and hock total ROM compared with walking.[6] Dancing backward results in greater hip extension and stifle flexion, whereas hip flexion, hip ROM, stifle extension, and stifle ROM are less than regular walking.

Wheelbarrowing alters motion of the forelimb joints, increasing shoulder extension and elbow flexion as compared with walking.[6]



Figure 30-4 Dancing encourages weight bearing on the rear limbs. Muzzling may be necessary for some dogs before this exercise, particularly in dogs that do not like to have their feet touched or feel threatened by the therapist's posture. The forelimbs are lifted off the ground, allowing the patient to bear weight only on the rear limbs.



Figure 30-5 Dancing activities may also be accomplished with assistance of a physioroll or egg ball. The patient places their front limbs on the roll or ball and the therapist slowly rolls the ball toward or away from the patient forcing them to move their hindlimbs backward or forward.

Exercise Considerations

It is important for the therapist or veterinarian to evaluate available ROM in the rear limb joints before attempting dancing to identify any potential limitations that may prevent the animal from safely performing the exercise. For example, in the early phases of a therapeutic exercise program for a dog that is painful with hip extension as a result of hip dysplasia, gluteal muscle strengthening may be more comfortable by dancing the dog forward rather than backward.

When a dog is using its affected limb consistently at a walk with minimal lameness, dancing may begin. Muzzling may be necessary for some dogs before this exercise, particularly in dogs that do not like to have their feet touched or feel threatened by the therapist's posture. The forelimbs are lifted off the ground, allowing the patient to bear weight only on the rear limbs (Figure 30-4). Dogs with normal proprioception will naturally move the rear limbs as the handler moves and the animal "dances" backward and forward. Some dogs may resist dancing forward if the handler stands in front of the dog; dogs may plant their rear limbs and stretch out as the handler moves until the forelimbs reach the ground. In this situation, the handler should stand behind the dog, placing the arms under the axillary region to support the dog, and walk forward. How far the dog is elevated off the ground depends on the amount of stress the animal is able to comfortably handle on the rear limbs. Dogs may be elevated as high as possible and also dance up and down inclines or hills to place additional stress on the rear limbs. Dancing activities may also be accomplished with the assistance of a physioroll or ball. The front limbs of the patient are placed on the roll or ball and the therapist slowly rolls the ball toward the patient, forcing the dog to move the hindlimbs backward (Figure 30-5). Most dogs will reposition their forelimbs, especially if they are standing on the ball, rather than resting their antebrachium on the ball. Once comfortable with walking backward in this fashion, the ball can be slowly rolled away from the patient, stimulating forward walking of the rear limbs. A harness to provide support for the patient is very helpful for this activity. Alternatively, a wagon or other flat surface on wheels can be used for this activity, particularly for forward dancing.

To perform the wheelbarrow exercise, the handler places the hands under the caudal abdomen and lifts the rear limbs of the dog off the ground, and the dog is moved forward (Figure 30-6). The handler may wish to place a muzzle on the dog. Dogs with normal proprioception will move the forelimbs so they do not fall. Some dogs may require sling support if they are weak. Dogs may be wheelbarrowed up and down inclines for greater muscle strengthening (Figure 30-7). Theoretically, the higher the animal is lifted, the greater the forces placed on the forelimbs. However, the increase in weight bearing may not be as great as anticipated because the stride length is much shorter when the animal wheelbarrows from a greater height. The shortened stride results in less force placed on the limbs while wheelbarrowing as compared with walking or trotting at a faster speed. A hindlimb sling, particularly one that lifts at the pubis rather than the abdomen, may be used to assist the dog while wheelbarrowing.



Figure 30-6 Wheelbarrowing encourages use of the forelimbs. The therapist supports the abdomen and lifts the rear limbs of the dog off the ground, and the dog is moved forward. The handler may wish to place a muzzle on the dog.



Figure 30-7 Dogs may be wheelbarrowed up and down inclines for greater muscle strengthening.

Sit-to-Stand Exercises

Sit-to-stand exercises help strengthen hip and stifle extensor muscles and improve active ROM. The act of sitting, then standing up, requires muscle strength of the gluteal, quadriceps, hamstring, and gastrocnemius muscle groups.

Joint Motion Considerations

Sit-to-stand exercise results in greater flexion of the tarsus and nearly twice as much flexion of the stifle and hip, whereas extension of these joints is less as compared with walking.[7,8]

Exercise Considerations

Some training may be necessary, and low-calorie treats may be offered as a training aid to provide motivation to perform the movement. It is important to perform these exercises correctly. Attention should be paid to sitting and standing straight, with no leaning to one side, and the joints of both rear limbs should be symmetrically flexed so that the dog sits squarely on its haunches (Figure 30-8). While on the leash, after a sufficient warm-up period of walking, the handler asks the dog to sit squarely for a few seconds and then asks the dog to stand, take a few steps forward, and then again sit. The sit-to-stands may be repeated a number of times before the dog is allowed to rest. It may be easier in some cases to back the dog into a corner, with the affected limb next to a wall so that the dog cannot slide the limb out while rising or sitting. Start with 5 to 10 repetitions once or twice daily, and work up to 15 repetitions three to four times daily.

This exercise may be particularly beneficial for dogs with osteoarthritis of the hips. These patients generally feel pain when the hip joints are extended. Going from a sitting position to a standing position results in movement of the hip joint from a greatly flexed position to a hip angle similar to normal standing. In addition, there may be atrophy of the gluteal muscles. The sit-to-stand exercise allows active contraction of the gluteal muscles, but the hip joint is not generally extended to the point that results in pain. Therefore strengthening of the gluteal muscles can occur without the excessive pain encountered with hip extension.

Of course, each patient must be assessed to be certain that sit-to-stands are not too stressful or painful for that individual. If normal sit-to-stands are too painful, or the patient is too weak to rise from a sitting position, several modifications may be tried. Doing sit-to-stands with the pelvis in an elevated position may be helpful in patients that are too weak to rise from a complete sitting position. Dogs may sit with their pelvis on a stool, step, stack of books, curb, firm cushion, or the therapist's leg or knee, and rise from this elevated position. Many dogs will need guidance from the therapist's hands to learn to sit on an object because it may feel strange for some patients. Alternatively, dogs may be assisted with a sling from a sitting to a standing position. Dogs may also do sit-to-stands on an incline, with the body facing downhill to make it easier to stand. Alternatively, dogs may do sit-to-stands on an incline with the body facing uphill or sideways to each direction to provide an additional challenge and greater strengthening. For this challenge, dogs may perform five repetitions in each direction, twice daily. Finally, dogs may find it easier to do sit-to-stands in the water, using the buoyancy of the water to help assist them to a standing position.

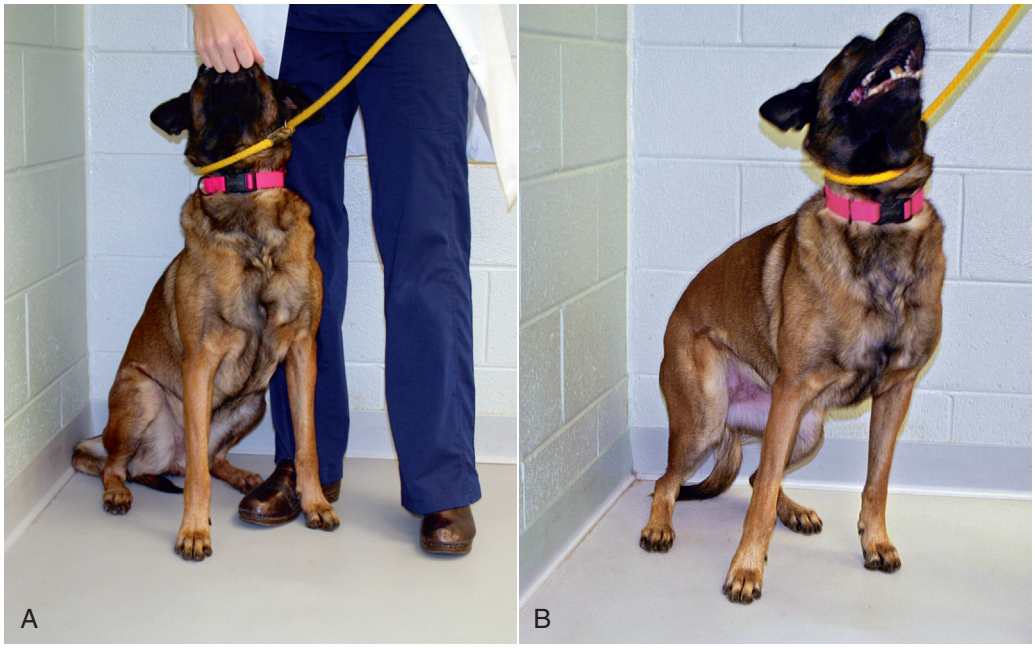


Figure 30-8 Sit-to-stand exercises. **A,** Note the symmetrical position of the dog in the sitting position. Placing the affected limb toward a wall will help the dog to sit in a square, symmetrical position. **B,** As the dog rises, it should push off equally with both rear limbs. Attention should be paid to sitting and standing straight, with no leaning to one side, and the joints of both rear limbs should be symmetrically flexed so that the dog sits squarely on its haunches.

Down-to-Stand Exercises

A variant of the sit-to-stand exercise is the down-to-stand. With this exercise, the dog is allowed to rise from a ventrally recumbent position to a standing position. In addition to strengthening the muscles of the pelvic limb, muscles of the forelimb are also engaged.

Joint Motion Considerations

In particular, the shoulder and elbow extensor muscles are activated through a greater ROM. The amount of motion has not yet been quantified, but joint motion of the hindlimbs should be similar to sit-to-stand exercises.

Exercise Considerations

It is important to have the dog rise symmetrically and push off equally with all four limbs. This exercise may require some additional time and training to perform correctly. Dogs will most commonly rise to a sitting position first and then stand, which is not the goal of this exercise. Similar to the sit-to-stand exercises, some training may be necessary with low-calorie treats to provide motivation to perform the movement. Attention should be paid to try to get the animal to use all four limbs to push up squarely and symmetrically. Start with 5 repetitions once or twice daily, and work up to 10-15 repetitions three to four times daily.

Cavaletti Rails

Cavaletti rails are poles that are spaced apart on the ground at a low height. Cavaletti rails may be used to encourage greater active ROM of certain joints, and lengthened strides in all limbs. They may also be used to challenge proprioception, balance, and coordination in animals returning to function following neurologic impairment.

Joint Motion Considerations

Walking over cavaletti rails results in significant increases in flexion and total ROM of the elbow, stifle, and hock as compared with walking, and the increase is related to the height of the cavaletti rails.[9] There is also a mild increase in hip flexion. There are no differences in joint extension, however.[9]

Exercise Considerations

Commercial cavaletti rails are generally used, although they can be constructed relatively easily. An alternative to cavaletti rails is to use a ladder and allow the rungs of the ladder to act as the low rails. Although ladders are readily available in most households, they have limited flexibility to change the distance between the rungs and the height that the animal steps over the rungs. This exercise can be beneficial for either orthopedic or neurologic patients in need of greater active flexion and ROM of targeted joints, improved voluntary motor control, and accuracy in placement of the limbs.

One or more poles may be used and should be spaced at appropriate distances apart, determined by the dog's natural stride length (Figure 30-9). The distance between the rails should allow for a single step; the exercise may not be as beneficial if dogs are allowed to take two or three



Figure 30-9 Cavaletti rails help increase active flexion of certain joints as the dog negotiates the rails. One or more poles may be used and should be spaced at appropriate distances apart, determined by the dog's natural stride length. Stride length may also be altered by placing the rails closer together or farther apart.



Figure 30-10 To provide additional flexibility to the trunk and proprioceptive awareness, dogs may do serpentine walking in combination with cavaletti rail walking.

steps between rails. In general, the height of the poles should be at the same level or higher than the carpus initially, but severely affected dogs may need very low poles to start. For very small dogs, cavaletti poles, broom handles, or PVC pipe can be taped to the floor at an appropriate width for the patient. After the animal becomes accustomed to the task, the handler can further challenge the dog by making simple modifications such as adding more poles, increasing the height of all the poles to encourage greater active flexion and ROM of joints, and altering the heights of alternating poles to encourage dogs to negotiate different situations. Begin with walking 3-5 times over the rails, and progress to 8-10 times over the rails. To provide additional flexibility to the trunk and proprioceptive awareness, dogs may do serpentine walking in combination with cavaletti rail walking (Figure 30-10). Dogs may also progress to trotting for greater rate of joint motion and proprioceptive challenges, or the cavalettis can be positioned over an unstable surface such as dense foam.

Walking in Tall Grass, Sand, or Snow

Walking a dog through a field of tall grass enhances muscle strengthening and endurance because of the resistance provided by the grass, as well as coordination to navigate varying terrain. In addition, dogs have a tendency to flex their joints to a greater extent as they negotiate the grass, resulting in greater overall ROM.

Exercise Considerations

Some caution should be used in dogs with allergies walking in grass, which may be aggravated in such conditions, and in dogs with conjunctivitis, which might also be exacerbated. Walking in deep sand or snow provides resistance to limb movement during walking and jogging and strengthens the flexor muscles, which must contract more to lift the limbs. However, one must be cautious with walking in deep snow that is dense or crusty as dogs may have difficulty pulling their limbs from the heavy, wet snow. Also, some dogs with metal implants (for example, a TPLO plate) may experience discomfort or postexercise lameness after walking in snow because the metal may contract, resulting in discomfort. Similarly, walking and jogging in tall grass strengthens the extensor muscles to some degree, which must contract to a greater extent to advance the limb. Exercising in sand and snow minimizes concussive forces placed on arthritic joints, while allowing strengthening of supporting periarticular muscles. Caution should be used when starting these exercises to avoid muscle soreness. Short, frequent sessions are recommended in the beginning, with a gradual increase in the time and repetitions. Standing in sand may also be beneficial to dogs in rehabilitation following neurologic conditions.

Tunnels and Limbo

Agility training tunnels or children's play tunnels may be used to promote flexion in the forelimbs and rear limbs, while strengthening the supporting muscles that hold the joints in this more flexed position. The size of the tunnel opening should be appropriate for the size of the dog, that is, just shorter than the dog's standing height. The dog is encouraged to crouch low and walk through the tunnel, which requires greater limb flexion and strength than that



Figure 30-11 A telescoping pole, such as a shower rail, may be adjusted in a doorway, with the height lower than the dorsal midline. The dog then walks under the pole to perform the limbo exercise.



Figure 30-12 For vertical pole weaving, the distance between poles should be adjusted so that sufficient side bending results. The distance between poles should be slightly less than the body length of the dog. In addition, the handler must lead the animal so that the head, neck, and body actually flex as the poles are negotiated.

needed for normal walking. Greater challenges may be instituted by using tunnels that are even smaller as dogs strengthen their muscles.

Alternatively, a telescoping pole, such as a shower rail, may be adjusted in a doorway, with the height lower than the dorsal midline. The dog then walks under the pole to perform the limbo exercise (Figure 30-11). The pole may be lowered to challenge the dog to crouch to a greater degree as strength and ability allow.

Under and Over Exercises

A combination of walking over cavaletti rails and alternating limbo walking is termed *under and over exercise.* This is a more advanced exercise that challenges strength, joint flexion, and proprioception.

Circling, Figure-Eight Walking, Vertical Weave Poles

Circling aids in lateral flexion of the spine, strengthening of the adductor and abductor muscles, balance, and proprioception. Walking in figure eights is a progression of circling. It adds additional challenges by changing directions repeatedly and helps to prepare the patient for irregular turning as the patient returns to more normal function. Weaving between vertical poles also helps to promote side bending of the dog's trunk and also challenges proprioceptive functioning and strengthening of limb abductor and adductor muscles, but with more sudden changes in direction.

Exercise Considerations

The size of the circles for circling should be large to begin with, and as limb use and proprioception improve, the size of the circles may become smaller. The same principles apply for figure-eight walking.

For vertical pole weaving, the distance between poles should be adjusted so that sufficient side bending results; in general, the distance between poles should be slightly less than the body length of the dog. In addition, the handler must lead the animal so that the head, neck, and body actually flex as the poles are negotiated (Figure 30-12). Alternatively, a very advanced version is to have the patient weave between the handler's legs, usually following a treat or toy (such as a Frisbee). The handler can then walk forward and backward while the patient continues to weave through the handler's legs. The patient should be released from exercise restrictions prior to performing this advanced exercise.

Aquatic Exercises

The reader is referred to Chapter 31 on aquatic therapy for specific information regarding aquatic exercise, especially underwater treadmill walking and trotting and swimming. In general, joint flexion is increased over typical land-based exercises, such as walking or trotting. With swimming, joint extension is less as compared with walking or trotting. Underwater treadmill walking has the advantage of increased joint flexion, with joint extension that is similar to land-based exercises. In addition, the amount of flexion of specific joints may be affected, to some degree, by the level of the water.[10]

Strengthening and Endurance Exercises

Many of the exercises used for strengthening and endurance are also used for joint motion exercises. The reader

is referred to additional information regarding specific exercises in the section regarding joint motion exercises. Strengthening exercises are used to improve the strength and speed of muscle contraction, whereas endurance exercises are performed to help with cardiovascular and musculoskeletal stamina. Although both types of conditioning may be performed, usually one form of conditioning is primarily undertaken, depending on the intended use of the dog. For example, a field trial dog will likely benefit more from endurance exercises, although some form of strengthening is also beneficial. Similarly, a racing greyhound may benefit more from strengthening exercises but endurance conditioning is also beneficial.

Strength exercises are those performed with maximal or near-maximal muscle contraction, with relatively few repetitions. The object of strength exercises is to increase the strength of muscle contraction. Myofiber and muscle hypertrophy occur with continued conditioning. However, there is little to no change in the aerobic capacity of the muscle.

Endurance exercises are those performed with repetitive contractions over a prolonged time, with relatively little load applied to the muscle. Although myofiber and muscle hypertrophy do not occur to any appreciable extent, the aerobic capacity and efficiency of muscle contraction are greatly enhanced.

Core Strengthening Exercises

The importance of strong core body muscles, including the back, abdominal, and upper limb muscles, has been recently emphasized in human rehabilitation and training. Strong core muscles make it easier to perform many physical activities and help provide support to the midsection when performing other exercises. Although these types of exercises are a bit more challenging in dogs because of the difficulty in consciously performing exercises that isolate particular muscle groups, there are several exercises that are useful and require relatively little training.

Caution should be used in performing any of these exercises in dogs with back, hip, or shoulder problems. In general, core-strength exercises may be repeated five times. As core strength improves, they can be increased to 10 to 15 repetitions, two to three times daily. A combination of exercises allows strengthening of different muscle groups, while preventing boredom.

Sit-to-Stands, Down-to-Stands on Ball, Physioroll or Trampoline

This variation of the sit-to-stand and down-to-stand is performed on an exercise ball, physio-roll, or trampoline. The additional challenge achieved by performing these activities on an unstable surface engages the core muscles, resulting in strengthening. These exercises are good to perform early in the course of a core strengthening program.

Standing on a Rolling Cart

The patient may be placed on a rolling cart, preferably with sides, and slowly pulled. Although this exercise also challenges proprioception, the core muscles are also engaged while trying to maintain balance. As the patient becomes stronger and corrects its position while being pulled in a straight line, turns and irregular movements may be incorporated during the therapy session for additional challenges and strengthening. The therapist should always provide support to prevent falls in the event that the patient loses its balance.

Rolling

Animals that roll from their side while in lateral recumbency or from their back in dorsal recumbency to a sternal position engage their abdominal muscles, in particular, their abdominal oblique muscles. If the dog can be trained to roll completely, that engages even more muscles and requires more strength. A low-calorie treat is helpful as a training aid.

Sit-Ups and Crunches and Hold, Twisting Crunches

Dogs may perform abdominal crunches or sit-ups with proper positioning and training. To perform this exercise, the handler sits on the floor with the legs spread far enough apart to support the dog in dorsal recumbency. A treat is then used to encourage the dog to lift its head and thorax up while maintaining the pelvis and lower limbs in contact with the ground and within the trough formed by the handler's legs. If possible, the treat may be held just out of the dog's reach to encourage the dog to hold the crunch for several seconds. In the beginning, it may be beneficial to have the dog receive a treat reward on every crunch. But as the training progresses, intermittent administration of a treat is likely adequate to keep the dog motivated. Peanut butter or Kong paste frozen into a Kong toy or coffee mug may work better than individual treats for some patients during this exercise because it is easier to get the patient to hold the desired position.

Another version of the abdominal crunch is the twisting crunch. To perform this exercise, the treat is held somewhat to the side, encouraging the dog to sit up in a diagonal fashion to reach the treat. This is one of the most effective crunch workouts, because it engages all of the abdominal muscle fibers at once.

Super Pointing

This exercise is relatively easy to perform and most dogs tolerate it well; however, two handlers may be necessary. The dog is placed in a square standing position and one handler maintains control of the dog's head and lifts one of the forelimbs. Another handler simultaneously lifts the diagonal rear limb. This position is held for several seconds. In patients with hindlimb injuries, it is often easier to lift the contralateral hindlimb first followed by the ipsilateral

forelimb. If possible, lifting of the legs may be done with an elastic resistance band so that the dog not only engages the core muscles, but also contracts the limb muscles to help maintain balance. Although having the handler lift both legs and hold them without bands also engages the core muscles, additional challenges and strengthening are achieved by using resistance bands. Another variation is to train the patient to shake or lift a forelimb while standing squarely without shifting weight significantly onto the hindlimbs or the contralateral side.

An easier version of this exercise is simply one-legged pointing. If only one handler is available, or if the dog is not strong enough or has proprioceptive deficits, one leg may be lifted and held at a time. The forelimbs and rear limbs should be raised one at a time to engage the different core muscles.

Hup and Praying Positions

Core strengthening may be achieved by having the dog stand with the forelimbs supported while the rear limbs remain on the ground, a maneuver sometimes called *hup* or a *standing plank position.* The handler may support the forelimbs of the dog or the limbs may be supported by a piece of furniture or stairs. This exercise is one of the earliest to help strengthen the core muscles. A reverse standing plank may be performed by placing the rear limbs in an elevated position while allowing the forelimbs to remain on the ground in a weight-bearing position.

Conversely, another maneuver is a praying stance. Some dogs do this naturally, whereas others will need some training to perform it correctly. With this position, the hindlimbs maintain a standing position while the forelimbs remain on the ground. It is important that the elbows be in contact with the ground to perform the exercise correctly. Clicker training is especially helpful to "capture" this position during training.

Plank on a Ball

A plank that provides additional challenges is one performed on a therapy ball or roll. The plank is performed by having the dog stand with its rear limbs on the floor and the forelimbs supported by a therapy ball or roll. This plank position may be held for several seconds. Additional challenges may be instituted by rolling the ball back and forth (ball roll-ins and roll-outs), side to side, or 360 degrees, doing ball circles. Initially, the excursions are kept small, but as strengthening and compliance improve, larger excursions may be instituted. For dogs requiring more support and less challenge, a therapy roll may be used that moves in only two dimensions. Alternatively, the rear limbs may be placed on the ball or roll, with the forelimbs on the ground. Again, additional challenges may be instituted by rolling the ball back and forth (ball roll-ins and roll-outs).

Another variation is for the dog to stand with its forelimbs on a donut, and use that as a pivot point around which the rear limbs move laterally. In addition to normal core strengthening, this helps to strengthen the abdominal oblique muscles as well as the thigh abductor and adductor muscles. For symmetrical strengthening, the dog should move equal distances in both directions. A balance dome (half ball–half flat surface) or a BOSU ball may also be used.

For more challenge, a modified plank on a ball may be performed. With the dog positioned as previously described, a forelimb or rear limb is raised off the floor and held for 3 to 5 seconds. The exercise is repeated holding up the other limbs. Super pointing may also be performed with the forelimbs on the ball by lifting diagonal limbs at the same time.

Back Extension

The muscles responsible for back extension may be strengthened by going from a sitting to a begging position or jumping up on the rear limbs and holding this position while pursuing a treat. As strength and coordination improve, the dog should maintain the two-legged standing position for several seconds; some dogs may be training to "walk" on two legs. Plank exercises also help to strengthen back extensor muscles.

Super Standing

Dogs may stand with all four limbs on a therapy roll. Although maintaining balance in a static position is highly challenging to the core muscles, moving the roll from side



Figure 30-13 Dogs may stand with all four limbs on a therapy roll. Although maintaining balance in a static position is highly challenging to the core muscles, moving the roll from side to side and rocking front to back provides additional strengthening.



Figure 30-14 Standing on a balance dome (half ball, half flat surface) or a BOSU ball may be used for core strengthening exercises.

to side and rocking front to back provides additional strengthening (Figure 30-13). One of the most challenging core exercises is for the dog to place both forelimbs on a therapy ball or roll, and the rear limbs on a separate ball or roll. Initially, larger balls with more stability are easier for the dog. As core strengthening improves, smaller balls or rolls may be used. Alternatively, the dog may stand on inflated discs with the forelimbs on one disc and the rear limbs on a separate disk. For additional challenges, the balls, rolls, or disks may be rocked back and forth, and in the case of rolls and balls, they may be moved closer or farther apart from each other to engage different core muscles.

The use of paw pods is less challenging for those dogs beginning a training program. A balance dome (half ball, half flat surface) or a BOSU ball may also be used (Figure 30-14).

Incline Walking, Trotting, and Running

Walking or jogging the dog up or down inclines aids in strengthening of the quadriceps, semitendinosus, semimembranosus, gastrocnemius, and gluteal muscles while ascending, and the triceps and shoulder stabilizing muscles while descending a ramp. Muscle strength in the hips and stifles is required for the dog to propel itself up an incline, whereas muscle strength in the shoulders and elbows is required to brake while going down a declined surface. Incline walking may be used as a low-impact activity.

Exercise Considerations

Inclines should begin with gradual slopes and increase to progressively steeper slopes if possible. As the dog's strength and stamina improve, the dog may be challenged by walking up longer, steeper slopes and by increasing the duration and speed of the climbing exercise. Further challenges are provided by doing inclines while wearing a weighted vest or leg weights (Figure 30-15). Jogging and running up slopes promotes strength and power.



Figure 30-15 Additional strengthening may be provided by walking up an inclined surface while wearing a weighted vest or leg weights.

Dancing and Wheelbarrowing

Dancing and wheelbarrowing enhances strength of the extensor muscles of the rear limbs and forelimbs, respectively. The amount of peak force placed on the limbs while dancing is slightly greater than what would occur at a trot, whereas wheelbarrowing results in peak force placed on the limb that is between walking and trotting.[6] Therefore if the stage of recovery is not adequate to withstand these stresses, the exercises should not be undertaken until adequate healing has occurred.

Exercise Considerations

Consider using a muzzle on dogs when first starting these exercises. To perform dancing, the forelimbs are lifted off the ground, allowing the patient to bear weight only on the hindlimbs. The handler then moves the dog backward and forward. Dogs may be elevated as high as possible and also dance up and down inclines or hills to place additional stress on the hindlimbs. Furthermore, leg weights may be placed on the limbs for additional strengthening.

To perform the wheelbarrow exercise, the handler places the hands under the caudal abdomen and lifts the rear limbs of the dog off the ground, and the dog is moved forward. Dogs may be wheelbarrowed up and down inclines for greater muscle strengthening. Theoretically, the higher the animal is lifted, the greater the forces placed on the forelimbs. However, the increase in weight bearing may not be as great as anticipated because the stride length is much shorter when the animal is wheelbarrowed from a

greater height. Similar to dancing, leg weights may be placed on the limbs for additional strengthening.

Sit-to-Stand Exercises

Sit-to-stand exercises help strengthen hip and stifle extensor muscles. The act of sitting, then standing up, requires activation and strength of the gluteal, quadriceps, hamstring, and gastrocnemius muscle groups. The back and abdominal muscles are also affected to some extent.

Exercise Considerations

Some training may be necessary, and low-calorie treats may be offered as a training aid to provide motivation to perform the movement. It is important to perform these exercises correctly. Attention should be paid to sitting and standing straight, with no leaning to one side, and the joints of both rear limbs should be symmetrically flexed so that the dog sits squarely on its haunches. The handler then asks the dog to stand, take a few steps forward, and then again sit. The sit-to-stands may be repeated a number of times before the dog is allowed to rest. It may be easier in some cases to back the dog into a corner, with the affected limb next to a wall so that the dog cannot slide the limb out while rising or sitting. Start with 5 to 10 repetitions once or twice daily, and work up to 15 repetitions three to four times daily.

Gluteal muscle atrophy may occur in dogs with hip arthritis. The sit-to-stand exercise allows active contraction of the gluteal muscles, but the hip joint is not generally extended to a point that causes pain because the hip only extends to a standing position. Therefore strengthening of the gluteal muscles can occur without the excessive pain encountered with hip extension. Each patient should be assessed to be certain that sit-to-stands are not too stressful or painful for that individual. If normal sit-to-stands are too painful, or the patient is too weak to rise from a sitting position, several modifications may be tried. Doing sit-to-stands with the pelvis in an elevated position may be helpful in patients that are too weak to rise from a complete sitting position. Dogs may sit with their pelvis on a stool, step, stack of books, curb, firm cushion, or the therapist's leg or knee, and rise from this elevated position (Figure 30-16). Many dogs will need guidance from the therapist's hands to learn to sit onto an object because it may feel strange for some patients. Alternatively, dogs may be assisted with a sling from a sitting to a standing position. Dogs may also do sit-to-stands on an incline, with the body facing downhill to make it easier to stand. Alternatively, dogs may do sit-to-stands on an incline with the body facing uphill or sideways to each direction to provide an additional challenge and greater strengthening. For this challenge, dogs may perform five repetitions in each direction twice daily. Further challenges may be provided by applying a weighted vest or backpack to encourage additional muscle strengthening. Super sits can be done using a donut or physioroll by asking the patient to sit and stand while keeping the forelimbs on the device.



Figure 30-16 Dogs may sit with their pelvis on a step and rise from this elevated position to modify the sit to stand exercise.

Rhythmic Stabilization

Rhythmic stabilization is a technique used to strengthen postural muscles in dogs with significant weakness of these muscles. In particular, this exercise is valuable for strengthening the triceps and quadriceps muscle groups. It also helps to provide core strengthening and neuromuscular feedback to joints, ligaments, and muscle-tendon units.

Exercise Considerations

Rhythmic stabilization may be performed with the patient on a therapy ball or roll, trampoline, or inflated mattress. With the animal standing in a square position, the therapist places pressure over the pelvis or interscapular region and gently "bounces" the animal up and down. With downward pressure, the animal will begin to sink, and then reflexively will contract the extensor muscles to recover to a normal standing position. The bouncing motion should be relatively rapid, with only enough recovery time to regain the normal standing position. Performing the exercises with the animal standing on a compressible surface generally prevents a well-trained animal from going to a sit position.

Hand Shaking

In some situations, the flexors of the elbow joint may be weak following conditions such as partial brachial plexus injury or intervertebral disk herniation. Having the dog do shake hands exercises with the affected limb helps with functional strengthening.

Exercise Considerations

Most dogs have been trained to shake hands with their owners, so this is a relatively easy exercise. It is most



Figure 30-17 Greater strengthening may be achieved by placement of a leg weight while doing shake hands exercises.

beneficial to begin with the affected limb on the ground with the dog in a sitting position. The therapist asks for the dog's paw, trying to achieve as high a placement as possible to achieve the greatest elbow flexion possible. Shoulder extensor muscle strengthening may also occur with this exercise. Initially, three to five repetitions may be adequate. Increased repetitions are added as the dog is able. Greater strengthening may be achieved by placement of a leg weight or resistance band on the antebrachium (Figure 30-17). To reduce the moment arm, the weight may be placed proximal on the radius and ulna. As the dog gains strength, the weight may be moved further distally or more weight may be added.

Stairs

Climbing stairs is useful to improve power in the rear-limb extensors, whereas descending stairs helps strengthen the forelimb extensors. In many instances, negotiating stairs is an activity of daily living that may be performed in the home environment. Quadriceps and gluteal muscle groups are strengthened as the animal pushes off, extending both hips and knees while propelling the body up the steps. Conversely, the biceps brachii, triceps, and extensors of the carpus are strengthened by descending stairs.

Exercise Considerations

The dog must begin slowly climbing stairs to encourage proper use of the rear limbs, as opposed to simply carrying the limb, hopping with both rear limbs, or skipping up stairs. Encourage the dog to go slowly and deliberately, climbing the stairs in a reciprocal stepping gait. Stairs should be introduced slowly because this is a challenging exercise for both the musculoskeletal and cardiovascular systems and the animal may fatigue quickly. Initially, some dogs may require assistance from the handler. Begin with 1 to 3 flights of stairs for early strengthening, and gradually increase to 5 to 10 flights of stairs once or twice daily. As the patient improves, dogs may jog and trot up stairs, and eventually may run up stairs.

Cavaletti Rails

Cavaletti rails may be used to encourage muscle strengthening and coordination through a greater active ROM of certain joints, and lengthen strides in all limbs. Similar to the shake hands exercise, they may be used to help increase strength of forelimb flexor muscles in patients with weak joint flexion. Unlike the shake hands exercise, however, walking over cavaletti rails may be used to strengthen weak flexor muscles of the rear limbs.

Exercise Considerations

Poles should be spaced at appropriate distances apart, determined by the dog's natural stride length. The distance between the rails should allow for a single step; the exercise may not be as beneficial if dogs are allowed to take two or three steps between rails. For muscle weakness, the rails are initially placed low to the ground. After the animal becomes accustomed to the task, the handler can further challenge the dog by adding more poles and increasing the height of all the poles to encourage greater active flexion and ROM of joints. Begin with walking 3-5 times over the rails, and progress to 8-10 times over the rails. Dogs may also progress to trotting for an additional rate of joint motion challenge. Leg weights may be added for additional strength training. Finally, for an extreme challenge, a gymnastic cavaletti ("bounces") can be created so that the dog must jump over each rail, land, briefly pause, and jump again without taking a stride between rails, creating a plyometric exercise.

Treadmill Walking and Jogging

Treadmills are excellent tools for helping to improve endurance, and they may be used to help with limb strengthening during gait. Not only can the walking or jogging time, length and speed increase, but the treadmill may be angled up or down to reduce or increase the forces placed on the forelimbs or rear limbs. Dogs that are trained to walk on leash generally are easily trained to walk on a treadmill. The flat, regular surface of the treadmill belt also allows exercise on a consistent surface with lower risk of a misstep. Patients should always be directly supervised, however, especially when the patient is becoming fatigued or the speed is at a fast walk or greater. In addition to monitoring for obvious fatigue, such as pulling on the harness or stumbling, it is possible to obtain respiratory rates and to record heart rates with an electrocardiogram machine or a telemetry unit to gain some insight regarding conditioning fitness over time. Care should be taken to not exercise beyond mild fatigue because the risk of injury may increase substantially.



Figure 30-18 To add resistance to limbs during treadmill activity to aid with muscle strengthening, an elastic band may be used to provide resistance to specific muscle groups.

Exercise Considerations

A warm-up period of walking and then fast walking is recommended prior to trotting on a treadmill. This also allows a period of adaptation to gaiting on the treadmill. It is important that the treadmill speed be rapidly increased when changing gait from a walk to a trot to avoid an awkward transition. In general, most medium to large dogs walk comfortably at a rate of 0.9-1.2 m/s (2-2.7 mph). A medium trot is generally 1.7-2 m/s (3.8-4.5 mph). To add resistance to limbs during treadmill activity to aid with muscle strengthening, an elastic band may be used to provide resistance to specific muscle groups (Figure 30-18). The band is secured to the involved extremity and the handler provides tension on the other end. For example, the band may be tied around the forearm with tension applied from the rear, providing resistance and strengthening of the elbow flexors and shoulder extensors during the swing phase of gait. Flexor muscle strengthening may be achieved by putting leg weights on the desired limbs.

Jogging

Jogging not only helps to improve cardiovascular conditioning, but it also helps with muscle strengthening. Jogging should be considered when increased speed is desired during the intermediate stage of recovery from injury or surgery, as a transition from walking to running. Early speed training aids muscle strength and the velocity of muscle contraction, important factors in improving speed. Consideration should also be given to the increased force placed on an affected limb when transitioning from walking to jogging. For example, walking places approximately 60% and 30% of body weight on a forelimb and hindlimb, respectively, at a walk.[11] Progressing to a medium-paced trot increases weight bearing to approximately 110% and 70% of body weight on a forelimb and hindlimb, respectively.[12]

The patient should not be exercised beyond mild fatigue. Exercising when fatigued leads to muscle weakness, poor control of accessory joint motion, and delayed proprioception responses, all resulting in possible joint injury.

Exercise Considerations

Jogging may be initiated in cases with stable surgical repairs when the dog is walking on the limb with minimal lameness and pain, and the stage of tissue recovery can withstand jogging. Begin slowly, jogging 0.5 to 3 minutes one to three times daily, and work up to 20 minutes two to three times daily. Be certain that lameness is not worse after jogging. As the patient develops adequate conditioning and strength to easily tolerate this level of activity, interval training may begin, which alters short bursts of speed activity with slower jogging. It is believed that this form of training may result in accelerated gains in speed and conditioning. An alternative if the therapist or owner cannot jog with the patient is to use an extendable leash to allow the dog to trot or jog out, and then return to the handler immediately, repeating this cycle several times for a total duration of 5-20 minutes.

Walking and Jogging in Tall Grass, Sand, or Snow

Walking or jogging a dog through a field of tall grass, sand, or snow enhances muscle strengthening, endurance, and cardiovascular conditioning because of the resistance provided by these media. In addition, dogs have a tendency to flex their joints to a greater extent as they negotiate these substances, resulting in additional cardiovascular conditioning and strengthening through a greater range of joint motion.

Exercise Considerations

Walking or jogging in deep sand or snow provides resistance to limb movement and strengthens the flexor muscles, which must contract more to lift the limbs. Extensor muscles may be preferentially recruited when walking or jogging in tall grass because they must contract to a greater degree to advance the limb. Exercising in sand and snow also minimizes concussive forces placed on arthritic joints, while allowing strengthening of supporting periarticular muscles. Further strengthening may be achieved by using leg weights, weighted backpack or elastic resistance bands.

Leg Weights and Resistance Bands

To add resistance to limbs during exercise and increase muscle strengthening, leg weights or elastic bands may be used to target specific muscle groups (Figure 30-19). Leg weights are frequently used in dogs with weakness as a result of injury to musculoskeletal tissues and subsequent surgery, or neurologic conditions. Leg weights may be fashioned from lead strips, or commercially available leg weights for people may be used. It is important that the

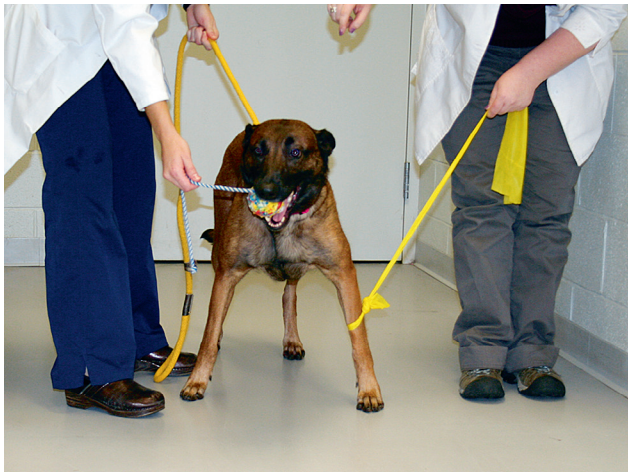


Figure 30-19 To add resistance to limbs during exercise and increase muscle strengthening, leg weights or elastic bands may be used to target specific muscle groups, such as the medial glenohumeral structures.

method of securing the weight is appropriate for dogs. Many leg weights designed for people may not fit properly. Many of these are filled with sand, and when the weight is applied to the thinner dog limb, the fastening straps may not provide a snug fit, allowing it to slip off. Removal of some of the sand may solve this problem. The leg weight must be firmly attached to the dog's limb because most dogs will attempt to shake the weight off when first introduced to it. In addition to considerations regarding securing the leg weight, placement on the limb should also be considered. The moment arm, and therefore the stress on the limb, is less when the weight is placed closer to the affected limb segment. If additional muscle force to move the limb is desired, the weight should be placed distal to the affected segment so that a larger muscle force is generated to move the limb and weight. A general rule of thumb is to use a weight that is between 1% and 3% of body weight, depending on limb strength and the stage of recovery. Therefore ½-lb leg weights may be used on dogs that weigh 10 to 20 lb, 1-lb weights on dogs weighing 20 to 40 lb, 1½-lb weights for 40- to 60-lb dogs, and 2-lb weights for dogs weighing more than 60 lb. Caution should be used when first applying the weights because some dogs may shake the limb or have exaggerated limb motion because of the altered sensation. It is possible that injury may occur, so it is important to gradually introduce the weight to allow a period of accommodation. Leg weights are most effective in strengthening the flexor muscles because these muscles are engaged to lift the limb during the swing phase of gait to advance the limb. In many dogs that are not accustomed to the leg weight, the limb will be held up in a flexed, non–weight-bearing position, and the therapist may be discouraged. However, the flexor muscles will fatigue in most cases, and the limb will be gradually lowered to the ground. Strengthening of the flexor muscles has occurred in this situation, and eventually the dog places the limb on the ground, resulting in improved weight bearing while standing or walking. Therefore a positive outcome has occurred.

Elastic resistance bands are also frequently used for limb muscle strengthening. Bands come in various degrees of resistance, with the lighter colors generally having the least resistance and darker colors having greater resistance. The band is secured to the involved extremity and the handler provides stability on the opposite end. For example, the band may be tied around the forearm with the line of pull from the rear, providing resistance and strengthening of the elbow flexors and shoulder extensors during the swing phase of gait. Although leg weights require no real intervention on the part of the therapist other than securing the weight, the therapist is usually engaged while using resistance bands. The tendency is to try to "time" the movement of the limb with the therapist's hand motions. Frequently, this is mistimed and results in disruption of the gait cycle of the dog. In most cases, it is better to simply apply some light constant tension to the band and allow the dog to contract the desired muscle group against the band to move the limb. The tension is placed on the band in the direction opposite the muscle group for which strengthening is desired. For example, if strengthening of the quadriceps muscle is desired, caudal tension is placed on the band. Therefore when the limb is advanced and the quadriceps muscles contract to extend the stifle during the end of the swing phase of gait, the caudal tension on the band results in increased contraction strength of the quadriceps muscle. Unlike leg weights, which result in strengthening of all muscle groups, resistance bands may be used to target specific muscle groups. For example, in a dog with early medial shoulder instability, it may be desirable to strengthen the medial shoulder muscles, including the subscapularis. An elastic band may be used to increase the contraction strength of this muscle group. For example, while walking on a ground treadmill, gentle lateral tension is applied to the band, so that the adductor muscles must contract to a greater degree, resulting in strengthening of that group. Similarly, resistance bands may be used to apply tension while hopping the animal laterally, similar to hopping performed during neurologic evaluation. The degree of tension applied should not result in discomfort to the dog or significantly alter the dog's gait. In general, elastic resistance bands are used most frequently while walking over ground or on a ground treadmill, but they can also be used while swimming or walking on an underwater treadmill. In these situations, the band should be stretched out and laid flat to dry to avoid sticking together.

Pulling or Carrying Weight

A variety of harnesses are available for dogs to attach to carts or sleds for pulling weight. The harness should be



Figure 30-20 Pulling a sled with added weight helps with muscle strengthening.

well padded and comfortable. Pulling a cart with a large wheel diameter is easier than pulling a sled that slides along the ground. The positions of the head and neck are important in determining whether a dog pulls the weight forward with the forelimbs or drives forward from the rear limbs (Figure 30-20). If the dog carries its head and neck low to the ground, it is likely pulling with the forelimbs. A dog with the head and neck held high will shift some of the weight caudally and tend to use the rear limbs to drive the body forward. A variety of sleds and carts are commercially available. In all cases, the harness should be adjustable and properly fit the animal and cart or sled to avoid abnormal pressure where the harness contacts the animal; a poorly fitted rig may result in pressure sores and alter the manner in which the dog pulls the weight.

Dogs may also strengthen the core and postural muscles by wearing a canine backpack filled with weights. Weights may be loaded unequally or equally on both sides. When a dog wears a weighted backpack during down-to-stand or sit-to-stand exercises, greater muscle force is required to rise to a standing position. There does not seem to be a consistent pattern regarding placing weight equally on both sides of the dog or asymmetrically to enhance weight bearing on an affected limb. A trial-and-error approach should be used on an individual to determine the best placement of weight in the backpack to increase weight bearing on a particular limb. In general, these devices should be limited to dogs that are either healthy and using them for conditioning, or for dogs that are rehabilitating from a specific injury (e.g. CCL tear) and are using them for a specific purpose in rehabilitation, such as building up the hip extensor muscles. Dogs with conditions such as OA may be made worse by increasing the weight they are carrying, which increases forces on abnormal joint.

Aquatic Exercises

The reader is referred to Chapter 31 on aquatic therapy for specific information regarding aquatic exercise, especially underwater treadmill walking and trotting and swimming.



Figure 30-21 Additional strengthening of the limbs may be achieved by using flotation devices placed on a limb while walking on an underwater treadmill.

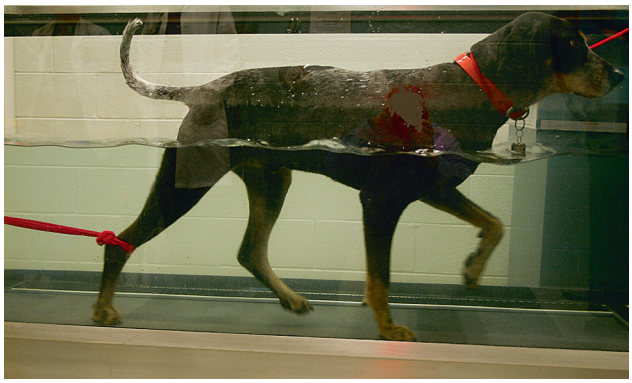


Figure 30-22 Resistance bands may be attached to a limb while walking on an underwater treadmill. The therapist pulls on the band in the direction opposite of the muscle group in which strengthening is desired.

Strengthening of muscle groups is achieved by moving the limbs against the resistance of the water. The effort to move the limbs increases by a factor of the velocity to the third power when the speed of swimming is increased; a similar phenomenon presumably occurs when the velocity of underwater treadmill walking increases. Further strengthening may be achieved by using flotation devices placed on the limbs (Figure 30-21). Similarly, resistance bands may be attached to the limbs; the therapist pulls on the band in the direction opposite of the muscle group in which strengthening is desired (Figure 30-22).

Speed Exercises

As the patient recovers and has very good to full weight bearing on the limbs, the goal should be to return the dog to as complete athletic function as possible. Although most dogs undergoing rehabilitation are not competitive

athletes, nearly all dogs enjoy running and playing. Therefore rehabilitation patients should be gradually conditioned to withstand the rigors of activity at moderate to high speeds. It is vital that this phase of training not occur before adequate tissue healing and muscle strength and endurance have occurred to prevent possible injury. Trotting, running, and running on a treadmill are all methods to increase speed. In addition, sport-specific activity should be gradually introduced to improve function. Interval training techniques may be especially effective in enhancing performance, including endurance training. With this form of training, near-maximal effort is used, followed by a less intense effort. The cycles are repeated during a single training session. The number and length of cycles are gradually increased to add conditioning stress to enhance performance.

Controlled Ball Playing

Ball playing is a fun and effective form of therapeutic exercise that dogs and their owners enjoy. It also has the potential to cause damage to surgical repairs. Controlled activity is the key. The degree of activity depends on the surgical procedure performed, the condition of the tissues, and the stage of tissue healing. Ball playing should begin on a relatively short leash to avoid explosive activity in the early postoperative period. As the patient progresses, the dog graduates to ball playing in an enclosed area, such as a run. As the animal nears full return to function, off-leash activity may be performed in a large fenced field free of irregular surfaces. The main benefits of ball playing are to increase power, speed, and muscle strength. In most conditions, jumping should be avoided to reduce the risk of injury. It is important to start by rolling the ball or throwing such that the ball is not bouncing high in the air, which encourages jumping.

Exercises for Cats

Physical rehabilitation of cats can be challenging. The principles of physiotherapy in cats are similar to those of dogs. However, the personality of some cats can alter the approach to physiotherapy. The most common reasons to perform physiotherapy in cats are generally related to injuries sustained as a result of trauma or joint conditions. Fractures of the long bones, especially the femur and tibia, as well as the pelvis are relatively common. Common joint conditions that undergo postoperative rehabilitation include cranial cruciate ligament rupture, deranged stifle, femoral head and neck excision, and patella luxation. On occasion, neurologic conditions occur in cats that could benefit from physiotherapy, including trauma and intervertebral disk herniation. Osteoarthritis is yet another condition for which physiotherapy may be beneficial.

Although most dogs improve and become more comfortable with rehabilitation as the sessions continue, most cats may actually become less comfortable and more suspicious of various rehabilitation activities. In addition, many cats are not motivated by food rewards as dogs are. Therefore a different approach to physiotherapy is often necessary.

Proprioceptive exercises may be performed similar to dogs, and include weight shifting activities, balance board, wobble board, and standing on a therapy ball or roll. A small foam roll or roll of paper towels underneath the cat to help provide support may make the proprioceptive activities easier. The therapist must be very observant and watch the cat for signs of agitation to prevent the cat from having an unpleasant experience during rehabilitation. Initial sessions may only last for 30 seconds on a balance board, for example. If the cat has a pleasant experience, the sessions may gradually extend to 1 or 2 minutes on the balance board.

Most cats are not trained to walk on leash, but if a particular individual is able to, especially with the harness, then leash walks are some of the earliest therapeutic exercises that may be performed. Ground treadmill walking with support and assistance is another way to improve limb use. With many therapeutic exercises, cats that are accustomed to going into their carrier may be lured into an open door. This technique can be effective for a number of exercises. As the cat improves with treadmill walking, strengthening can be achieved with small leg weights or elastic resistance bands to increase the strength of muscle contraction. Many cats will resist this type of activity and it is important to be certain that the cats do not injure themselves while resisting.

Many cats will also play with feathers or toys tied on the end of the string to encourage activity and motion. A catnip mouse works well as a lure to get the cat to move. Cats that are conditioned to come running when the can opener is running can be exercised by putting them in a different area of the house and then starting the can opener, resulting in the cat coming to the open can.

Active ROM of joints is also important after many surgical procedures. One such exercise is to have the cat walk over low cavaletti rails. Again, an open carrier at the end of the cavaletti rails may entice the cat to walk over the elevated rails. It is important to let the cat win every so often by actually letting them advance into the carrier. Some cats respond quite well to a flashlight beam or laser light moving along the ground. Although cats tend to move in a relatively rapid manner as opposed to slow consistent movements, it can be an effective way to entice movements as well as stretching along a wall or vertical surface. However, caution should be exercised so that cats do not develop an obsessive-compulsive disorder with light chasing.

Aquatic therapy is possible in many cats. Although many cats do not like water, a number of cats will tolerate either swimming or walking on an underwater treadmill.

The therapist generally will need to accompany the cat in the aquatic environment. A small life jacket is advised in cats that will undergo swimming.

Although physiotherapy in cats is more challenging than in dogs, it is still an important part of recovery to help return them to full function as quickly as possible. Careful attention to behavior differences in cats and choosing activities that the cat enjoys are critical to successfully provide rehabilitation to cats.

Factors Affecting Progression of Exercises

Many variables must be considered in the design of a therapeutic exercise program to enhance strength, endurance, and speed, including the type and severity of the condition; the stability of any surgical repairs; the number of involved limbs and joints; the size of the animal; the preexisting physical condition of the patient; the available facilities; the skill and experience of the therapist; and whether the exercises will be administered by a professional, the owner, or a combination of the owner and a professional. Unfortunately, it is impossible to have a single protocol for each specific condition. Some basic guidelines may be applied, however.

The goal of any therapeutic exercise program should be to restore the animal to as full and active use as possible. The goal may need to be periodically adjusted if the patient progresses to a greater or lesser degree than expected. As the animal nears return to function, strategies should be developed to encourage muscle mass and strength, endurance or power, depending on the intended function of the patient.

It is very important to perform the exercises correctly and to maintain a consistent level of activity daily or every other day. When considering a muscle strengthening program, strengthening activities should only be performed two to three times per week to avoid overtraining and muscle pain. Overdoing activity will likely be detrimental to the animal's recovery. Appropriate nutrition is also important when undergoing muscle strengthening, especially appropriate protein and amino acid balance in the diet.

If pain or lameness increases during or several hours after stepping up an activity, the level of activity should be reduced by 50% for 3 to 7 days. If the lameness and pain then resolve, the activity may be slowly increased back to the pre–step-up activity level over 3 to 5 days, with careful observation to be certain that the activity is not increased too aggressively or rapidly. The rate of subsequent step-up activity should be half of what it originally was to avoid a recurrent episode of exacerbated lameness and pain.

It is also important to vary the routine for the animal's and the owner's sake to avoid boredom and to allow incorporation of function-specific activities in the animal's home or work environment. During chronic management of the patient, weight control is extremely important. It is also important to continue to oversee the protocol and document improvement. Progress will occur at a slower rate during the chronic phase of recovery, but continued rehabilitation is just as important to achieve as full a return to activity as possible.

Several medications may be appropriate for use in the chronic rehabilitation period, including intermittent nonsteroidal antiinflammatory agents and chondroprotective agents. Caution should be used, however, to be certain that any analgesic medications do not mask pain and discomfort associated with the level of activity. They should facilitate rehabilitation and recovery rather than allow rehabilitation to progress faster than normal conditions and tissue healing should allow.

Summary

Therapeutic exercises to increase joint motion, strength endurance, and speed are undoubtedly the most important aspects of rehabilitation. They may (and should) be incorporated with other modalities to enhance recovery, but it is the exercises that help improve muscle strength, joint mobility, limb use, and endurance to maximize function. Although a variety of techniques have been described, the ingenuity of the rehabilitation team, including the owner, will allow the development of other exercises that are specific to a patient's recovery. The keys to a successful therapeutic exercise program are to have site- and condition-specific exercises whenever possible, to use a variety of exercises and techniques to keep the therapy team and patient from becoming bored, and to allow the animal to appropriately progress so that tissues are adequately challenged for strengthening, but not so rapidly as to result in complications and tissue damage.

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31 Aquatic Therapy

David Levine, Darryl L. Millis, Jeffrey Flocker, and Lauren MacGuire

Aquatic therapy has become a very valuable component of veterinary physical rehabilitation. There are specific principles of aquatic therapy that provide unique benefits to the patient that other forms of therapy do not provide. In addition aquatic therapy comes naturally to many patients, which is especially beneficial because exercises in animal patients must be in a form that they naturally perform or can be easily trained to do. Although dogs cannot follow verbal instructions, they generally perform aquatic therapy very well.

Principles of Aquatic Therapy

It is necessary to understand the basic principles and properties of water, including relative density, buoyancy, viscosity, resistance, hydrostatic pressure, and surface tension to appreciate the benefits of aquatic therapy. These are important components to consider when planning an aquatic rehabilitation program.

Relative Density

The relative density of an object is the ratio of the weight of the object to the weight of an equal volume of water.[1] Relative density depends on the composition of an object. Densities of various substances are defined by a pure number value called *specific gravity*. The specific gravity of pure water is 1.0.[2] The specific gravities of fat, lean muscle, and bone are 0.8, 1, and 1.5 to 2, respectively.[3] The relative density and specific gravity of an object determine how well an object will float. If the ratio of an object's specific gravity to that of water is greater than 1, the object will tend to sink, and if the ratio is less than 1, the object will tend to float.[4] The specific gravity of an object also determines how much of the object's volume will float under water.[4] For example, the specific gravity of ice is approximately 0.92; therefore 92% of ice will be submerged to displace enough water so that the upward force of buoyancy will equal the downward force of gravity, and 8% will float above the surface of water.[2,5]

A lean person's specific gravity may be as high as 1.10, whereas an obese person's specific gravity may be as low as 0.93.[4] An object whose specific gravity is equal to that of pure water (1) will float just below the surface of the water. If a person's specific gravity is greater than 1, that person will tend to sink. The greater the specific gravity, the faster the sinking velocity.[2] Thus a lean animal that is not moving will have a tendency to sink faster than an obese animal.

Buoyancy

A body immersed in water is subjected to two forces, gravity and buoyancy. *Buoyancy* is defined as the upward thrust of water acting on a body that creates an apparent decrease in the weight of a body while immersed.[2] Buoyancy stems from Archimedes' principle: When a body is fully or partially submerged in a fluid at rest, it experiences an upward thrust equal to the weight of the fluid displaced.[1,2,4,6] The amount of water displaced depends on the density of the body immersed relative to the density of water.[2] An immersed limb or body with a relative density less than that of water will be assisted to the surface by buoyant forces.[7] An object with a relative density less than 1 floats because the weight of the object is less than the weight of the water displaced.[5,8] Buoyancy acts directly through the center of buoyancy.[3] The center of buoyancy is the centroid of the displaced volume of water and depends on the distribution of the displaced volume of fluid relative to the body.[9] Problems arise when the center of gravity and the center of buoyancy are not in the same vertical line. This can occur with the use of improperly placed floating devices, which can disrupt the vertical line, causing an animal to tilt or flip over so as to reach a state of equilibrium.[3] The location of the center of buoyancy varies depending on the percent of body fat and distribution of adipose tissue. The more caudal the distribution of adipose tissue (which tends to occur more often in human females than males), the closer the center of buoyancy is located to the center of mass.

Buoyancy aids in the rehabilitation of weak muscles and painful joints. It allows the patient to exercise in an upright position and may decrease pain by minimizing the amount of weight bearing on joints. For example the percentage of weight borne by human females when immersed in water is approximately 5.9% to 8.7% at the seventh cervical vertebral level, 25% to 31% at the level of the xiphoid, and 40% to 51% at the level of the anterior superior iliac

spine.[10] Males bear 6.8% to 10%, 30% to 37%, and 50% to 56% of their body weight in the upright position at these same levels.[10] Other studies of humans have also found similar percentages of weight bearing at these same levels.[11,12] In a study performed on dogs the amount of body weight borne when immersed in water (as a percentage of body weight on dry ground) was approximately 91% when the water was at the level of the lateral malleolus of the tibia, 85% at the level of the lateral condyle of the femur, and 38% at the level of the greater trochanter of the femur (Figure 31-1).[13] This information may be particularly useful when treating patients with arthritis because joints may be unloaded as a result of the buoyant properties of water.

Hydrostatic Pressure

Pascal's law states that fluid pressure is exerted equally on all surfaces of an immersed body at rest at a given depth. This fluid pressure is directly proportional to both the depth and density of the fluid.[4] Therefore the deeper a body is immersed in water, the greater the pressure exerted. The atmospheric pressure at the surface of water is 1.00 kPa (15.7 psi), and fluid pressure increases by 0.029 kPa (0.43 psi) per foot of depth.[3] It can be estimated that the pressure exerted on a part of the body immersed 3 feet below the surface will experience a fluid pressure of 1.09 kPa (17 psi). Because hydrostatic pressure provides constant pressure to a body or limb immersed in water, it may provide an improved environment for working with swollen joints or edematous tissues.[7] Hydrostatic pressure opposes the tendency of blood and edema to pool in the lower portions of the body and can therefore aid in reducing swelling.[3] The benefits of edema reduction because of hydrostatic pressure are used by pregnant women who exercise in water to help minimize swelling in their lower extremities during exercise. The reduction in edema is probably caused by several factors, including a general increase in circulation with exercise, and the prevention of further pooling of fluids in the lower extremities by the pressure exerted on them by water.

Hydrostatic pressure may also decrease pain during exercise. It is theorized that hydrostatic pressure provides phasic stimuli to the skin afferents (sensory receptors) that cause a decrease in nociceptor hypersensitivity. This acts to decrease a person's or animal's pain perception, which may allow the patient to perform a variety of movements with less pain.[7,14]

Viscosity and Resistances

Viscosity is a measure of the frictional resistance caused by cohesive or attractive forces between the molecules of a liquid.[15] The viscosity, or resistance to fluid flow, is significantly greater in water than in air,[7] making it harder to move through water than to move through air. Water can therefore provide resistance that may strengthen canine muscles and improve cardiovascular fitness. Viscosity is important in aquatic therapy for several other reasons. Viscosity may increase sensory awareness and assist in stabilizing unstable joints.[7] It can also help prevent falling by increasing the time span for patients to react,[7] which may reduce patient anxiety. For example dogs with paraparesis, obesity, osteoarthritis, or severe deconditioning may be more willing to walk in water than on land because of water's combined properties of buoyancy and viscosity, which help to support the dog.

Any body movement occurring in water must overcome water viscosity.[16] The resistance created by the viscosity of the liquid is proportional to the velocity of movement through a liquid. With increased velocity of movement in water, there is a uniform increase in resistance.

Various movements in water may increase or help reduce resistance. Streamline flow is the steady, continuous movement of fluid. There is little friction between layers of fluid in streamline flow because the layers separate to move around the object and smoothly rejoin behind it.[5] Turbulent flow consists of irregular movements of the layers of fluid.[1,5] The irregular movement in turbulent flow causes increased friction between the molecules of the fluid and between the object and the fluid.[5] Eddies are a form of turbulent flow in which the layers of fluid following a moving object in water move in circular directions.[5] Eddies resist an object's movement in water by pulling the object backward. Because of all these factors, resistance in aquatic exercise may be increased by increasing the velocity of movement of the patient, increasing the surface area of the object or body part moving in water, or by increasing the length of the lever arm of the object moving in water.[16]

Surface Tension

The tendency for water molecules to adhere to each other is known as cohesion.[2] Water molecules have a greater tendency to adhere together on the surface. Resistance to movement is slightly greater on the surface of water because there is more cohesion on the surface of water.[2] Surface tension is the force of attraction between surface molecules of a fluid.[3] Surface tension is not a factor if the moving body part is completely submerged in water. It becomes a significant factor when a limb breaks the surface of the water. Therapeutically if a patient is extremely weak, movements may be performed more easily in the water just beneath the surface rather than at or on the surface.

Aquatic Exercise Research

There are many benefits of aquatic therapy. Exercising in water is effective for improving strength, muscular endurance, cardiorespiratory endurance, range of motion (ROM), agility, and psychological well-being, while minimizing pain.[17-19] The purpose of this section is to provide the reader

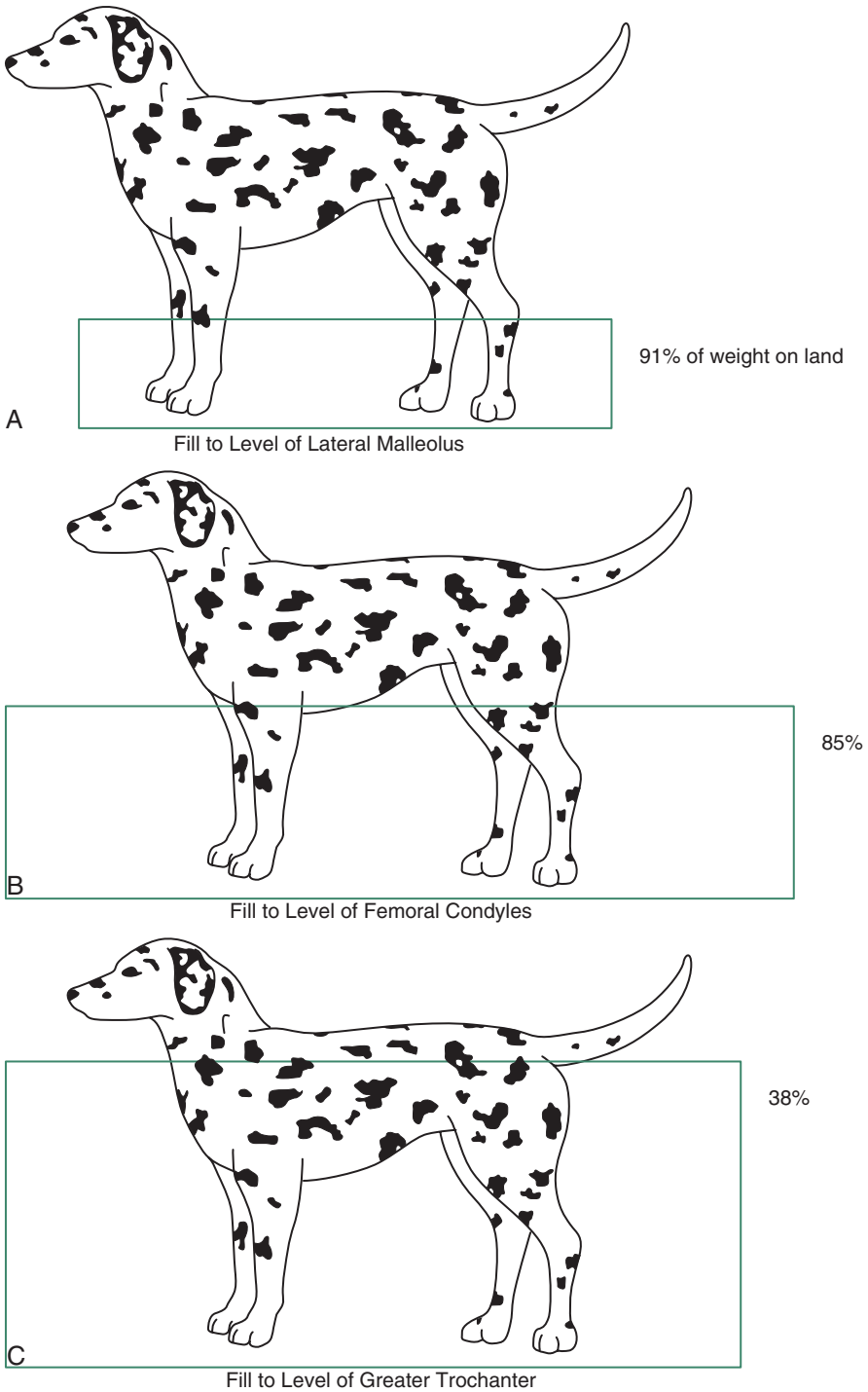


Figure 31-1 **A,** Dog in water to the level of the lateral malleolus. **B,** Dog in water to the level of the lateral epicondyle. **C,** Dog in water to the level of the greater trochanter. (Data from Levine D, Marcellin DJ, Millis DL et al: Effects of partial immersion in water on vertical ground reaction forces and weight distribution in dogs, *Am J Vet Res* 71:1413-1416, 2010.)

with information regarding human studies of the benefits of aquatic therapy. Table 31-1 provides a summary of these studies. It is likely that similar principles apply to animals.

Gravity is the primary resistant force to exercise on land, whereas viscosity, friction, and turbulence are the primary resistant forces to exercise in water; these properties have a direct effect on heart rate and oxygen uptake.[20] Arm exercises on land produced an increase in heart rate from a resting rate of 77 beats per minute to 92 beats per minute in men (age 21 to 27) and from 74 to 96 beats per minute in women (age 20 to 29). Exactly the same arm exercises in water produced an increase in heart rate from 60 to 99 beats per minute in men and from 60 to 98 beats per minute in women. Compared with the subjects exercising on land, the subjects exercising in water exercised at slightly higher heart rates and had a greater overall increase in heart rate because of the initial lower resting heart rate. Subjects in this study had lower resting heart rates in water than on land because of two factors: the reflex response of the cardiovascular system to the cold receptors of the skin, and the effect of hydrostatic water pressure exerted on the submerged body.[20] The water temperature ranged from 26° to 26.5° C (78.8° to 79.7° F) during aquatic exercise. As discussed later, water temperature directly affects heart rate. Leg exercises further increased heart rate during water exercises: 76 (resting) to 124 (exercise) beats per minute for men on land versus 60 (resting) to 139 (exercise) beats per minute in water, and 73 (resting) to 126 (exercise) beats per minute for women on land versus 60 (resting) to 126 (exercise) beats per minute in water. The increased weight of legs as compared with arms may increase the necessary energy expenditure for exercises performed on land

Table 31-1 Summary of Human Studies on the Benefits of Aquatic Therapy

Study	Study Subjects	Study Parameters	Outcome
CARDIOVASCULAR STUDIES IN AQUATIC THERAPY			
Avellini et al., 1983	Three groups of five men	Subjects trained on cycle ergometer 5 days/wk for 1 hr at 75% $V_{O_{2max}}$; group 1 exercised on land and groups 2 and 3 exercised in water at temperatures of 32° C and 20° C, respectively	Water groups exercised at a lower heart rate than land group
Bravo et al., 1997	77 postmenopausal women	Exercised in waist-deep water for 60 min 3 days/wk × 12 months	Improved functional fitness and psychological well-being
Choukroun and Varene, 1990	11 healthy volunteers (four women, seven men)	Subjects were immersed to neck in 25°, 34°, and 40° C water; cardiovascular demands measured at rest	Cardiac output increased significantly at 40° C versus lower temperatures
Evans et al., 1978	Six men	Walked and jogged at similar metabolic intensities on a treadmill in waist-deep water	Only one third to one half of the normal walking and jogging speed was required to work at the same level of energy expenditure in water as compared with on land (on a treadmill)
Hall et al., 1998	Eight women (mean 30.25 years old)	Submaximal exercise on land and on a treadmill in chest-deep water at water temperatures of 28° and 36° C	Heart rate higher at 36° compared to 28° C; walking in chest-deep water yields higher energy costs than walking at similar speeds on land
Johnson et al., 1977	Four men (mean 24 years old) and four women (mean 24.5 years old)	Same exercises performed by the same subjects on land and in water: shoulder ABD/ADD and hip flexion/extension with limbs extended	Decreased resting heart rate in water and increased V_{O_2} demands when exercising in water
Melton-Rogers et al., 1996	Eight adults with adult-onset rheumatoid arthritis (30-40 years old)	Graded maximal exercise test on a stationary bike on land and running in water wearing a flotation device	Higher maximum perceived exertion and respiratory rate were seen in water running; water provides a means for exercise for individuals with rheumatoid arthritis

Table 31-1 Summary of Human Studies on the Benefits of Aquatic Therapy—cont'd

Study	Study Subjects	Study Parameters	Outcome
Perk et al., 1996	20 stable COPD patients	Cardiorespiratory parameters were measured on land and in temperature-controlled water (32° C) at rest and during a 15-min submaximal upper-body exercise program	At rest in water there was a decrease in systolic and diastolic BP
Whitley and Schoene, 1987	12 healthy female college students	Heart rate was measured immediately after walking in waist-deep water and then on treadmill at same speed, duration, and distance	Heart rates were higher at each speed when walking in water vs. walking on land
RANGE OF MOTION AND STRENGTH STUDIES IN AQUATIC THERAPY			
Suomi and Lindauer, 1997	17 women with arthritis	Exercise subjects participated in three 45-min aquatic training sessions/wk for 6 wk; control subjects were asked to refrain from participating in any organized physical activity	Greater increase in strength and ROM occurred in the aquatic therapy subjects
Tovin et al., 1994	14 men and six women (16-44 years old) with ACL reconstructions	10 subjects participated in traditional rehabilitation protocol (stationary cycling, gait training, steps, hip flex/ext, ABD/ADD in standing, and knee flexion in sitting), whereas other 10 subjects performed same exercises in a pool; performed over first 8 postoperative wk	Rehabilitation program for patients with intraarticular ACL reconstruction performed in a pool was more effective in reducing joint effusion and facilitating recovery of lower extremity function as indicated by Lysholm scores; rehab in water is equally effective as on land for restoring knee ROM and quadriceps strength

ABD, Abduction; *ACL*, anterior cruciate ligament; *ADD*, adduction; BP, blood pressure; ROM, range of motion.

because of the increased mass and pull of gravity. This study demonstrated that heart rate and oxygen uptake were greater when performing exercises in water compared with performing the same exercises on land.[20]

The metabolic requirements are also greater for exercises performed in water than for exercises performed on land. In a study by Johnson et al.,[20] men and women performed the same arm and leg exercises in water and on land. Oxygen uptake during exercise in water was 34% greater in men and 27% greater in women as compared with the same exercises on land.[20] The differences between men and women are probably owing to different body compositions (male body composition tends to be more muscular, which requires more oxygen) and heights (men tend to be taller and have longer extremities, thereby having a longer lever arm, which requires more exertion to move). Another study found that subjects could walk or jog at slower speeds in water than on land while expending the same amount of energy as measured by oxygen consumption. In this study subjects walked at 2.6 to 3.5 km/hour (1.6 to 2.2 mph) in water, whereas subjects on land walked or jogged at speeds of 5.5 to 13.4 km/hour (3.4 to 8.3 mph). Both expended the same amount of energy, despite the greater velocity of people walking on land.[21] A study by Whitley and Schoene[19] supports these data with the observation that various types of exercises performed in water (aquatic calisthenics, shoulder and leg exercises, bicycle ergometry exercises, walking, and jogging) require a significantly greater amount of energy expenditure compared to the same exercises performed on land. Subjects in a study by Hall et al.[22] also expended more energy walking in water than on land at speeds greater than 4 km/hour (2.5 mph).

When aerobic exercise is used in the rehabilitation process, it may aid in improving cardiovascular fitness and weight reduction. Melton-Rogers et al.[23] compared exercise during land bicycle ergometry with running in water with a flotation device in individuals with rheumatoid arthritis. Higher ratings of perceived exertion (RPE) by the subjects and respiratory exchange ratios were seen during water running.[23] These studies demonstrate that walking in water at specific speeds may be sufficient to meet heart rate requirements for developing and maintaining cardiorespiratory fitness.

Aquatic exercises are also beneficial for muscle strengthening. Bravo et al.[17] performed a study on postmenopausal women who exercised in a pool with waist-deep water for 60 minutes, three times a week for 12 months. The average strength gain as a component of functional fitness was approximately 18%. Because of buoyancy water walking can provide an excellent alternative method of achieving and maintaining muscle strength and ROM in individuals with lower limb joint problems such as osteoarthritis (OA). Although exercise in water may not be as effective in achieving maximum muscle performance as land exercises, rehabilitation in water may minimize the amount of joint effusion and lead to greater functional improvement.[24] Closed-chain exercises such as walking in water are more closely associated to movements in everyday activities than open-chain exercises because they recruit muscles in a more functional manner.[25] Performing closed-chain exercises in an environment in which weight-bearing forces are decreased may minimize or eliminate damage and inflammation to the soft tissues, while maximizing functional training. The aqueous environment may also aid in reducing knee pain and joint effusion,[24] which may facilitate the recovery of lower-extremity function after cranial cruciate ligament (CCL) stabilization in dogs.

Water's buoyancy can ease the performance of exercise activities while also providing proprioceptive feedback to aid in the rehabilitation process. The effect of buoyancy allows for gentler active exercises by decreasing the loads placed on the injured tissues compared with exercises performed on land.[18] Aquatic exercises may be used as a transition to land-based exercises in postsurgery or postinjury rehabilitation. Water exercises are generally less painful than land exercises because of the support that buoyancy provides. Therefore water exercises may result in less discomfort maintain ROM and functional movement, and provide a better sense of security when initiating active movements.[18]

There are many physiologic effects resulting from exercise in heated water. Among them are increased circulation to muscles, increased joint flexibility, and decreased joint pain.[26] Templeton et al. performed a study to determine whether subjects with rheumatic diseases experienced changes in joint flexibility and functional ability following an aquatic therapy program.[26] Subjects exercised in water for 8 weeks. Exercises consisted of therapeutic games emphasizing both isotonic and isometric exercises of the trunk, neck, and upper and lower extremities. Water temperature was maintained at 32.9° C (91.3° F), and the air temperature was maintained at 31.7° C (89° F). Active ROM and a Functional Status Index test that recorded the degree of assistance, pain, and difficulty experienced in the performance of 18 tasks were measured before and after the 8-week aquatic therapy session. The results of the study indicated that pain decreased with therapy, and active ROM and functional ability increased. The decreased pain and improvement in performing daily activities had a significant impact on the overall increased functional ability of the subjects.[26] These findings support aquatic therapy as an effective rehabilitation technique for increasing functional ability and joint flexibility in people with severe joint disease. Osteoarthritic and rheumatoid arthritic patients participated in another study of the effects of aquatic exercise on ROM and strength.[27] Control subjects were asked to refrain from participating in any new physical activity throughout the duration of the study, whereas the exercise group participated in an aquatic exercise program. After 6 weeks, hip strength and ROM were measured. Hip strength in the aquatic exercise group increased 10.9%, whereas ROM increased 11.8% as compared with the control group.[27]

Water temperature may have a significant effect on the cardiovascular response to exercise. Subjects in cool water exercise at lower heart rates than those exercising on land at the same workload for several reasons.[28] If the water temperature is low, peripheral vasoconstriction occurs, blood moves centrally, venous return is enhanced, and stroke volume increases. Therefore to maintain the same cardiac output, heart rate decreases. In one study, subjects training in cool water (20° C [68° F]) had enhanced stroke volume and decreased heart rate, thereby increasing exercise efficiency.[28] It is also our experience that dogs exercise at lower heart rates walking on underwater versus land treadmills at the same velocity and length of time (unpublished data, Millis DL, 2000).

Blood pressure is also affected by water temperature. In a study of subjects at rest while submerged in 32° C (89.6° F) water, systolic blood pressure decreased 14 ± 14 mm Hg and diastolic pressure decreased 6 ± 10 mm Hg because of peripheral arterial dilation. On the other hand, exercising in water at temperatures that exceed body temperature may increase cardiovascular demands above those of exercise alone.[12] In one study, cardiac output was measured at rest while people were immersed in water temperatures of 25°, 34°, and 40° C (77°, 93.2°, and 104° F).[30] Cardiac output of those immersed in 40° C water increased significantly. The authors of this study recommend that elite athletes train in water temperatures between 26° C and 28° C (78.8° F and 82.4° F) to prevent any heat-related complications.[30] This temperature range is also recommended by the authors for pools in which healthy dogs are training. Lower temperatures are also apparently tolerated quite well by dogs, especially those with thicker hair coats.

All of the benefits of aquatic therapy in humans (improved strength, muscle endurance, cardiorespiratory endurance, increased ROM, decreased stress on healing tissues, and minimized pain) may also apply to dogs. The type of aquatic therapy used by therapists, veterinarians, and owners depends on the specific rehabilitation needs for each individual. An understanding of the benefits of aquatic therapy aids the prescription of specific protocols.

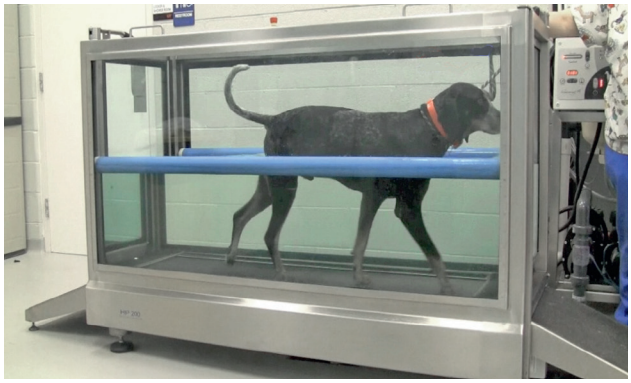


Figure 31-2 Dog walking on underwater treadmill.

Aquatic Therapy Equipment

This section provides the reader with information concerning the types of aquatic therapy and equipment currently available for dogs. The anticipated use, size of the dogs receiving therapy, and funds available help determine the type of equipment best suited for rehabilitation. Some of the available forms of aquatic therapy include underwater treadmills, pools of various sizes, and whirlpools (Figure 31-2).

Underwater treadmills are very useful for aquatic therapy. In addition to the buoyancy and resistance effects of water, the treadmill encourages walking or jogging with a more normal gait pattern than is achieved with swimming. The height of the water can be adjusted, which alters the buoyancy and alters joint motion. Underwater treadmills are very useful in neurologic patients to help with gait patterning with the support of the water. Units typically have entrance and exit doors, a storage tank for water, heater, filters, and water treatment with chlorine or bromine. Dogs typically walk into the unit, the doors are sealed shut, and the water is pumped into the chamber until the desired height is reached. The treadmill is then turned on at the appropriate speed. On occasion, a therapist may accompany a patient to assist with gait training. After treatment is completed, the water is pumped out, the doors are opened and the patient is rinsed off and dried. Units are available in some countries that have the water in the treadmill compartment, with an adjustable treadmill that lowers into the water. This type of unit helps to save space because a storage tank is not necessary, but the dog must be elevated on a lift to step on the treadmill; therefore, caution must be used to avoid the danger of the dog jumping from the lift or treadmill.

Swimming pools may also be used for rehabilitation. Several options must be considered when installing a pool. Some of the considerations include whether jets or propellers used to circulate the water and add resistance to exercises will be useful; the location of the jets; the possibility of adding an underwater treadmill into the pool; hooks on the inner walls to which safety straps may be attached; manual versus computer-controlled water treatment and heating systems; above-ground versus in-ground pools; and stairs, lifts, or ladders to enter the pool. In addition the physical aspects of the enclosure and surrounding area must be considered. For example the humidity and condensation associated with a heated indoor pool must be considered. The floor surface around the pool must also be carefully designed to avoid slipping and falling by both patients and handlers.



Figure 31-3 Dog receiving aquatic therapy in whirlpool.



Figure 31-4 Dog wearing life vest.

Other forms of aquatic therapy include hot tubs or whirlpools used in hospitals (Figure 31-3). Cost-effective forms of aquatic therapy for smaller dogs include bathtubs, large basins, and plastic pools. Taking a dog to a lake or river is also a cost-effective means of providing aquatic therapy, but caution must be exercised when swimming dogs with recent incisions in these environments. Strong water currents and natural hazards must also be avoided.

Equipment used in aquatic therapy for dogs can be relatively simple and inexpensive. One useful piece of equipment is a pet life preserver (Figure 31-4). These are designed to support the dog and allow free breathing. They



Figure 31-5 Using a life vest to guide a dog during pool exercises.



Figure 31-6 Dog being transferred into a pool using a lift. (Courtesy Trish Penick, San Diego, CA.)

may include quick-release buckles with adjustable straps and handles for an attendant to assist the dog while swimming. Various sizes are available. They are also useful to control and guide the dog while performing pool exercises (Figure 31-5). Other devices used in aquatic therapy are balls and sticks for fetch games, a lift for entering or exiting the water (Figure 31-6), and harnesses to direct and maintain contact with the dogs if the attendant is not in the pool.

Persons working in the water with dogs may also benefit from specialized equipment such as wetsuits or other protective clothing to guard against prolonged exposure to pool chemicals or cold water (see Figure 31-6). Those working from outside the pool with dogs in water should also expect to get wet and should take precautions.

The equipment required to clean and maintain tanks and pools includes disinfectant solutions, protective gloves, buckets, a water hose, brushes with handles to enable scrubbing of crevices, and clean, dry towels.[2] According to Hecox et al., there is no conclusive evidence regarding clear advantages of any one specific type of disinfectant over another.[2] Studies and reported observations suggest sodium hypochlorite (5.25%), sold as household laundry bleach, is an inexpensive and effective disinfectant. However, bleach may corrode the surface of stainless steel over time.[31] Although manual scrubbing with standard germicidal detergents may reduce bacterial contamination, complete removal of bacteria from the drains, edges, and bottom of the tank may not be possible.[2]

Chlorine or bromine are commonly used to sanitize the water in pools and underwater treadmills. These levels are checked on a daily basis and adjusted as needed. Occasionally a reaction such as a skin rash occurs but by rinsing dogs off with water after aquatic therapy this is minimized. Other values such as the pH and alkalinity are also checked and adjusted. Dogs that defecate or urinate in the water or are incontinent pose additional problems in maintaining the cleanliness of tanks or pools. If an animal eliminates or is incontinent in the water, the best way to minimize cross-contamination is to drain the pool after each use and sanitize it. It is also recommended that this precaution be taken with dogs that have open wounds. If draining the pool is not possible because of its size, the pool should be shocked with a chemical disinfectant, and dogs that have open wounds should not use the pool for a period of time recommended by the manufacturer to ensure that the water has been sanitized. Although soiling the water poses potential problems, screening dogs before aquatic therapy may help to reduce or avoid this problem. Dogs should be walked before engaging in aquatic therapy to allow them an opportunity to eliminate.

Aquatic Therapy for Dogs

Conditions Benefiting from Aquatic Therapy

There are many conditions for which aquatic therapy may be beneficial, including rehabilitation of postoperative fractures, CCL stabilization, neurologic conditions, tendinitis, conditioning, and other disorders in which a dog is reluctant to use the limb or there is a lack of strength, ROM, proprioceptive ability, or weight-bearing status.[32] The potential benefits such as strengthening, cardiovascular endurance, and improved function have been documented in humans, and aquatic therapy is now becoming more common in veterinary medicine. Outcome assessments of aquatic therapy programs are important for documenting progress and to determine the effectiveness of programs.

Contraindications and Precautions to Aquatic Therapy

Some dogs fear water or are reluctant to swim, and this must be considered before attempting treatment. In general, dogs tolerate an underwater treadmill better because the water rises from the bottom rather than having the dog enter or be lowered into the water, such as a swimming pool. If a dog panics the dog and handler could be placed in a potentially dangerous situation. The dog may injure itself with excessive thrashing. The handler may be injured

trying to restrain a panicked dog, or may also risk attack by the animal. Questioning the owner regarding a dog's history of swimming is critical, in addition to prudent safety and handling techniques. If possible, dogs that will be receiving aquatic therapy following nonemergency surgical procedures should be introduced to aquatic therapy before surgery. This allows for evaluation of the dog's disposition toward swimming and also allows the dog to be introduced to the therapy received postoperatively. Preoperative evaluation is not possible for some traumatic injuries, such as fractures. When receiving aquatic therapy, dogs should never be left unattended in the water.[33]

Some veterinarians believe it is better to wait until incisions or open wounds are healed before placing the animal in water to minimize the risk of infection; others may begin aquatic therapy after the incision is sealed but before the incision is completely healed. A safe recommendation is to wait until after suture removal if there are no wound complications, including discharge from the incision, gapping of the wound edges, or any evidence of infection. However, there may be conditions that warrant earlier use of aquatic therapy. Regardless of when therapy is started, the wound should be sealed before initiation. The cleanliness of the water may influence this decision, as well as the animal's overall health and medical history. Betadine impregnated adhesive drapes may be also be used to help seal an incision area prior to entering the water to protect incisions.

The cardiovascular fitness of the dog must be considered because many dogs are initially unable to swim more than a few minutes before fatiguing. Swimming the dog several times daily for only 2 to 5 minutes may still result in significant gains in strength, ROM, function, and overall cardiovascular fitness.[33,34]

Aquatic Therapy Basics

Reluctance to use a limb following injury is a common problem. Dogs that are reluctant to use a limb on land may use it in water to swim or walk on the bottom. Dogs that may benefit from extra buoyancy because of weakness may be assisted by placing a life jacket or other flotation device around the dog's trunk, or by the use of an overhead harness.[33] Owners may provide additional motivation to encourage dogs to walk or swim. Having the animal fetch a ball or other object may have the same motivational effect. If possible swimming with the dog is another excellent way of encouraging therapy, in addition to ensuring that someone is present if the animal has any trouble. Caution must be used when swimming injured dogs in lakes and rivers. Dogs must be prevented from diving or lunging off of docks or the shore when entering the water to prevent injury. Wading into the water with the dog until the dog is buoyant is recommended. Maintaining contact with the dog at all times is important. In addition, water sanitation and natural hazards must be considered.

Walking in the water, either on the bottom of the pool or on an underwater treadmill, is an excellent way to allow a dog with painful joints to exercise more comfortably because of the buoyancy provided by the water. This type of exercise may be used as a progression to swimming if the animal is either too weak or in too much pain to initially swim.

In addition to the buoyancy effects of swimming or walking in water, joint kinematics are also altered during aquatic therapy. Knowledge of aquatic kinematics is useful in designing specific exercise programs. For example in one study comparing swimming with ground treadmill walking, dogs that swam had significantly greater stifle flexion and total stifle ROM compared with ground treadmill walking (Table 31-2).[35] Marsolais et al. published data supporting swimming as an effective rehabilitation modality in dogs following extracapsular repair of cranial cruciate rupture. Two groups of dogs, control and surgical, were enrolled in the study. The surgical group consisted of 26 adult dogs that underwent debridement of a ruptured CCL, partial or complete medial meniscectomy, and extracapsular stabilization. The control group consisted of 26 healthy dogs without cruciate injury. All of the dogs in the surgical group underwent the same aquatic rehabilitation program focused on swimming. The rehabilitation sessions were performed twice daily, 5 days per week and consisted of 10 minutes of limb massage and passive ROM, 10 minutes of walking on a treadmill, and 10 to 20 minutes of pool time. Pool time consisted of alternating 1 minute of swimming and 1 minute of rest for a total of 5 to 10 minutes of swimming. None of the dogs were allowed to touch the floor of the pool during the swimming session except when entering and exiting the pool. The owners of the control dogs were instructed to allow normal activity several days before data collection. The control dogs did not participate in any part of the rehabilitation program. Data collection consisted of measuring hip, stifle, and hock flexion and extension with slow (0.9 m/s) and fast (1.3 m/s) walking on a treadmill and while swimming. Data collection revealed increased hip, stifle, and hock flexion ROM in the control group during swimming over walking on the treadmill and increased stifle (50° swimming and 30° walking), and hock (50° swimming and 35° walking) flexion ROM in the surgical group during swimming. Stifle and hock extension, however, was less when swimming compared with walking in both groups. Hip joint ROM was not affected in the surgical group when swimming compared with walking; however, the control group demonstrated increased hip ROM with swimming over walking. The authors summarized that swimming is an effective modality to gain stifle and hock flexion following extracapsular stabilization of CCL because it allows early mobilization without the added stress of weight bearing. Peak vertical forces were 18.5% higher in the rehabilitation group 6 months after surgery.[36]

Table 31-2 **Results of Kinematic Evaluation of the Hip, Stifle, and Tarsal (Hock) Joints During Swimming, Fast Walking (1.3 m/s), and Slow Walking (0.9 m/s) in Healthy Dogs (Control; n = 13) and Dogs (7) That Had Undergone Surgical Management of a Ruptured Cranial Cruciate Ligament and Were Enrolled in a Postoperative Aquatic Rehabilitation Program**

		SWIMMING		FAST WALKING		SLOW WALKING	
Joint	**Variable**	**Control**	**Rehabilitation**	**Control**	**Rehabilitation**	**Control**	**Rehabilitation**
HIP							
	Maximal flexion (°)	83.6 ± 15.1[a]	75.6 ± 7.9[d]	95.9 ± 11[a]	104.5 ± 12.5[e]	98.6 ± 11.4[a]	104.7 ± 13.8[e]
	Maximal extension (°)	128.2 ± 14.4[a]	114.9 ± 11.7[d,*]	130.5 ± 8.3[a]	138.4 ± 15.1[e]	130.7 ± 10.7[a]	137.1 ± 18.6[e]
	Angular velocity (°/s)	160.9 ± 68.1[a]	122.7 ± 47.0[d]	88.2 ± 15.0[b]	87.4 ± 21.8[e]	62.8 ± 15.3[c]	68.7 ± 11.3[f]
STIFLE							
	Maximal flexion (°)	46 ± 10.7[a]	46 ± 6.3[d]	97.1 ± 8.7[b]	100.1 ± 7.1[e]	101.5 ± 8.0[b]	102 ± 9.3[e]
	Maximal extension (°)	112.9 ± 11.8[a]	94.4 ± 16.8[d,*]	133 ± 7.7[b]	130 ± 9.3[e]	135.1 ± 9.2[b]	129.4 ± 10.6[e]
	Angular velocity (°/s)	151.5 ± 39.3[a]	94.1 ± 19[d,*]	182.3 ± 50.9[a]	138.9 ± 48[d,*]	154 ± 38.2[a]	105 ± 47.6[d,*]
HOCK							
	Maximal flexion (°)	52.9 ± 12.1[a]	74.7 ± 7.8[d,*]	107 ± 7.3[b]	120.2 ± 13.7[e]	111.9 ± 7.8[b]	124.8 ± 14.3[e]
	Maximal extension (°)	127.7 ± 10.8[a]	126.8 ± 12.6[d]	141.6 ± 8.7[b]	153.8 ± 12.4[e,*]	141.9 ± 8.7[b]	152.7 ± 11.3[e,*]
	Angular velocity (°/s)	216.9 ± 62.7[a]	134.1 ± 48.9[d,*]	80.9 ± 20.7[b]	70.5 ± 10.8[e]	55.8 ± 14.8[c]	48.6 ± 8[f]

Data are given as mean ± SD.

[a,b,c]For dogs in the control group, values in each row with different letter superscripts were significantly ($P < .05$) different.

[d,e,f]For dogs in the rehabilitation group, values in each row with different letter superscripts were significantly ($P < .05$) different.

*Significantly ($P < .05$) different from corresponding value for control group.

From Marsolais GS, McLean S, Derrick T, et al. Kinematic analysis of the hind limb during swimming and walking in healthy dogs and dogs with surgically corrected cranial cruciate ligament rupture. *JAVMA* 222:739-743, 2003.

Table 31-3 Conditions for Achieving Maximum and Minimum Joint Flexion and Extension during Walking on Either a Ground or an Underwater Treadmill

	MAXIMUM JOINT MOTION		MINIMUM JOINT MOTION	
Joint	**Extension**	**Flexion**	**Extension**	**Flexion**
FORELIMB				
Shoulder	142 degrees—ground	93 degrees—water	132 degrees—water (level 1)	125 degrees—ground (level 1)
Elbow	159 degrees—water (level 3)	70 degrees—water (level 3)	124 degrees—ground	98 degrees—water (level 4)
Carpus	193 degrees—water (level 4)	97 degrees—ground	185 degrees—water (level 1)	119 degrees—water (level 4)
HINDLIMB				
Hip	141 degrees—ground	93 degrees—water (level 2)	120 degrees—water (level 1)	115 degrees—ground
Stifle	141 degrees—ground	64 degrees—water (level 2)	113 degrees—water (level 1)	109 degrees—ground
Hock	156 degrees—water (level 3)	81 degrees—water (level 2)	135 degrees—water (level 1)	115 degrees—ground

Values indicate the mean maximum and minimum joint flexion or extension angles of 8 dogs that occur under the indicated conditions during walking.
Ground, Walking on a ground treadmill; *water,* walking on an aquatic treadmill; *level 1,* water level at the greater trochanter; *level 2,* water level at the stifle joint; *level 3,* water level at the hock joint; *level 4,* water level at the bottom of the foot.
From: Jackson A, et al.: *Joint kinematics of dogs walking on ground and aquatic treadmills.* In Proceedings of the 2nd International Symposium on Rehabilitation and Physical Therapy in Veterinary Medicine, Knoxville, 2002.

However, stifle extension was less in swimming dogs than in dogs undergoing ground treadmill activity. In addition dogs had less stifle flexion, extension, and total ROM following stifle surgery as compared with normal dogs.

Rehabilitation of dogs on an underwater treadmill may provide additional benefits regarding joint kinetics. The relative degree of buoyancy and resistance to walking in water can be adjusted by changing the water level. In addition, adjusting the water level to the level of various joints may further influence joint kinematics because those particular joints must gain the necessary momentum and force to break through the water surface and overcome surface tension of the water. One study compared dogs walking on a ground treadmill to dogs walking on an underwater treadmill at the same velocity (Table 31-3).[37] In addition the water level was adjusted to the greater trochanter (level 1), stifle (level 2), hock (level 3), or the bottom of the foot (level 4). Joint motion was determined in both front and hind limbs, with each limb in a fully flexed non–weight-bearing position, with each limb in the final propulsion stage of weight bearing, and with each limb in the initial braking stage of weight bearing. Unlike swimming there was no significant reduction in joint extension with underwater treadmill walking during the initial braking phase of stance with water levels at and distal to the stifle. Therefore dogs had full active extension of all joints during a limb cycle, unlike joint motion during swimming. However, hip, stifle, and hock extension during the late phase of propulsion was decreased when water was filled to the level of the greater trochanter. Joint flexion was increased with underwater treadmill walking. In general joint flexion was greatest with water levels at or higher than the joint of interest. Therefore the advantages of additional joint flexion achieved with swimming are also present with underwater treadmill walking, but full extension of joints also occurs with treadmill walking, which is not the case with swimming.

A flotation device, such as a children's water wing, applied to a limb may also be used to alter joint kinematics. For example, if additional motion of all joints of a particular limb is desired, the device may be placed on the distal limb. If additional joint extension and increased stance time are desired, the device may be placed on the contralateral limb. The altered sensation and unbalanced buoyancy of opposite limbs contribute to altered joint kinematics and may be used to achieve certain goals during rehabilitation.

Underwater Treadmill Therapy

Aquatic therapy performed in an underwater treadmill allows for active range of motion similar to that in a normal gait using the warmth, buoyancy, and resistance of water. Underwater treadmill therapy is used when there is a need for walking or jogging using a relatively normal gait pattern, with increased muscle or cardiovascular demand and minimal risk of injury. Secondarily, it may be used to

reduce muscle spasm or peripheral edema and improve mood or bolster confidence.[1-3]

Advantages

Activity in an underwater treadmill has several advantages over swimming or immersion in water. Dogs move in a natural walking or jogging gait pattern, and joint extension is more complete than with swimming, The speed of the treadmill may be adjusted. Slow speeds are used for early postoperative conditions, moderate neurological conditions, and patients beginning a conditioning program. Faster speeds may be used for higher levels of conditioning. The level of the water may be adjusted to alter the degree of buoyancy, This may reduce the risk of injury and reduces joint concussive forces. The resistance to movement through water increases the activity of muscles during the swing phase of gait. Warm water temperatures increase circulation, caloric expenditure, and nerve conduction velocity; improve coordination; and allow for increased soft-tissue elasticity.[1-3] Additionally, hydrostatic pressure exerted by the water on extremities aids in the reduction of edema.[4]

Underwater treadmill therapy allows for earlier intervention as compared with swimming. It promotes earlier active range of motion of joints especially in flexion, and strengthening of muscles. Functional carryover to land-based activities is usually seen very quickly.

Underwater Treadmill Temperature

Most underwater treadmills are maintained between 78 to 90 F. Warmer temperatures may be beneficial for relaxation of muscles. Cooler temperatures allow for more vigorous exercise with less risk of overheating. When treating a neurologic patient warmer water (85º to 90ºF) may be preferable. Orthopedic patients and dogs in conditioning programs do not require the same warm temperatures (78º to 83ºF).

When to Initiate Underwater Treadmill Therapy

Determining when therapy should begin is a decision made by the surgeon or veterinarian overseeing patient care. Following most orthopedic surgeries, 5 to 7 days is adequate if the incision is sealed with no drainage and the wound edges are well apposed. The incision may be covered with an adherent water-resistant bandage. However, some elect to wait until the time of suture removal to avoid possible incisional complications. This allows for sufficient initial tissue healing and initial therapeutic exercises. For surgeries such as total hip replacements, 4 weeks or more may be recommended. Neurologic rehabilitation postsurgery varies depending on the procedure and the quality of the stabilization. Particular care should be taken to support the patient and not allow the incision to become wet. Dogs with conservatively managed intervertebral disk disease should not have therapy prior to 1 month after injury. These are common time frames for early aquatic intervention and vary from patient to patient.

Equipment

Before getting in the water with the patient, precautions should be taken on the part of the therapist. Patient may be fitted with a flotation vest to provide additional buoyancy. Be certain that the personal flotation device (PFD) conforms well to the body and does not interfere with either shoulder extension or hip flexion. Therapists may want to wear either a shortie or full-length wetsuit depending on their skill level and ambient temperature above the water. Wearing a wetsuit minimizes injury by flailing paws or dogs climbing onto the therapist. It is also important to have a supply of float toys, balls, and assistive devices like cuffs made from pool noodles, weight cuffs, elastic bands, rubber bands, and/or hair scrunchies easily accessible. These are used to encourage different range of motion of the affected or unaffected limbs. Pool noodles may also be placed along side, in front or behind the animal to keep it centered while walking on the treadmill.

Transfers

Transferring the patient into and out of the underwater treadmill is generally easy because patients typically walk into the chamber. The primary concern is safety as well as the comfort of the patient. Therefore, support with a sling may be necessary if the patient is weak or reluctant to enter at first. Weak patients may also benefit from sling support while in the underwater treadmill. This may be supported by the therapist or a support pole that runs through the handle of the sling.

Evaluation in the Underwater Treadmill

After the patient is in the underwater treadmill, the therapist may observe the gait of the patient from the side, front, back or top. Gait evaluation in the underwater treadmill is often very revealing because it is like watching the patient in slow motion, with exaggerated motion of the limbs and joints. If the therapist accompanies the patient in the treadmill, it is possible to palpate joints and muscles while they are moving through voluntary active range of motion. Quality of joint movement, joint integrity, and determination of individual muscle contractions can all be assessed quickly and easily.

Treatment

Techniques for performing rehabilitation in the underwater treadmill include interval training, continuous walking or jogging, walking that is enhanced by passive or manual techniques by the therapist, and walking while wearing a resistive or adaptive device. Interval training is performed early in rehabilitation and conditioning and consists of short periods of slow walking alternating with short rest

periods. This generally results in more rapid progress in strength and cardiovascular conditioning rather than allowing the dog to walk for a longer period until exhaustion. The speed is generally slow to begin with and may be increased every other session if the patient is doing well. The total time may be increased 10 to 15% per week if adequate recovery and progress are made. In enhanced passive or manual walking, the therapist provides manual assistance to limb and joint movement at the appropriate times of the gait cycle to facilitate normal gait or larger range of motion. When doing resisted walking the dog is restrained by holding a vest, or using a leash or harness.

Adaptive devices to enhance underwater treadmill therapy include flotation cuffs, small weight cuffs, and elastic bands. These can be attached to different segments of the limb to encourage exaggerated movement or provide added resistance. Facilitation of joint and limb movement, particularly in attempting to improve active range of motion, is relatively easy in the water.

During the treatment session the therapist must constantly monitor the patient. Be aware of the patient's level of anxiety and fatigue to allow adequate rest periods. Labored breathing or loss of the rhythmic gait pattern are indicators of stress or fatigue. Patients that are older, poorly conditioned, or obese should especially not reach the point of fatigue.

Precautions and Contraindications

There are several precautions and contraindications for underwater treadmill rehabilitation. Patients that have a high level of fear of the water should not undergo aquatic therapy. Typical conditions that do not respond well to underwater treadmill rehabilitation are acute biceps tendonitis and other severe muscle strains as well as fracture repair with external fixators. Aquatic therapy should be used with caution or avoided with cardiac conditions that limit exertion levels, especially in warm water. Patients with borderline respiratory conditions may have difficulty because of the hydrostatic pressure placed on the thorax. Patients with open wounds or surface infections should not be placed in the underwater treadmill. Finally, incontinent patients or patients with diarrhea should not undergo underwater treadmill therapy.

Pool Therapy

Aquatic therapy performed in a swimming pool, or any deep body of water, allows for active range of motion or passive range of motion performed using the warmth, buoyancy, and resistance of water. Pool therapy is used when there is a need for increased muscle or cardiovascular demand and minimal risk of injury or when weight bearing and balance are problems. Secondarily, it may be used to reduce muscle spasm or peripheral edema and improve mood or bolster confidence.[1-3]

Advantages

Immersion in water to perform exercise has several advantages over an underwater treadmill. The patient is almost completely non–weight bearing and the thoracic and lumbar spines are suspended in a neutral position by water buoyancy. This may reduce the risk of injury and eliminates almost all joint concussive forces. The antigravity muscles are essentially inactive, resulting in only concentric muscle contractions that may minimize the potential for delayed onset muscle soreness. Warm water temperatures increase circulation, caloric expenditure, and nerve conduction velocity; improve coordination; and allow for increased soft-tissue elasticity.[1-3] Warming of the tissues through water temperature and increased metabolic demand by way of swimming may also stimulate the release of endorphins and enkephalins that contribute to improving mood, relaxing muscles, and managing pain.[3] Additionally, hydrostatic pressure exerted by the water on extremities aids in the reduction of edema.[4]

Pool therapy allows for early intervention leading to faster recovery. It promotes earlier active range of motion of joints especially in flexion, and strengthening of muscles. Functional carryover to land based activities is usually seen very quickly. Another benefit to deep-water therapy is the capacity for performing vigorous sustained exercise especially when land-based activity is not possible, such as in an overweight dog.

Pool Temperature

Most therapy pools are maintained between 82° to 90° F. The most appropriate pool temperature for aquatic rehabilitation will vary depending on the type of facility and the predominant patient population. Outdoor facilities most often keep the water a few degrees warmer in the cool months (88° to 90° F) and a little cooler in the summer (82° to 86° F). The warmer temperatures are most beneficial for relaxation of muscles. The cooler temperatures allow for more vigorous exercise with less risk of overheating. Indoor facilities maintain more consistent temperatures based on their patient population.[5] When treating a neurologic patient warmer water (85° to 90° F) is preferable. Orthopedic patients and dogs in conditioning programs do not require the same warm temperatures (78° to 86° F). Overheating can be easily avoided by using above-water rests that allow evaporative cooling or by having the dog stand in a small cool pool placed next to the therapy pool. The pool temperatures noted here are variable based on the ambient temperatures if the pool is outside with a range of temperature from 50° to 115° F.

When to Initiate Pool Therapy

Determining when pool therapy should begin is a decision made by the surgeon or veterinarian overseeing patient care. Following most orthopedic surgeries, 14-30 days is

adequate. This allows for sufficient initial tissue healing and initial therapeutic exercises. For surgeries like TPLO, TTA, and joint replacements, 4-6 weeks or more is required to allow early bone healing. Neurologic rehabilitation post-surgery varies depending on the procedure and the quality of the stabilization. Conservative (nonsurgical) neurologic rehabilitation for conditions such as degenerative myelopathy or fibrocartilagenous embolic myelopathy begins as soon as the patient is medically stable, often immediately. Dogs with conservatively managed intervertebral disk disease should not have therapy prior to 1 month after injury to allow a period of healing of the weakened annulus fibrosis. Postsurgical patients with severe spinal cord injuries with minimal motor function may benefit from swim therapy as early as 5 to 10 days after surgery. Particular care should be taken to support the patient and not allow the incision to become wet. These are common time frames for early aquatic intervention and vary from patient to patient.

Equipment

Before getting in the water with the patient, precautions should be taken on the part of the therapist. Nearly every patient is fitted with a flotation vest to provide control and convey a sense of buoyancy to the dog. There are many reliable personal flotation devices (PFDs) made for dogs, such as Cascade Outfitters, Outward Hound, and Ruff Wear. A good PFD conforms well to the body and does not interfere with either shoulder extension or hip flexion. The therapist may want to wear either a shortie or full length wetsuit depending on their skill level and ambient temperature above the water. Wearing a wetsuit minimizes injury by flailing paws or dogs climbing onto the therapist. It is also important to have a supply of float toys, balls, and assistive devices like cuffs made from pool noodles, weight cuffs, elastic bands, rubber bands, and/or hair scrunchies easily accessible. These are used to make therapy fun or encourage different swim strokes.

Transfers

Transferring the patient into and out of the water can be performed in many ways. The primary concern is safety as well as the comfort of the patient. Entering and exiting the water are often the portions of rehabilitation in which the patient is at most risk of injury. Leaping into the water is the least desirable method. When using stairs to enter and exit the pool dogs may push back or push off and this can put an extremity at risk of injury owing to rapid or poorly controlled loading. Steep pool steps also place extremities in more demanding positions. Ramps work well especially for ambulatory patients. Dogs are less likely to launch themselves from a ramp. Stretchers can be most helpful for nonambulatory patients or those with some degree of paralysis. The therapist can easily slide a patient directly from the stretcher into the water. Lifts are often used. They may



Figure 31-7 A harness lift system can also be used.

be hydraulic or electric and use either a sling or a platform to safely place the patient in the pool. A platform lift (see Figure 31-6) has the added advantage of allowing the patient to be lowered into the pool while its feet remain on a solid surface. Depth adjustability of the platform lift also allows the therapist to control how much weight bearing the patient performs when standing at rest on the lift. This provides an opportunity for balance and weight shift activities during rest periods between swims. A harness lift system can also be used (Figure 31-7).

Evaluation in the Water

After the patient is in the pool positioning it in front of or beside the therapist allows for evaluation that cannot be performed on land. It is possible to palpate joints and muscles while it is moving through voluntary active range of motion. Quality of joint movement, joint integrity, and determination of individual muscle contractions can all be assessed quickly and easily. As an example, for a hip joint assessment, the therapist places his or her hand flat over the dorsal aspect of the hip while the dog is swimming. A normal healthy hip will move smoothly and all that will be felt is a rhythmic muscle contraction under the evaluator's hand. Changes in articular cartilage or loose bodies in the joint have a crepitant feel, as though a little sand is in the joint. Joint laxity is recognized by a range of sensations from subtle bumping, to clicking, to distinct clunking, to snapping often accompanied by an audible pop. These are indicators of abnormal femoral head movement within the joint capsule during a vigorous swim stroke. Other joints can also be assessed, to a lesser degree, with skillful palpation.

During the evaluation the therapist has ample opportunity to educate the owner about their findings. Encourage owners to ask questions about their dog's condition. In human practice it has been shown there is a strong



Figure 31-8 Dog swimming in a pool.

correlation between a thorough understanding of the dysfunction and good home program compliance.

Treatment

Techniques for performing rehabilitation in the water include interval swimming, free swimming (Figure 31-8), resisted swimming, swimming against a current, swimming that is enhanced by manual techniques, swimming while wearing an adaptive device, or passive techniques performed by the therapist. Interval swimming is performed early in rehabilitation and conditioning and consists of short periods of swimming alternating with short rest periods. This generally results in more rapid progress in strength and cardiovascular conditioning rather than allowing the dog to swim for a longer period until exhaustion. Free swimming often involves following the owner or therapist around the pool or retrieving. When doing resisted swimming the dog is restrained by holding the vest, or using a leash or harness. In enhanced swimming, the therapist provides manual contacts to the feet or extremity to facilitate a stronger contraction or larger range of motion. This technique is reminiscent of proprioceptive neuromuscular facilitation (PNF) used in human physical therapy.[6]

Adaptive devices to enhance swimming include flotation cuffs, small weight cuffs, and elastic bands. These can be attached to different segments of the limb to encourage exaggerated movement or provide added resistance. Facilitation of joint and limb movement particularly in attempting to improve active range of motion is relatively easy in the water. Passive range of motion can easily be performed with the dog either floating in the vest or standing in shoulder deep water. Watsu is a technique in which the therapist supports and moves a relaxed patient along the surface of the water using the resistance of the water to perform passive range of motion on the spine and/or extremities of the patient.[3]

During the treatment session the therapist must constantly monitor the patient. Be aware of the patient's level of anxiety. Know when to allow the dog to rest. Pushing the chin down, flattening the ears, and searching the pool perimeter are indicators of stress or fatigue. When exercising the patient look for other signs of fatigue such as a change in the swim stroke or in their breathing. It is also helpful to monitor the patient's tongue and gum color; cyanosis is indicated by a blue color to the tongue and mucous membranes. This is an indication to discontinue exercise immediately and allow a period of rest. Signs of distress previously listed precede this point, and patients that are older, poorly conditioned, or obese should especially not reach this point. It is important to keep patients motivated by changing the routine or positioning them so that they work at a level that maximizes recovery.

Precautions/Contraindications

There are some contraindications for pool rehabilitation. At times it is difficult to rule out if pool therapy is appropriate for a patient without attempting a session to monitor the patient's response. Typical conditions that do not respond well to pool rehabilitation are acute biceps tendonitis and other severe muscle strains as well as early stages of TPLO or fracture repair recovery. Swim therapy should be used with caution or avoided with cardiac conditions that limit exertion levels, especially in warm water. Patients with borderline respiratory conditions may have difficulty because of the hydrostatic pressure placed on the thorax. Patients with open wounds or surface infections should not be placed in pool. Finally, incontinent patients or patients with diarrhea should not undergo swim therapy.

The following case demonstrates the usefulness of aquatic therapy in restoring physical condition to a dog that had lost a great deal of function.

Case Report

Blackie, a 2-year-old hound dog, was presented with a nonunion fracture of the left femur. The fracture was a short oblique fracture of the midshaft of the femur. The original repair consisted of an intramedullary pin and cerclage wire. The fixation devices were prematurely removed 2 months after surgery. Blackie refractured the same bone 4 weeks later. At this time, an external skeletal fixator was placed. Complications associated with pin loosening and continued instability of the fracture site resulted in placement of an intramedullary pin. At this point the stifle was stiff. A recheck examination 2 weeks after surgery indicated that the dog was not using the leg. The dog was barely weight bearing while standing and was non–weight bearing at a walk. Twelve weeks after the last surgery, all fixation devices were removed. Following pin removal instability of the fracture site was palpated.

Seven months after the original fracture Blackie was examined at a surgery referral clinic. At that time the dog would occasionally toe-touch the limb during walking,

but would hold the affected limb up most of the time. In addition the leg was painful, and the stifle had only 5 to 10 degrees ROM. The quadriceps muscles were adhered to the fracture callus and the femur. Adhesions and excessive callus were surgically removed to increase the stifle ROM. A bone plate was applied to stabilize the fracture and align the bone. Following surgery maximum flexion of the knee was 85 degrees and maximum extension was 160 degrees.

The day after surgery a nonsteroidal antiinflammatory drug was administered twice daily. ROM and stretching exercises were performed on the hip, stifle, and hock for 20 repetitions, holding each stretch for 10 seconds. These were performed four to five times daily. Following ROM exercises an ice pack was applied to the area for 10 minutes. Two days after surgery stifle extension was 155 degrees and flexion was 70 degrees. Three days after surgery Blackie was discharged from the hospital with instructions to limit activity to leash walks only and to perform ROM exercises four to five times daily. The therapy sessions were to be preceded by hot packs, and cold packs were to be applied after therapy. Following suture removal, swimming was recommended. The owner was warned that Blackie might never regain full function of the limb because of the stiff stifle.

For 2 months the owner continued performing the ROM exercises and walking. In addition, following suture removal the owner would allow the dog to swim in a river daily. The length of time was gradually increased so that he was swimming for 30 minutes daily by 1 month after surgery.

One month after surgery Blackie returned for a recheck examination. It was noted that the stifle had improved ROM. The thigh muscles were increasing in size, and the dog was using the leg quite well, although there was still an abnormal gait at faster speeds. Radiographically, there was continued but incomplete healing of the fracture site. Blackie was discharged with instructions to continue physical rehabilitation and increase the length of leash walks.

The swimming activity was continued, and the dog was exercised up and down hills by the owner. This activity was continued daily with the amount of hill activity increasing to 1 mile by 8 weeks after surgery.

Twelve weeks after surgery Blackie returned for a recheck examination. At this time Blackie had full flexion (35 degrees) and extension (170 degrees) of the stifle. There was a slight limp at a faster gait, but there were no signs of pain and there was increased musculature of the left rear limb. The fracture had reached union, and return to full activity was recommended.

Although several exercises were used in the management of this case, the early swimming activity was likely instrumental in the recovery of the dog. Because swimming was allowed very soon after surgery the dog was able to actively flex and extend the stifle joint and contract and relax muscle groups with minimal weight-bearing activity. The gradual reintroduction of weight-bearing activity as the fracture site became more stable enhanced rehabilitation. In this case the rehabilitation was primarily performed by the owner after careful instruction, and with minimal facilities.

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SECTION VI

Physical Therapy for Specific Diagnoses

32 Common Orthopedic Conditions and Their Physical Rehabilitation

Jacqueline R. Davidson and Sharon Kerwin

Shoulder Conditions

Articular Fractures of the Shoulder

It is critical that articular fractures be treated surgically with anatomic reduction and rigid stabilization of the articular surfaces. This is generally achieved using implants such as screws, wires, pins, or small plates. Nonsteroidal antiinflammatory drugs (NSAIDs), other analgesics, passive range of motion (PROM) exercises, and short leash walks are used for the first few weeks after surgery. The goal of the early postoperative period is to maintain and improve joint range of motion (ROM), limit periarticular fibrous tissue, and allow time for adequate bone healing to support more vigorous active weight-bearing exercises. After adequate healing has occurred, physical rehabilitation consists of continued ROM exercises with stretching if needed, and active weight-bearing exercises such as progressive leash walking and aquatic therapy (Table 32-1).

Avulsion of the supraglenoid tubercle is an injury that occurs in immature dogs and results in displacement of the tubercle. This injury may be classified as a traction physeal injury. Affected dogs have an acute non–weight-bearing lameness and pain on palpation of the distal scapula. Surgical repair of this injury involves reduction and stabilization of the supraglenoid tubercle with pins, wires, or screws. Alternatively the fragment may be removed and the biceps tendon left unattached or released. Physical rehabilitation includes NSAIDs, other analgesics, cryotherapy, leash walks, and PROM for the first 3 weeks. After this initial healing period, limb strengthening, especially of the biceps brachii and brachialis muscles, is begun.

Scapular fractures may occur in any breed of dog as a result of trauma. If the neck of the scapula or articular surface of the glenoid is involved, open reduction and internal fixation are usually indicated. Alternatively many nondisplaced or comminuted scapular body fractures are best treated conservatively. In either case it is important to maintain or regain normal scapular motion in all three planes. Postoperative physical rehabilitation includes NSAIDs, cryotherapy, and PROM exercises to the glenohumeral joint for the first several weeks. As healing progresses scapular PROM is performed in increasing amounts in all planes to prevent fibrosis and decreased ROM. Superficial heat applied with hot packs may be used before exercise to allow increased tissue extensibility, and cross-fiber frictional massage may help to prevent or break down adhesions. Joint mobilization using scapular glides is also effective for increasing ROM. Aquatic therapy (underwater treadmill [UWTM] and swimming), and gait activities such as treadmill walking may also be incorporated to regain muscle strength.

Both conservatively managed and surgically operated articular shoulder fractures are often supported in non–weight-bearing slings postoperatively for several weeks. The small pieces of bone involved combined with a potentially large amount of force normally exerted on the shoulder joint predispose the animal to failure of the repair unless the repair is supported until healing has progressed. Unfortunately, unwanted side effects of non–weight-bearing slings (carpal flexion, Spica, or Velpeau bandages), include loss of nutrition to cartilage, joint stiffness, loss of range of motion, and muscle atrophy. Massage, PROM, and measurement of flexion/extension angles should be performed during each bandage change, and owners warned that several months of rehabilitation may be required after the bandage is removed to re-establish function. Commercially available, removable slings may be more ideal, allowing for some motion without weight bearing and in some cases, removal to allow intermittent, controlled weight-bearing, or rehabilitation exercises on a daily basis.

Osteochondritis Dissecans of the Shoulder

Osteochondrosis is a developmental disease affecting the growth plates and joint cartilage in medium and large breed dogs. Abnormal endochondral ossification of the

Table 32-1 Rehabilitation After Surgical Repair of Articular Fracture

Goals	Treatment
Control inflammation	Cryotherapy immediately after surgery and then daily until inflammation resolves, particularly after exercise
Maintain range of motion and joint health	Passive range of motion and/or joint mobilizations for 3 to 4 weeks. Consider aquatic walking to minimize joint stress.
	Add cavaletti walking after several weeks, depending on stability of repair.
Control pain	Nonsteroidal antiinflammatories plus other analgesics if needed. ± Transcutaneous electrical nerve stimulation
Strengthen muscle	Begin with controlled slow leash walking. Amount of activity depends on stability of repair and stage of healing. Short, slow leash walks for at least the first few weeks.

deep layers of articular cartilage results in focal areas of thickened cartilage that are prone to injury. Normal activity may cause enough stress to create a fissure in the articular cartilage. Contact of the synovial fluid with the deep layers of degenerating cartilage may cause synovitis. In the absence of further stress, the lesion may heal. However, further stress on the cartilage may result in formation of a cartilage flap. This condition is termed *osteochondritis dissecans* (OCD). Osteochondritis dissecans has been described in various joints of the dog, but occurs most commonly in the caudal aspect of the humeral head. The etiology is multifactorial in all species. Risk factors in dogs include young age, large and giant breeds, rapid growth (high calorie diet), and excess calcium consumption.[1]

Dogs are typically presented with mild to moderate weight-bearing lameness between 4 and 8 months of age. Many dogs have bilateral lesions, although one side may be clinically more severe. Muscle atrophy may be apparent in the affected limb if the dog has had a chronic lameness. Pain may be elicited on extreme flexion or extension of the shoulder. Radiographs may demonstrate a defect in the subchondral bone under the cartilage flap on the caudal aspect of the humeral head (Figure 32-1). Uncommonly, advanced imaging or arthroscopy is needed to confirm the diagnosis.[2]

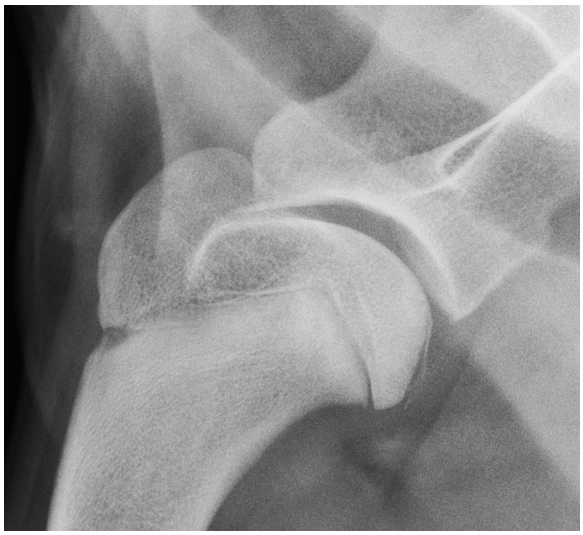


Figure 32-1 Osteochondritis dissecans of the caudal aspect of the head of the humerus. The right caudal humeral head is flattened and the linear density adjacent to the caudal humeral head is consistent with an osteochondral flap.

If shoulder osteochondrosis is diagnosed early, decreased activity may prevent cartilage flap formation. Cryotherapy and NSAIDs may be used to limit inflammation. Use of disease-modifying osteoarthritis agents (DMOAs), such as glucosamine and chondroitin sulfate, may support cartilage health. Passive range of motion exercises help to maintain joint range of motion and provide joint nutrition and stress along normal lines of movement.[3] Once a flap has formed it will not heal and should be removed. If the dog is more than 7 months of age or the lameness has persisted more than 4 to 6 weeks, surgery is indicated.[4]

Surgery is generally the treatment of choice in dogs with clinical signs. It is performed via arthrotomy or arthroscopy to remove the defective cartilage and forage or curettage the bed of the lesion (Figure 32-2). This encourages vascular ingrowth and healing by the formation of fibrocartilage. After surgery NSAIDs, cryotherapy, PROM, and controlled leash walks are instituted for the first 2 to 4 weeks (Table 32-2). After this initial healing period the duration of the leash walks is progressively intensified. In addition treadmill walking, UWTM exercise, and swimming may be initiated. By 6 weeks, light jogging usually can be started.[3] Seroma formation is a common complication after shoulder surgery by arthrotomy, but is usually self-limiting and resolves with rest. If a shoulder seroma is observed, the activity level should be reduced until it resolves. The value of treating a seroma with hot packs or cold packs has not been documented, but is commonly recommended. Long-term management of OCD is focused on limiting or treating osteoarthritis (OA). The long-term prognosis for shoulder OCD is excellent in most cases.[2,5]

Bicipital Tenosynovitis

Bicipital tenosynovitis is a degenerative or failed healing response of the tendon and tendon sheath of the biceps brachii muscle.[6,7] Inflammation is not consistently present. Tendonitis implies the presence of inflammation in the tendon and musculotendinous junction, and may not be accurate for many cases of tendon pain.[8,9] Tendinopathy is an inclusive term used to describe tendon pathologies such as tendonitis, tendinosis, or paratenonitis. Tendon injuries may be caused by acute trauma or repetitive loading. Repetitive tendon overload tends to respond with tendon sheath inflammation, tendon degeneration, or both. There is some evidence that acute tendinitis is quickly replaced by tendon degeneration.[10] Tendon pain should not be assumed to be caused by inflammation, but can occur with tendinosis as well.

Bicipital tenosynovitis is commonly seen in middle- to older-age large-breed dogs. Affected dogs usually have a weight-bearing forelimb lameness that is insidious in onset, progressive or intermittent, and exacerbated by exercise. Pain is not a consistent finding, but may be elicited on direct palpation of the bicipital groove on the proximal craniomedial aspect of the humerus. The pain response may be increased by flexing the shoulder with simultaneous elbow extension, which increases biceps tendon tension. Bicipital tenosynovitis may occur in conjunction with OA of the shoulder or glenohumeral ligament damage. Atrophy of the supraspinatus and infraspinatus muscles may be palpable, particularly in chronic cases. Mineralization of the biceps tendon, osteophytes in the intertubercular groove, or other degenerative changes may be seen on radiographs. Diagnosis can be difficult and is sometimes made by diagnostic ultrasound, magnetic resonance imaging (MRI), or observation during arthroscopy.

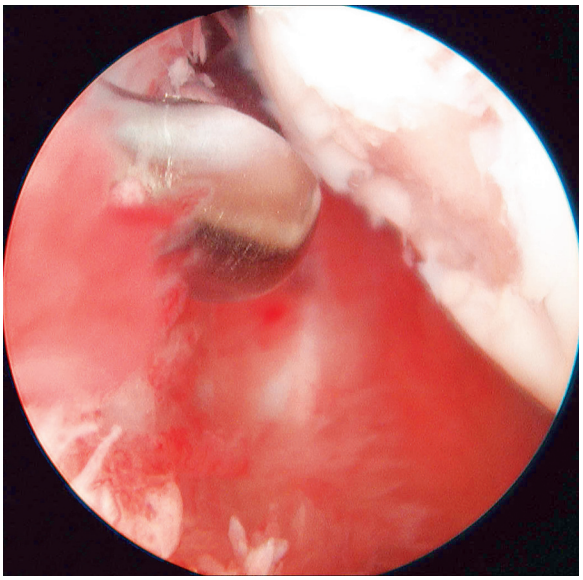


Figure 32-2 Arthroscopic view of osteochondral defect on the caudal humeral head. Subchondral bone is visible in the center of the lesion on the right of the image. The metallic object is a bone curette.

Conservative management of tendinopathies or tendinitis in humans primarily consists of rest, icing, and eccentric strengthening exercises. There are little data to support the use of therapeutic ultrasound, shock wave therapy, orthotics or massage,[8] but these are used in some cases. Analgesics are indicated, but not necessarily NSAIDs or corticosteroids. Deep transverse friction massage has been advocated as part of the treatment for tendinopathy pain. Studies of the effectiveness of deep transverse friction massage for the treatment of tendinopathies in humans are inconclusive.[11] Common recommendations for the conservative management of tendinopathies in dogs include 4 to 12 weeks of rest and NSAIDs as needed (Table 32-3). As an alternative to oral NSAID therapy, intraarticular corticosteroids may be administered because the shoulder joint communicates with the biceps tendon sheath. Pulsed-mode 3-MHz therapeutic ultrasound is advocated over the tendon and musculotendinous junction to promote tissue healing. Because of the close proximity to bone, the dog must be monitored for any signs of pain, and the ultrasound intensity adjusted accordingly. Pulsed-mode ultrasound should not result in significant tissue temperature increase. Laser therapy may also be used to decrease pain and aid healing. Cryotherapy may be prescribed to reduce inflammation and pain. Response to conservative therapy varies, and may

Table 32-2 Joint Rehabilitation After Osteochondritis Dissecans Surgery

Goals	Treatment
Control inflammation	Cryotherapy immediately after surgery and then daily until inflammation resolves, particularly after exercise
Maintain range of motion and joint health	Passive range of motion for 1 to 2 weeks
Control pain	Nonsteroidal antiinflammatories for first few weeks, as needed
Strengthen muscles	Begin with controlled slow leash walking and progress over 2 to 4 weeks. Decrease activity if seroma or lameness develops. May begin jogging or swimming in 6 weeks.

Table 32-3 Rehabilitation for Tendinopathies

Goals	Treatment
Control inflammation	Cryotherapy immediately after surgery and then daily until inflammation resolves, particularly after exercise
Promote tissue healing	Limited activity ± pulsed ultrasound ± cross-fiber frictional massage
Control pain	Analgesics ± transcutaneous electrical nerve stimulation ± laser
Strengthen soft tissues	May need several weeks of rest after acute injuries or surgery. Gradual progression of strength exercises, starting with eccentric exercises for involved muscles, such as walking downhill.
Maintain joint range of motion	Joint mobilizations or passive range of motion

be related to the inciting cause of the tendinopathy.[12,13] Full recovery can be expected to require 3 to 6 months of therapy.[8]

Surgical treatment is recommended for dogs that do not respond to medical treatment or have significant tears of the tendon. Surgical techniques include tenodesis or tendon release, both of which have been described as arthroscopic or open procedures.[12,14-16] Cryotherapy, PROM, and short leash walks (preferably with a harness that does not rub over the surgical site) are used for the first 3 postoperative weeks to promote normal joint movement while the tendon has minimal strength. Joint mobilizations may also help increase joint ROM. Exercises to gradually increase limb strength and promote remodeling of the peritendinous scar are performed after 3 weeks. Exercises should emphasize strengthening of the brachialis muscle, to improve the dog's ability to flex the elbow joint. Shake hands exercises with limb weights that are gradually increased, as well as walking over cavaletti rails with limb weights, are specific exercises to increase the strength of elbow flexor muscles. Aquatic therapy and treadmill exercises may be beneficial. Superficial heat therapy may be used before exercise to facilitate tissue extensibility. Neuromuscular electrical stimulation may be applied to the brachialis muscle. Cross-fiber frictional massage can be used to prevent or diminish adhesions. Cryotherapy may help to minimize postexercise inflammation. Most surgically treated dogs regain near normal function and gait.[12,13]

Supraspinatus Tendinopathy

Supraspinatus tendinopathy may cause mild to moderate lameness in medium and large breed dogs. The etiology of supraspinatus tendinopathy is unknown, but it is speculated to be caused by overuse and repetitive trauma.[17] Affected dogs typically have chronic, intermittent, unilateral lameness. Palpation of the greater tubercle, near the tendon insertion, is usually painful. Mineralization may be observed on radiographs, and may contribute to bicipital tenosynovitis or impingement of the biceps tendon. However, the presence of mineralization can also be asymptomatic and may be an incidental finding. Diagnostic ultrasound or MRI may be needed to identify non-mineralized tendinopathy. Medical treatment includes rest, PROM, NSAIDs, local steroid injection, heat or cold therapy, deep massage therapy, and therapeutic ultrasound. Response to these treatments is limited. However, pulsed therapeutic ultrasound resulted in some clinical improvement in one study of calcific tendonitis in humans.[18] Extracorporeal shock wave therapy may decrease the size of the mineralized region in dogs.[19] If medical treatment is inadequate, surgical excision of the mineralized tissue is recommended.[17,20-22] After surgery, a carpal flexion bandage may be applied for 2 weeks to prevent weight bearing. Activity is limited for an additional 2 to 3 weeks. Swimming may cause too much tendon stress and is not advised for several months. The prognosis varies with the severity of the mineralization.[17] Most dogs will be sound within 3 to 4 weeks. Lameness may recur with persistent bicipital tenosynovitis or recurrence of supraspinatus mineralization.

Medial Shoulder Instability

Medial glenohumeral instability is thought to result from chronic repetitive activity or overuse, such as jumping. Dogs typically develop a chronic, intermittent weight-bearing lameness that is nonresponsive to treatment with NSAIDs. Instability may be assessed subjectively by palpating for increased translocation of the humerus.[23] Medial instability can be confirmed by goniometric measurements of shoulder abduction,[24] although the clinician should consider the role of pain-related muscle atrophy as a potential cause for increased shoulder abduction as opposed to true instability of the shoulder. The normal abduction angle in a large breed dog is approximately 30 degrees.[24] The affected shoulder will have a much larger abduction angle as compared with the normal shoulder.[24] The degree of instability may be characterized as mild, moderate, or severe.[25] Mild instability is associated with minimal pathologic changes and abduction angles of 35 to 45 degrees. Dogs with moderate instability have abduction angles of 45 to 65 degrees and more advanced pathology is seen arthroscopically. Dogs with severe instability typically have shoulder abduction angles greater than 65

degrees. Severe disruption of the medial glenohumeral ligament, subscapularis tendon, and joint capsule are seen arthroscopically.[25]

Conservative treatment with the shoulder supported and restricted by shoulder hobbles can be used in mild cases. Extracorporeal shockwave treatment to the medial shoulder structures may be beneficial. As healing progresses, strengthening of the medial supporting muscles may be achieved by walking on a treadmill with lateral tension supplied by an elastic band during the swing phase of gait. As strength improves progressively stiffer elastic bands may be used. Dogs with moderate shoulder instability are treated arthroscopically with imbrication, thermal capsulorrhaphy, reconstruction of the medial glenohumeral ligament, or transposition of the biceps tendon.[26,27] After surgery a shoulder support wrap with hobbles is applied to protect the surgical repair. Cryotherapy, PROM, and strictly controlled activity are instituted for the first 3 postoperative weeks.[25] After 3 weeks weight-bearing exercises are gradually introduced. Function improves within three to four months, and optimal function is not attained until 5 to 6 months following thermal capsulorrhaphy.[26] Severe shoulder instability is usually caused by trauma and is treated as for acute shoulder luxation.

Shoulder Luxation

Shoulder luxation is usually a result of trauma, but may also be seen with congenital joint malformation. Shoulder luxation may be apparent on physical examination, but radiographs are recommended to evaluate for concomitant fractures or congenital bony malformations. If it is a recent injury with no evidence of fracture or malformations, closed reduction and immobilization in a sling for 1 to 3 weeks may be successful. Medial shoulder luxations are immobilized with the limb flexed in a Velpeau sling, whereas lateral luxations are immobilized with a spica splint. After the sling is removed, gentle PROM is initiated. Motion is limited to the sagittal plane to avoid excess stress on the affected joint capsule and ligament. Weight-bearing exercises are also limited.

If conservative management is unsuccessful or fractures are present, surgical stabilization is indicated. Surgical techniques for medial shoulder luxation include imbrication or reconstruction of the medial glenohumeral ligament and joint capsule, or transposition of the biceps tendon to a more medial position to assist with joint stability. Postoperatively treatment with cryotherapy and NSAIDs can be initiated to minimize inflammation, and the shoulder may be placed in a Velpeau sling for 2 weeks. After the Velpeau sling is removed the front limbs may be placed in hobbles to prevent abduction of the limb, but ROM must be restored. Passive ROM exercises are performed, but are limited to the sagittal plane (flexion and extension) to avoid stressing the medial or lateral joint capsule. Exercise is initially restricted, with gradual progression of weight-bearing exercises. Full recovery may require 1 to 3 months.

The prognosis is generally good, although OA may develop. In the case of severe congenital malformations or irreparable joint fractures, a salvage procedure may be indicated. Salvage procedures of the shoulder include excision arthroplasty (glenoid excision), arthrodesis, or amputation.

Fibrotic Contracture of the Infraspinatus Muscle

Contracture is a shortening of the tendon-muscle unit that is not caused by active muscle contraction. Most or all of the muscle and/or associated tendon is replaced by fibrous tissue. Most cases of contracture have a history of acute injury with lameness and inflammation that subsides with supportive care. Weeks to months later the animal is presented with clinical signs, which may include pain, weakness, firmness within the muscle, and an abnormal posture and gait.[28] Proposed causes include infection, trauma, repetitive strains, fractures, infectious diseases, immune-mediated diseases, ischemia, and eosinophilic myositis. Muscle contracture leads to permanent shortening of the affected muscle and can limit ROM of the associated joint. Muscle contracture can occur in any muscle, but has been reported in the infraspinatus, supraspinatus, teres minor, brachialis, quadriceps, semitendinosus, gracilis, and sartorius.[28] If the contracture is not severe, splinting with the muscle and tendon in tension and stretching activities may lengthen the tendon-muscle unit. In many cases, surgery is recommended to lengthen or transect the tendon.

Contracture in the front limb has been most commonly reported in the infraspinatus muscle. Infraspinatus contracture causes a mild weight-bearing lameness mainly in hunting or working dogs. The cause is hypothesized to be acute muscle trauma, which results in incomplete rupture of the infraspinatus muscle. The injury leads to inflammation and subsequent compartment syndrome.[28-30] Replacement of muscle fibers by fibrous tissue occurs over days to weeks. The supraspinatus and deltoid muscles also may be involved and undergo atrophy or contracture.[31] The humerus is held in adduction, whereas the antebrachium is externally rotated with the foot abducted. The scapulohumeral joint cannot fully extend. There is a weight-bearing lameness in which the limb circumducts as it is advanced during the stride and the paw flips into extension before foot placement.

Physical rehabilitation may be beneficial if the condition is diagnosed early. Continuous mode therapeutic ultrasound with stretching exercises may help to lengthen contracted tissues, but the degree of contracture is usually so severe at the time of diagnosis that it is difficult to improve the condition.[3] When the dog is presented with contracture, the treatment of choice is surgical transection of the infraspinatus tendon and associated fibrous tissue.[31]

This immediately restores motion of the shoulder joint and the prognosis is good.

Postoperatively full weight bearing is allowed, but activity should be restricted for the first few weeks. Uncontrolled activity may cause tissue damage and recurrence of fibrous tissue.[3] Passive ROM exercises to the forelimb joints several times daily maintain joint ROM and promote normal alignment of the healing tissues.[3] Cryotherapy may be used to reduce inflammation after exercise. In cases in which there is significant disuse atrophy of the forelimb muscles, general conditioning exercises for the limb are used to gradually return the muscle to normal size and strength. Conditioning exercises include walking, jogging, and aquatic therapy. Neuromuscular electrical stimulation (NMES) may be used if the atrophy is severe and the dog is too weak to use the leg well.

Teres Minor Strain

Teres minor strain has been reported in athletic dogs of various breeds.[32] All dogs in one report had front limb lameness that was improved by rest, but recurred with return to activity. The shoulder was painful on full extension and pain was elicited with direct palpation of the teres minor muscle. Treatment included ultrasound, stretching, balancing exercises, leash walking, gait retraining, and acupuncture point stimulation. All dogs returned to previous levels of activity.[32]

Elbow Conditions

Articular Fractures of the Elbow

Fractures of the articular surface of the distal humerus are common and typically result from trauma. Articular fractures of the proximal radius and ulna are much less common. Salter-Harris type IV fractures of the lateral condyle of the humerus are commonly seen in skeletally immature dogs, and fractures of the lateral humeral condyle are also seen in older dogs. In spaniel breeds, such as Cocker spaniels, it is not uncommon for mature animals to present with articular fractures with minimal evidence or history of trauma (Figure 32-3). These dogs appear to have incomplete ossification of the humeral condyles, although there may be other predisposing factors.[33] As with any articular fracture, decreased ROM and destruction of articular cartilage may be sequelae of an articular fracture. In addition Salter-Harris fractures in immature dogs may have alteration in bone growth that is necessary for a congruent elbow joint. Repair is accomplished with placement of a transcondylar screw with compression of the fracture site and placement of a small antirotational pin driven up the lateral epicondyle (Figure 32-4).

Articular fractures demand both rigid fixation and anatomic reduction to maintain an even, stable cartilage surface. Because of the necessity for anatomic reduction, the surgical approach may be extensive, including tenotomies or osteotomies. The surgeon must carefully balance the degree of soft tissue manipulation with the need for exposure of the fracture to facilitate reduction. Extensive dissection increases postoperative pain and swelling, and may increase the degree of muscle scarring and periarticular fibrosis after surgery. In our experience excessive postoperative scarring and fibrosis leading to decreased range of motion are particularly likely after articular fractures involving the distal humerus or distal femur in the dog and cat. The use of minimally invasive techniques, such as closed reduction and fixation via the use of fluoroscopy, or

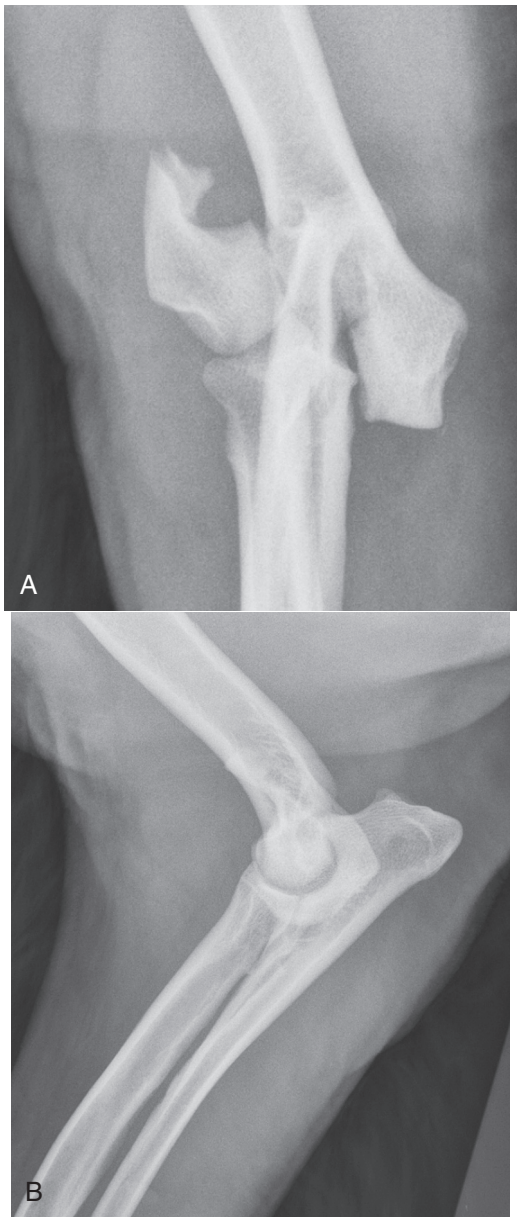


Figure 32-3 Fracture of the lateral condyle of the humerus in a cocker spaniel. **A,** A-P view, **B,** Lateral view.

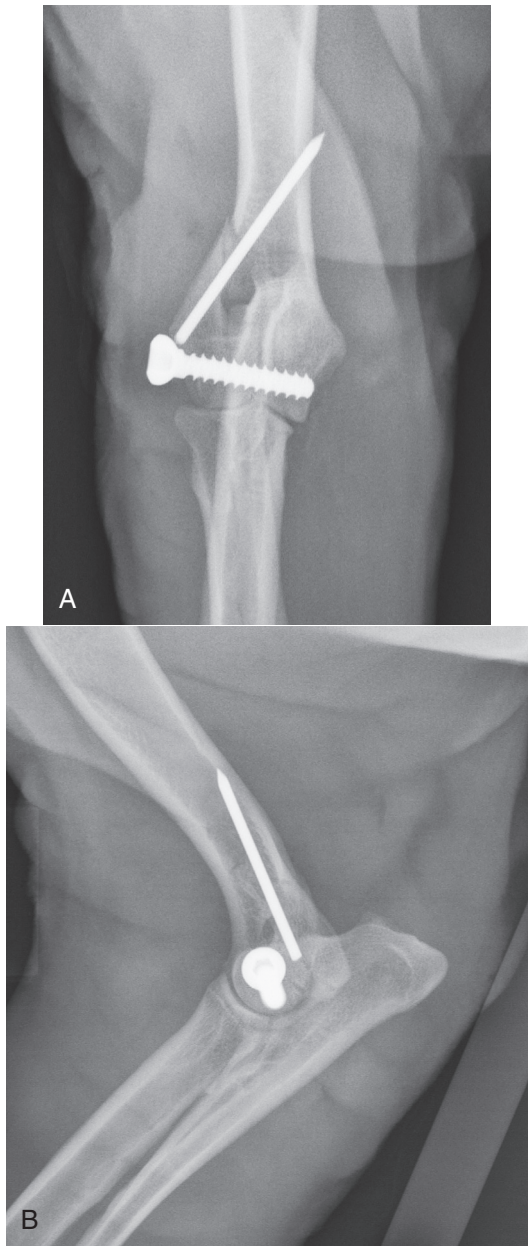


Figure 32-4 Repair of the fracture shown in Figure 32-3 with a transcondylar screw placed in lag fashion to compress the fracture fragments and a small pin in the lateral epicondyle to counteract rotational forces. **A,** A-P view, **B,** Lateral view.

arthroscopic-assisted fracture reduction should be considered (Figure 32-5).[34] Whenever possible, osteotomies rather than tenotomies should be performed to gain access to the fracture because osteotomies heal by reforming normal bone, whereas tenotomies heal via scar tissue. When osteotomies are repaired the surgeon must pay particular attention to pin and wire placement. Inadvertent wire placement into the joint causes mechanical loss of range of motion and pain for the patient, rendering even the most sophisticated rehabilitation strategy useless.

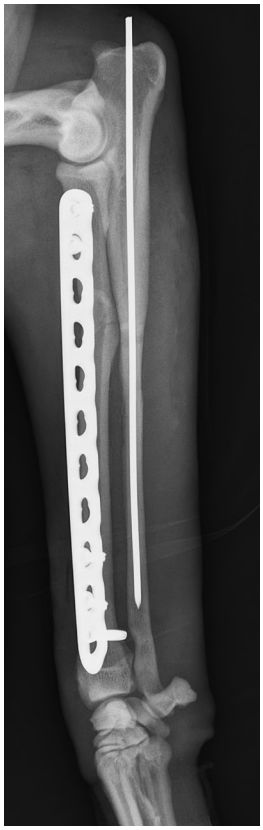


Figure 32-5 Lateral postoperative radiograph of a radial fracture that was plated using a limited approach to minimize disruption of the soft tissues.

In addition to the demand for anatomic reduction, rigid fixation of articular fractures may rely on implants placed across a small epiphyseal segment, resulting in a somewhat tenuous repair, especially in young patients with soft bone. In the case of distal humeral fractures, particularly Salter type IV fractures in young dogs, the surgeon may be tempted to splint the limb or place a carpal flexion bandage to prevent weight bearing. Although these strategies may effectively prevent mechanical overload of the repair during the healing process, they may have the unintended effect of promoting muscle scarring around the operative site as well as cartilage atrophy.[35] Although a carpal flexion bandage still allows some ROM exercises, contracture of muscles around the carpus may lead to longstanding pain, loss of ROM and poor limb use despite good fracture healing. ROM exercises should be performed frequently but are best left to professionals as excessive (or too little) ROM can cause failure of fixation or joint contractures respectively. Weight-bearing exercises should be used with extreme caution, especially during the first 3 weeks after repair to avoid fixation failure. Short leash walks may be permitted, but it is important to properly confine the patient to avoid catastrophic failure, especially if the patient appears to be doing very well.



Figure 32-6 Therapy ball may be used to encourage elbow flexion.

Cryotherapy, analgesic medication, massage, and passive ROM may be used for articular fractures of the elbow (see Table 32-1). In small breed dogs use of a therapy ball may be helpful to encourage limb use and flexion of the elbow without direct manipulation of the limb (Figure 32-6). It is imperative that ROM exercises continue for 3 to 4 weeks after surgery to be certain that ROM is maintained. Normal ROM may return 10 to 14 days after surgery, but fibrous tissue and other healing periarticular tissues are not yet mature. If ROM exercises are discontinued too soon, it is possible that some maturation and contracture of tissues may occur with some loss of normal joint motion. These fractures can present a very difficult challenge to the surgeon and the rehabilitation practitioner. Although overuse may result in catastrophic failure of the repair, failure to perform appropriate ROM exercises may lead to crippling loss of ROM.

An even more challenging fracture to repair and rehabilitate is a bicondylar (sometimes referred to as a T or Y) fracture, in which both the medial and lateral condyles of the humerus are fractured from the main shaft of the humerus and from each other. It is fairly common for several additional fragments to be separated from the shaft of the humerus. Repair involves stabilization and fixation of the condyles, and then stabilizing them to the main shaft of the humerus with plates, intramedullary pins, or an external fixator. In many instances, an osteotomy of the olecranon is necessary to repair this type of fracture. The olecranon osteotomy is then repaired with a pin and tension band or screw technique. Decreased ROM is very common, especially after an olecranon osteotomy, along with OA development after surgery. Newer techniques that use separate medial and lateral approaches to the joint to avoid an olecranon osteotomy seem to result in maintenance of greater ROM. It is critical that rehabilitation be performed following repair of bicondylar fractures of the humerus. Particular attention is paid to achieving as complete ROM as possible, using passive and active ROM exercises. Control of periarticular swelling and edema with cryotherapy, NSAIDs, and bandages helps to improve motion and reduce postoperative pain. Swimming or walking in an underwater treadmill has the additional benefit of limb usage in a buoyant environment. As weight-bearing improves, other activities, such as walking over cavaletti rails may be initiated. Daily rehabilitation for at least 2 to 3 weeks is recommended.

Avulsion fractures of the olecranon may also occur. In immature dogs, there may be an avulsion fracture of the olecranon with separation across the physis. Olecranon fractures prevent extension of the elbow, which is necessary for normal weight bearing and ambulation. This condition is treated by surgically replacing and stabilizing the avulsed fragment with a screw or pin and tension band technique, or if the fragment is large enough, a bone plate. Fracture through the articular surface of the ulna may also occur, necessitating excellent anatomic reduction and rigid internal fixation, generally with a bone plate and screws. Physical rehabilitation is important to achieve optimal elbow extension. Treatments in the first 3 weeks may include gentle PROM, NSAIDs, and cryotherapy. Following adequate healing more vigorous ROM and stretching exercises may be initiated, along with active exercises to rebuild atrophied muscles.

Elbow Dysplasia

Elbow dysplasia includes fragmented medial coronoid process (FCP), osteochondritis dissecans (OCD), and ununited anconeal process (UAP). Dogs with elbow dysplasia usually have only one of the three conditions. Elbow incongruity may also be present. The pathogenesis is poorly understood. Factors that have been implicated include genetics, improper nutrition, and trauma. Other proposed mechanisms are osteochondrosis, trochlear notch dysplasia, and asynchronous growth of the radius and ulna.[36] Elbow dysplasia usually occurs in large or giant breeds of dogs. Affected dogs typically have bilateral problems, although one elbow may be more severely affected.

Fragmented Medial Coronoid Process of the Ulna

Dogs with FCP are presented with a mild to moderate weight-bearing lameness, which is usually noted between 4 and 7 months of age.[37] Physical exam findings include pain on flexion and extension of the elbow, pain on palpation of the medial aspect of the joint, and palpable joint effusion. In dogs older than 11 months, crepitus, decreased ROM, and general joint thickening may be evident. Radiographs often show degenerative changes in the joint, but the actual lesion may not be seen (Figure 32-7). In many

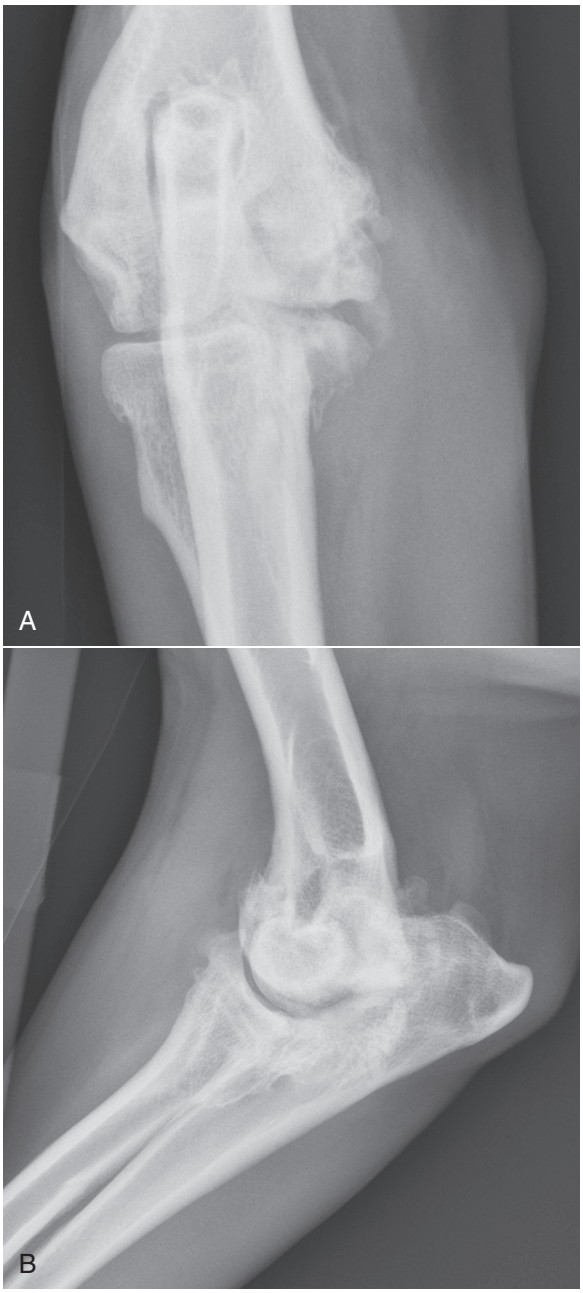


Figure 32-7 Severe elbow osteoarthritis in a middle-aged dog suspected to be secondary to a previously unrecognized fragmented medial coronoid process. **A,** A-P view, **B,** Lateral view.

cases, computed tomography or arthroscopy is needed to make a definitive diagnosis.

Treatment is removal of the abnormal process via arthrotomy or arthroscopy, with possible subtotal coronoidectomy or ulnar osteotomy in some cases. Postoperatively activity is limited for 2 to 4 weeks. The prognosis is good if FCP is removed before there is advanced OA. Osteoarthritis will progress regardless of treatment, but changes are thought to be more severe in untreated cases. If the OA is severe at the time of diagnosis, the value of surgical treatment must be questioned. These dogs may be better managed by conservative treatment for the OA.[37] In all cases rehabilitation is directed toward preventing, slowing, or treating OA to maintain an acceptable quality of life. Therapy includes maintaining an appropriate body weight, controlled low-impact exercise, aquatic exercise, monitoring pain, NSAIDs, and chondroprotective agents. See section on OA for guidelines on rehabilitation.

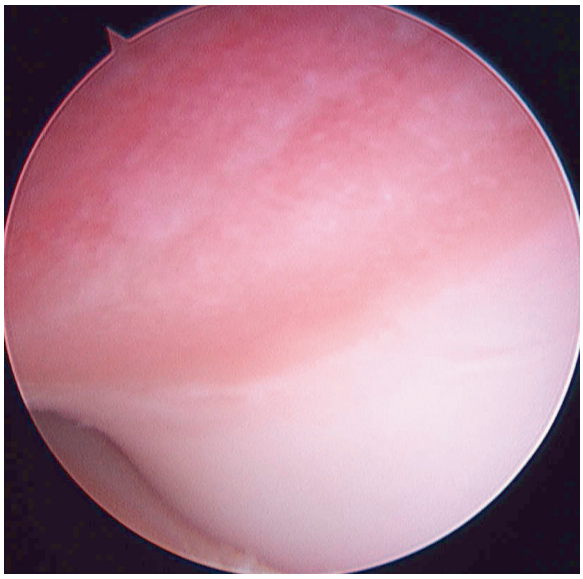


Figure 32-8 Arthroscopic view of an erosion on the humeral articular cartilage that was created by a fragmented medial coronoid process. This is known as a kissing lesion and may be confused with osteochondritis.

Osteochondritis Dissecans of the Elbow

Dogs with elbow OCD are typically presented with mild to moderate lameness between 4 and 9 months of age. Many dogs have bilateral lesions, although one side may be clinically more severe. Atrophy of the muscles may be apparent in the affected limb if the dog has been lame for several weeks. Pain may be elicited on extreme flexion or extension of the affected joint. Chronic elbow OCD may also have signs of OA such as joint effusion, thickening of the periarticular soft tissues, decreased ROM, and crepitus. Radiographs may demonstrate a defect in the subchondral bone under the cartilage flap on the medial humeral condyle. Fragmented medial coronoid process can create an erosive lesion on the medial humeral condyle, called a *kissing lesion*, which can look very similar to OCD at the time of surgery (Figure 32-8). In some cases especially with elbow OCD, the diagnosis may be difficult and is best made by computed tomography or arthroscopy.

Surgery is usually the treatment of choice in dogs with clinical signs. It is performed via arthrotomy or arthroscopy to remove the defective cartilage and forage or curettage of the lesion. After surgery NSAIDs, cryotherapy,



Figure 32-9 Ununited anconeal process. An irregular radiolucent line is visible between the olecranon and the anconeal process on a flexed, lateral radiograph of the elbow, indicating that the anconeal process has not fused with the ulna.

PROM, and controlled leash walks are instituted for the first few weeks (see Table 32-2). After the initial healing period, the duration of the leash walks are progressively intensified. Long-term management of OCD is focused on limiting or treating OA. See section on OA for guidelines on rehabilitation. The prognosis for elbow OCD is good if surgery is performed before OA is advanced, but it is more guarded than for OCD of the shoulder.

Ununited Anconeal Process

Ununited anconeal process (UAP) is a failure of the anconeal process to fuse with the olecranon by 5 months of age, and is apparent on a flexed lateral radiograph (Figure 32-9).[37] Instability of the anconeal process causes inflammation and eventual OA. The dog usually is presented with a weight-bearing lameness. Decreased range of motion and joint effusion of the caudolateral aspect of the elbow may be apparent on palpation

Treatment options include surgical removal of the anconeal process, screw fixation combined with osteotomy of the proximal ulna, or osteotomy of the proximal ulna alone to relieve joint incongruity and allow healing of the anconeal process. Regardless of the treatment, OA progresses, therefore ongoing treatment is directed at managing OA. (See the section on OA for guidelines on rehabilitation.)

If the UAP is treated by screw fixation, the rehabilitation program should have a slower progression of weight-bearing activities until there is radiographic evidence of healing. This may take as long as 12 weeks in some cases. If the UAP was treated by removal, the rehabilitation program may progress more rapidly, as dictated by the degree of joint effusion, pain, ROM, and weight bearing. Immediately after surgery PROM, aquatic therapy, and light leash walks are recommended. Cryotherapy and NSAIDs may be used as needed to control inflammation and pain. Aquatic activity can be very beneficial because of the active ROM while weight bearing in a buoyant environment.[38,39]

Elbow Incongruity

Asynchronous growth of the radius and ulna can cause elbow incongruity in chondrodystrophoid dogs, and is usually evident by 4 to 5 months of age. Ununited anconeal process and FCP have been associated with elbow incongruity, although they can occur in the absence of incongruity. Traumatic closure of a radial or ulnar physis also leads to asynchronous growth with elbow subluxation and angular limb deformity.

Surgical options to improve congruity include corrective osteotomy or ostectomy of the radius or ulna, and may involve stabilization with a bone plate or external fixator. The prognosis is worse if the incongruity is severe and the animal is older (>9 months of age).[40]

After surgery normal ROM of the joints proximal and distal to the repair should be maintained. Pain and edema may be controlled by NSAIDs and cryotherapy. If the goal was to lengthen the limb following surgery, increased tension on the soft tissues may result in decreased ROM, joint stiffness, and lameness. In these cases therapeutic ultrasound with simultaneous stretching and ROM activities may be helpful. As bone healing progresses more aggressive weight-bearing exercises may be initiated.

Elbow incongruity invariably results in some degree of OA of the elbow. Some dogs may not present for rehabilitation until they have end-stage OA. In either case rehabilitation is directed at managing OA so as to maintain an acceptable quality of life. (See the section on OA for guidelines on rehabilitation.) Conservative management of OA includes NSAIDs, chondroprotective agents, cryotherapy, PROM, and physical rehabilitation. Dogs with end-stage OA often have chronic synovial effusion, reduced ROM, crepitus of the joint, pain on manipulation of the elbow, muscle atrophy, and lameness. Physical rehabilitation cannot correct the underlying problem, but efforts to improve joint health, increase ROM, reduce weight, and rebuild atrophied muscles may significantly improve the patient's quality of life. Physical rehabilitation considerations would include PROM and stretching, active ROM activities, redeveloping muscle strength with exercise or neuromuscular electrical stimulation, low-impact exercises, laser, extracorporeal shock wave, and aquatic therapy. Careful monitoring of ROM is important with a home program to avoid permanent losses in ROM.

Elbow Luxation

Elbow joint luxations are generally a result of trauma, although congenital malformations of the elbow can predispose to luxation. Traumatic elbow luxations are usually lateral and can be diagnosed by palpating the radial head laterally displaced relative to the humeral condyle. Radiographs are recommended to assess for concomitant fractures and for congenital malformations. Radiographic findings may affect the treatment choice and the prognosis.

If there are no congenital malformations or concurrent fractures, closed reduction of the elbow is usually successful because the joint is inherently quite stable, despite significant collateral ligament damage. Open reduction and surgical stabilization are indicated if closed reduction is not achieved, closed reduction fails, or congenital abnormalities or articular fractures are present. Surgical repair often involves direct repair of the collateral ligaments or replacement of the collateral ligaments with a suture prosthesis (e.g., heavy suture secured by bone anchors or screws). After closed or open reduction, joint healing is protected by maintaining the elbow in an extended position. This is accomplished by placing the limb in a spica splint for 1 to 4 weeks.

Regardless of whether closed or open reduction is performed, rehabilitation is begun after the spica splint is removed. Range of motion must be restored without causing the joint to reluxate. Cryotherapy and NSAIDs are used to minimize inflammation in the early postoperative period. After elbow luxation, passive and active ROM exercises should be limited to the sagittal plane, and internal, external, varus or valgus stresses should be minimized. Activity is gradually increased, but aggressive activities such as jogging, swimming, or uncontrolled play are limited until the joint has healed. This may take 1 to 3 months, depending on the degree of tissue damage. The prognosis for traumatic joint luxations is generally good, although OA is a common sequela.

Joint reduction and preservation of the joint may not be recommended if there are irreparable fractures involving the joint or severe congenital malformations. In these cases a joint salvage procedure, such as elbow arthrodesis, may be a better choice. Total elbow replacement surgery is relatively new in dogs and is not commonly performed, but may result in better function.[41] Limb amputation may be elected in some cases.

Antebrachial Conditions

Fractures of the Antebrachium

Fractures of the radius and ulna commonly occur. These injuries may be treated by closed reduction and immobilization in a cast in cases of simple fractures, or they may be repaired by open reduction and fixation with wires, plates, screws, or external skeletal fixation. In small dogs distal radius and ulna fractures have a high incidence of delayed or nonunion healing when treated with external coaptation (Figure 32-10). For optimal healing these fractures require anatomic reduction and rigid surgical fixation, while using appropriately sized implants to reduce the risk of stress protection. In addition to problems with bone healing, prolonged immobilization by splinting or casting leads to disuse atrophy of bone, cartilage, ligaments, tendons, and muscle in the affected limb. In some cases there is frank loss of bone distally in response to disuse of the leg. Following the first few weeks of physical rehabilitation, which involves reduction of pain and swelling, rehabilitation efforts can be undertaken to increase weight-bearing activities to load the bone during fracture healing and help stimulate normal healing. One of the main impediments to limb use is decreased ROM of the carpus as a result of flexural contracture of the soft tissues. Assisted extension of the carpus to a normal position is often painful and often results in a non–weight-bearing lameness, despite healing of the fracture. If a coaptation device is used, it is essential that particular attention be paid to stretching and ROM exercises of the carpus and digits. In some cases, an extension splint molded to the normal contralateral side may be placed on the affected limb, applying some tension to position the digits and metacarpals more normally in between rehabilitation sessions. It may take 10 to 14 days to achieve a normal angle of the carpus for weight bearing.



Figure 32-10 **A,** Distal radial fractures in toy breed dogs that are treated with external coaptation (splint or cast) have an increased risk of nonunion. **B,** In some cases, the radius and ulna may atrophy.

Angular Limb Deformities

Angular limb deformities can occur as a developmental abnormality or as a result of trauma to a physis. The most common deformity is carpal valgus as a result of fracture of the distal antebrachium. As the animal continues to grow, it may develop subluxation of the elbow and carpal joints with subsequent OA in these joints. Correction of angular limb deformities involves a corrective osteotomy of the affected bone(s) and stabilization with a bone plate or external fixator. In some cases, a circular fixator is used to gradually correct the angular deformity and lengthen the bones by distraction osteogenesis. After surgery cryotherapy and NSAIDs may be used to limit inflammation and pain. It is important to maintain normal ROM of the joints proximal and distal to the repair. This is particularly important for animals undergoing distraction osteogenesis, because the concurrent stretching of soft tissues in the region may cause reduced joint ROM, stiffness, and lameness. Stretching and ROM activities with simultaneous application of heat or therapeutic ultrasound may be beneficial. As bone healing progresses the intensity or duration of aquatic therapy and weight-bearing therapeutic exercises may be increased.

Contracture of the Flexors of the Antebrachium

After limb immobilization or prolonged disuse, the antebrachial flexor muscles may undergo contracture (shortening of the tendon-muscle unit). The result is limited extension of the joints distal to the elbow, which can impair normal weight-bearing. Methods of lengthening and stretching the contracted tendon-muscle units include 3-MHz therapeutic ultrasound or hot packs, manual stretching, exercises, and massage.[42-44] If stretching is too aggressive it may cause damage to soft tissues or bone fractures. A prolonged stretch may be applied with a custom splint on the distal limb. The splint should be applied intermittently to minimize the risk of pressure injuries from the splint. The splint may be applied for longer periods each day until it can be worn all day, if desired. The goal is to stretch and realign the tissues without damaging them.

Carpal, Metacarpal, and Phalangeal Conditions

Carpal, Metacarpal, and Phalangeal Fractures

Fractures of the carpal bones are rare but may involve the accessory carpal bone or the radial carpal bone in racing athletes such as greyhounds. In other breeds carpal fractures are often the result of automobile or other traumatic insults. Some carpal fractures may result in medial or lateral instability if the fracture involves the origin or insertion of the respective collateral ligament. A relatively common injury is a shearing injury, in which the dog's limb becomes trapped beneath a skidding automobile tire. The soft tissues including skin, subcutaneous tissues, tendons, ligaments, and in severe cases, bone and tendon, are eroded away, leaving the joint unstable. Many of these injuries cannot be primarily repaired, and an arthrodesis is performed in these cases. Fortunately, function is usually very acceptable after bony fusion has occurred.

Metacarpal and phalangeal fractures usually occur as a result of trauma, and phalangeal fractures are often open fractures (Figure 32-11). Carpal, metacarpal, and phalangeal fractures may be treated using closed reduction and immobilization in a coaptation device, or with a variety of implants including intramedullary pins, wires, and plates and screws (Figure 32-12). A splint or light cast is usually applied to the limb after internal fixation to protect the relatively small implants that must be used. If a cast or splint is used elbow ROM must be preserved during the time of coaptation application, and after coaptation removal. Sagittal plane ROM exercises and joint mobilization work well to restore ROM to the area. In cases of fracture and carpal instability, splinting is often used to protect the repair. If possible removing the splint on a weekly basis to perform ROM exercises to maintain joint and tendon mobility is advised. The splint is reapplied to protect the fracture after performing ROM exercises.

Carpal Hyperextension

Carpal hyperextension injuries occur after jumping or falling from a height, spraining the palmar fibrocartilage and the carpal ligaments. Damage may occur to antebrachiocarpal, middle carpal, carpometacarpal joints, or any combination of these joints. Carpal hyperextension



Figure 32-11 When all four metatarsal or metacarpal bones have fractured, surgical repair is generally preferable to external coaptation. **A,** A-P view, **B,** Lateral view.

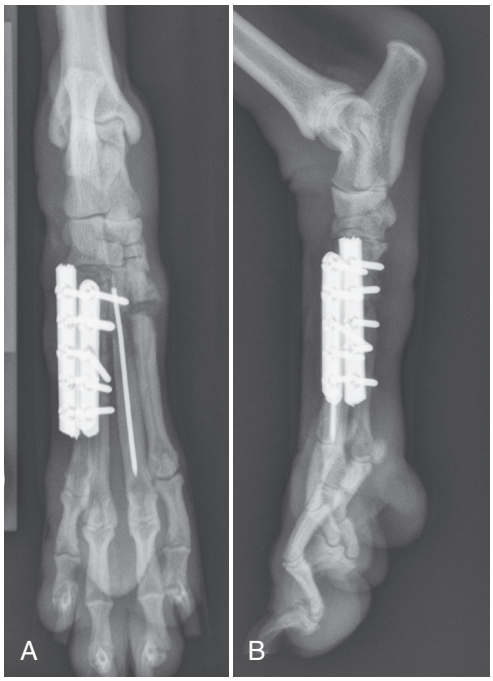


Figure 32-12 Metatarsal fractures that have been stabilized with bone plates and intramedullary pin. **A,** A-P view, **B,** Lateral view.

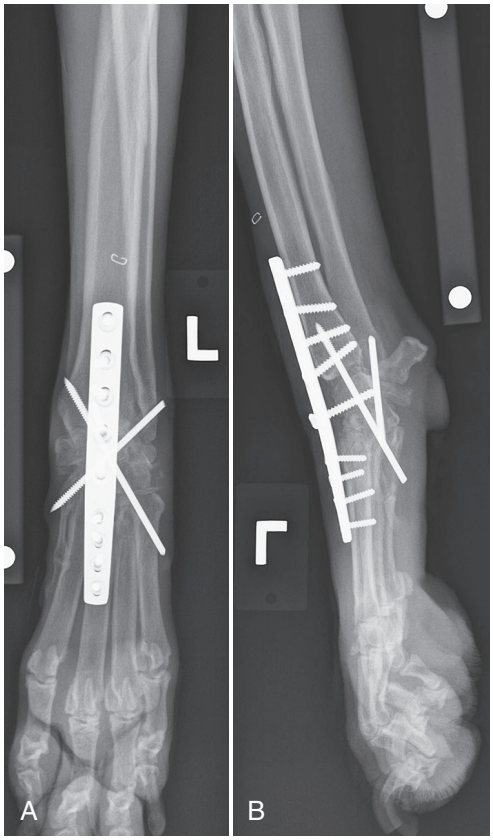


Figure 32-13 Pancarpal arthrodesis performed in a patient with chronic carpal hyperextension. **A,** A-P view, **B,** Lateral view.

may also be caused by immune-mediated joint disease, or endocrinopathy such as hyperadrenocortism. Affected animals are lame and walk with the carpus in hyperextension. In severe cases, the carpus may touch the ground. In general healing of a sprain (ligament injury) may take months, and occurs by the formation of fibrous connective tissue to replace the torn ligament, rather than primary healing of the ligament. Conservative management of carpal hyperextension injuries is generally unsuccessful and results in subluxation.[45] The recommended treatment is panarthrodesis (surgical fusion of all three joint levels) or partial arthrodesis (fusion of only the middle and distal joints), depending on the level of the injuries (Figure 32-13). Rehabilitation is performed as for any arthrodesis, with an emphasis on gait training after the initial healing period (see General Physical Rehabilitation of Joint Arthrodesis). Although the limb function is not normal, prognosis for return to daily activity is good after carpal arthrodesis.[46] The prognosis is more guarded if instability is caused by rheumatoid arthritis because multiple joints are typically affected.

Carpal laxity syndrome has been described in puppies, which walk with the carpus in a hyperextended position.[47] The cause is unknown but is speculated to be caused by asynchronous growth between the skeleton and the musculature. Long-term application of supportive splints for hyperextension is avoided because it may lead to muscle atrophy and increased tendon weakness (Figure 32-14). Treatment recommendations generally include balanced diet and controlled exercise on surfaces that provide good



Figure 32-14 This puppy had increased carpal laxity after long-term use of a splint.

traction. Carpal arthrodesis may be a treatment option for hyperextension that does not resolve with exercise and nutrition, but this cannot be performed until the physes are closed. Carpal laxity syndrome is usually self-limiting, with a short duration and good prognosis.

Carpal flexural contractures have also been recognized in puppies. Poor nutrition has been implicated as a factor, so altering the diet may be beneficial. Splinting may be helpful for puppies that are developing flexural contractures. In severe cases of flexural contracture, tenotomy may be performed.

Trauma of the Superficial and Deep Digital Flexor Tendons

Laceration or avulsion of superficial and deep digital flexor tendons are traumatic injuries that can occur in the front or rear feet. Avulsion or laceration of the tendon insertion on P2 and P3 results in abnormal toe carriage. The tendons may be reattached or repaired for cosmetic reasons and for improved athletic function. Postoperatively the foot is immobilized in a splint for 2 to 3 weeks to limit edema and provide support. Active motion is limited to prevent disruption of the weakened tissue and allow the tendons to heal with minimal adhesions.

Weekly splint changes with ROM exercises of adjacent joints should be performed if possible.[3] Passive range of motion and light activity are used to maintain normal tissue length and encourage normal joint movement. Removal of the splint is recommended within 3 weeks of the injury, because prolonged immobilization may delay healing and weaken the repaired tissue. Some tendon motion promotes remodeling of the peritendinous scar, which allows the tendon to glide. However, restricted activity must be enforced, because running or jumping could place catastrophic stress on the healing tissues and result in failure. By 6 weeks the tendons should have achieved adequate strength to sustain normal physiologic stress. At this point the focus of therapy is to restore ROM and strength. If there is contracture of muscle-tendon units, 3-MHz continuous-mode therapeutic ultrasound or hot packs with simultaneous stretching is indicated.[3]

Luxation of the Metacarpophalangeal and Interphalangeal Joints

Metacarpophalangeal and phalangeal luxations are treated by closed reduction or surgical repair of the collateral ligaments and joint capsule. After repair the foot is splinted for 2 to 3 weeks. It is beneficial to change the splint once weekly so as to perform gentle PROM on the immobilized joints. After the splint has been permanently removed, ROM exercises in the sagittal plane are continued. Limited weight-bearing activities are initiated and gradually increased over another 3 weeks.

Other options for metacarpophalangeal and phalangeal luxations include arthrodesis of the joint or amputation of the digit. Arthrodesis is a better choice if a high level of function, such as racing, is required. Postoperatively the foot is splinted until there is radiographic evidence of fusion. Weekly splint changes with PROM or mobilization of the other distal joints may be beneficial.

Pelvis and Hip Conditions

Pelvic Fractures

Pelvic fractures account for a large percentage of fractures seen in veterinary practice. Because of the rigid, box-like anatomy of the canine pelvis, trauma usually results in two or more fractures of the pelvis (Figure 32-15, *A*). In general surgical repair is recommended for fractures within the weight-bearing regions of the pelvis, including the sacroiliac joint, ilial body, and acetabulum. Surgical repair is also recommended if there is neurologic impairment (e.g., possible sciatic nerve entrapment) or medial displacement of fracture fragments impinging on the pelvic canal. Surgical repair results in a quicker return to function with fewer changes secondary to tissue disuse, and the level of function will generally be superior. It is important that surgical repair be undertaken within the first 7 to 10 days after injury to minimize damage to soft tissues and nerves during reduction of the fractures. In some instances, conservative treatment in the form of cage rest may be adequate to allow healing. Examples of injuries amenable to conservative treatment include greenstick fractures of the body of the ilium, mild displacement of the sacroiliac joint with minimal pain or lameness, and fractures of the caudal 20% of the acetabulum. Although pubic and ischial fractures may appear quite severe on a radiograph, these injuries are rarely repaired because they are not part of the weight-bearing axis of the pelvic limb and will heal with rest. Repair of the injuries to the sacroiliac joint, ilium, and acetabulum generally results in adequate reduction and realignment of pubic and ischial fractures for healing.

It is important to identify any concurrent injuries with pelvic fractures. Trauma to the spinal cord or peripheral nerves is relatively common, especially with fractures of the sacrum, fractures of the body of the ilium with medial displacement of the caudal fracture fragment, and fractures of the cranial ischium in the region traversed by the sciatic nerve. It is important to perform a thorough neurologic examination, checking for pain sensation, motor function, and reflexes. Because injuries to the pelvis often involve both pelvic limbs and other injuries to the distal limbs are frequently present, evaluation of the patient while ambulating or performing proprioceptive tests may not be possible. In addition abdominal and thoracic trauma is relatively common in animals with pelvic injuries.

Sacroiliac joint luxations are surgically managed by placing one or more screws in lag fashion to compress the joint. The screw(s) should engage the body of the

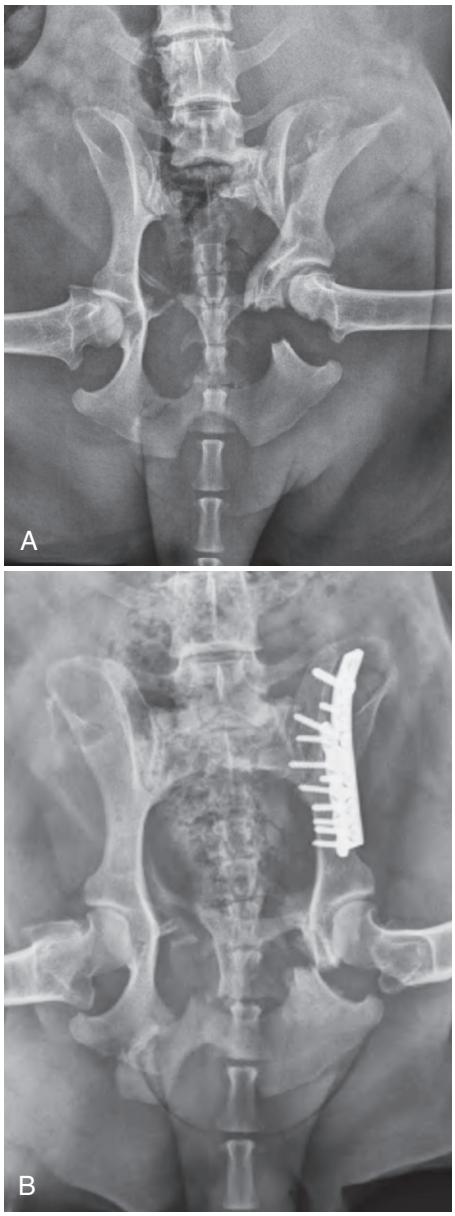


Figure 32-15 The pelvis usually has more than one fracture because of its box-like construction. Typically only the weight-bearing regions of the pelvis are surgically stabilized. **A,** V-D view prior to surgery, **B,** V-D view after surgical stabilization.

sacrum and be deeply seated into the bone, so that the screw engages at least 60% of the width of the sacrum (Figure 32-16). Fractures of the sacral body or wing can be challenging to repair, and concurrent nerve injuries are quite common.

Fractures of the body of the ilium are usually repaired with a bone plate and screws (Figure 32-15, *B*). It is important that the plate be properly contoured to maintain the caudal half of the pelvis in anatomic alignment to avoid future problems associated with a narrowed pelvic canal, such as constipation and obstipation. During the fracture

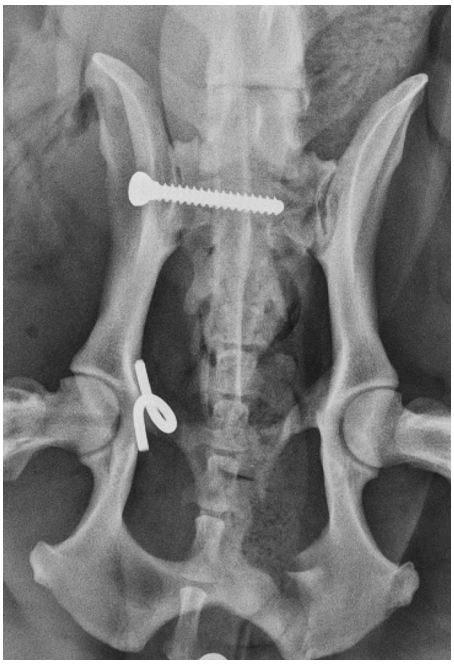


Figure 32-16 A sacroiliac luxation has also been stabilized with a screw. An ipsilateral hip luxation is repaired using a toggle pin as evidenced by the metal implant medial to the acetabulum and bone tunnel in the femoral head and neck.

reduction, fragments must be carefully manipulated to avoid damage to the lumbosacral plexus, which is located just medial to the body of the ilium.

Fractures of the acetabulum secondary to automobile trauma are common in dogs. Most acetabular fractures should be treated with open reduction and internal fixation. Depending on the size of the patient and the nature of the fracture, repair may involve plates, screws, wires, or polymethylmethacrylate. In many acetabular fractures, anatomic reduction is difficult to achieve, and many animals with these fractures develop OA. Comminuted fractures may be difficult or impossible to reduce, and femoral head and neck ostectomy (FHO) may be necessary. The surgeon should be aggressive in removing as much bone as is necessary, from both the femoral and the acetabular side of the FHO, to prevent bone rubbing on bone postoperatively. It is not uncommon for scar tissue to develop, causing late bony contact in FHOs performed to treat acetabular fractures; therefore early and intense rehabilitation is critical in both preventing and detecting problems before they cause loss of use of the limb (see section on excision arthroplasty).

Rehabilitation should be started immediately postoperatively, even if the animal is nonambulatory. The biomechanical limitations of the fracture repair must be considered, but analgesia, cryotherapy, and passive ROM exercises can usually be initiated immediately postoperatively. On the first postoperative day sling therapy may be

started if the patient is stable. This typically involves short, 5-minute sessions once or twice daily. Advantages of sling therapy include psychological benefits of the patient being in a more normal position, the ability to begin assisted active motion and early, limited weight bearing, and improved access for the caretakers to perform massage and ROM exercises. Rear limb slings can be used with pelvic trauma, whereas whole body slings can be used for patients with forelimb and rear limb trauma. After the incisions are sealed and the patient no longer has intravenous catheters or other invasive lines, water treadmill, or swimming therapy can be considered.

Water treadmill therapy for polytrauma patients is very useful because it allows the therapist to tailor the amount of weight bearing (a water level at the height of the greater trochanter allows the dog to walk with only 38% weight bearing on its limbs). The pressure and buoyancy of the water can also help improve blood flow and decrease edema. Many dogs seem to enjoy being in the underwater treadmill. It can be very demanding for the dog to walk for any length of time after trauma, so the cardiovascular status of the dog should be monitored and very short (several minutes) sessions should be used for the initial treatments.

Hip Dysplasia

Hip dysplasia is an abnormal development of the hip joint, usually bilateral, that occurs primarily in medium and large breed dogs. The causes are multifactorial and include genetic predisposition, rapid growth rate, and diet. Puppies with a genetic predisposition for hip dysplasia develop clinical disease much later in life and to a much lesser extent when fed a restricted calorie diet, as compared with littermates that are fed ad libitum.[48] The hips are normal at birth but develop instability in the early stages of the disease, between 4 and 12 months of age. At this stage the dog may exhibit difficulty rising, decreased activity level, "bunny-hopping" gait, and loss of muscle mass in the hindquarters. Early in the disease process the diagnosis is made by palpation of joint laxity (subluxation). Radiographs may appear normal, although some degree of subluxation may be evident, especially with distraction views (Figure 32-17). As the disease progresses periarticular fibrosis provides some joint stability and the pain may be significantly decreased. However, with further progression of the disease, OA results in pain, crepitus, decreased range of motion, waddling gait, and reluctance to exercise. Thigh and hip muscles atrophy and shoulder muscles may hypertrophy because the body weight is shifted toward the forelimbs. Varying degrees of OA with remodeling of the femoral head and acetabulum are seen radiographically. The rate of progression of hip dysplasia varies between individuals and is difficult to predict. Some dogs may have degenerative changes before 1 year of age, whereas many individuals develop advanced OA in midlife or later.



Figure 32-17 Hip subluxation is typically present bilaterally as a condition of early hip dysplasia. The degree of visible subluxation may be affected by pelvic positioning when making the radiograph.

In young dogs, there are a number of preventive surgical procedures that are designed to change the joint alignment to improve joint stability and slow the progression of OA. The most common of these procedures are the triple pelvic osteotomy (TPO) and juvenile pubic symphysiodesis. Triple pelvic osteotomy is performed on dogs that have early signs of hip dysplasia and joint laxity, but have not progressed to the point of having significant radiographic evidence of OA. Most dogs that fit these criteria are between 4 and 10 months of age. They usually have atrophy of the gluteal and thigh muscles. The technique involves making three osteotomies to mobilize the acetabulum and change its orientation. A bone plate is used to stabilize the ilial osteotomy (Figure 32-18). Postoperatively activity is restricted for 4 to 6 weeks to allow bone healing. Cryotherapy, NSAIDs, PROM, and assisted ambulation, followed by controlled, low-impact therapeutic exercises, are indicated. Leash walking, sit-to-stand exercises, and aquatic walking may be useful to attenuate muscle atrophy while avoiding excessive stress on the repair. After adequate bone healing has occurred the focus of rehabilitation is to strengthen muscles of the hindquarters. Strengthening activities should parallel tissue healing and tissue strength.

Juvenile pubic symphysiodesis is a performed in dogs between 16 and 18 weeks of age that are considered to be at risk for developing hip dysplasia. The pubic symphysis is surgically damaged, causing it to fuse and alter pelvic growth. These puppies are often clinically normal at the time of surgery and the surgical trauma is minimal. The focus of rehabilitation is to promote muscular development of the hindlimbs with low-impact exercise.

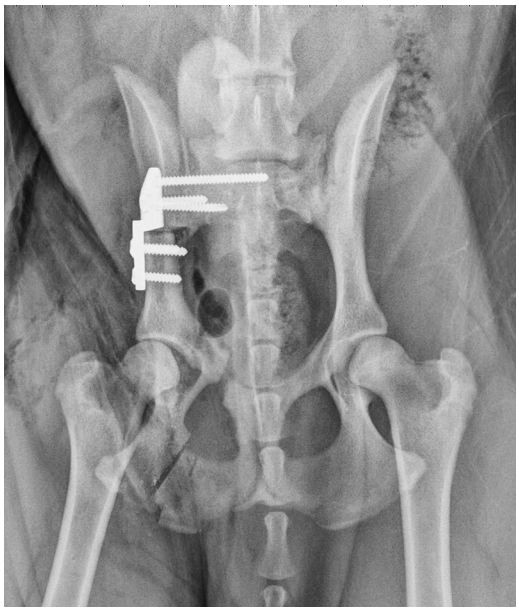


Figure 32-18 Triple pelvic osteotomy has been performed to improve coverage of the head of the femur by the acetabulum.



Figure 32-19 Cementless total hip replacement performed in a patient with severe osteoarthritis secondary to hip dysplasia.

Dogs with mild or intermittent signs related to chronic hip OA may be treated by conservative methods to improve function and limit the progression of OA. This includes a combination of NSAIDs, DMOAs, modalities such as heat, electrical stimulation, therapeutic ultrasound, laser, diet and exercise, and is discussed in the section on OA. For dogs having disability or pain that is not adequately managed by conservative methods, there are two salvage surgical options: total hip replacement (THR) or femoral head and neck ostectomy (FHO). Both procedures eliminate the normal joint, thus eliminating the pain. In general, THR is not recommended following an FHO that results in poor function. For rehabilitation after FHO, see the section on excision arthroplasty.

Total hip replacement eliminates the joint, thus eliminating joint-associated pain. The femoral head is removed and the acetabulum is replaced with a prosthetic acetabular cup. A femoral prosthesis is implanted in the medullary canal of the femur. The prostheses may be secured with bone cement, but some systems do not use cement and rely on bony ingrowth for stability (Figure 32-19). Most dogs have excellent weight bearing almost immediately after surgery. In the initial postoperative period NSAIDs, cryotherapy, and PROM are indicated. One of the most common postoperative complications is hip luxation,[49] so muscle strengthening after a period of healing is important, especially because there is often preexisting muscle atrophy. Close confinement is enforced when the dog is unsupervised during the first postoperative month. During early ambulation the dog is supported with a sling to prevent adduction or abduction of the limb and dislocation of the prosthesis and inadvertent falls. In some cases, PROM and stretching exercises are indicated to improve hip mobility. Muscle strengthening can be achieved using controlled walking, treadmill activity, and sit-to-stand exercises. The duration of these activities is gradually increased during the first 3 months. Balance and proprioception reeducation may also be important. Dogs are restricted to leash walking, with no running or jumping for the first 3 postoperative months to reduce the chances of implant loosening or dislocation. Prognosis is good to excellent in most cases.

Legg-Calve-Perthes Disease

Legg-Calve-Perthes disease is noninflammatory aseptic necrosis of the femoral head and neck that occurs in small breed dogs. The etiology is unknown, although a genetic component has been identified in some breeds. Dogs usually develop lameness between 6 and 10 months of age. With chronic lameness there is muscle atrophy of the hip and thigh muscles. Radiographs show deformation of the femoral head and neck, with joint incongruity (Figure 32-20, *A*) In some cases there may be advanced OA or fractures of the femoral head and neck. If the condition is diagnosed early, a non–weight-bearing sling may be used for 3 to 4 weeks.[50] However, the treatment of choice is generally FHO (Figure 32-20, *B*). Prognosis is good after surgery, provided appropriate rehabilitation is performed beginning immediately after surgery (see General Physical Rehabilitation Management of Excision Arthroplasty).

Hip Luxation

Coxofemoral luxation is common and usually a result of automobile trauma. However, the presence of hip

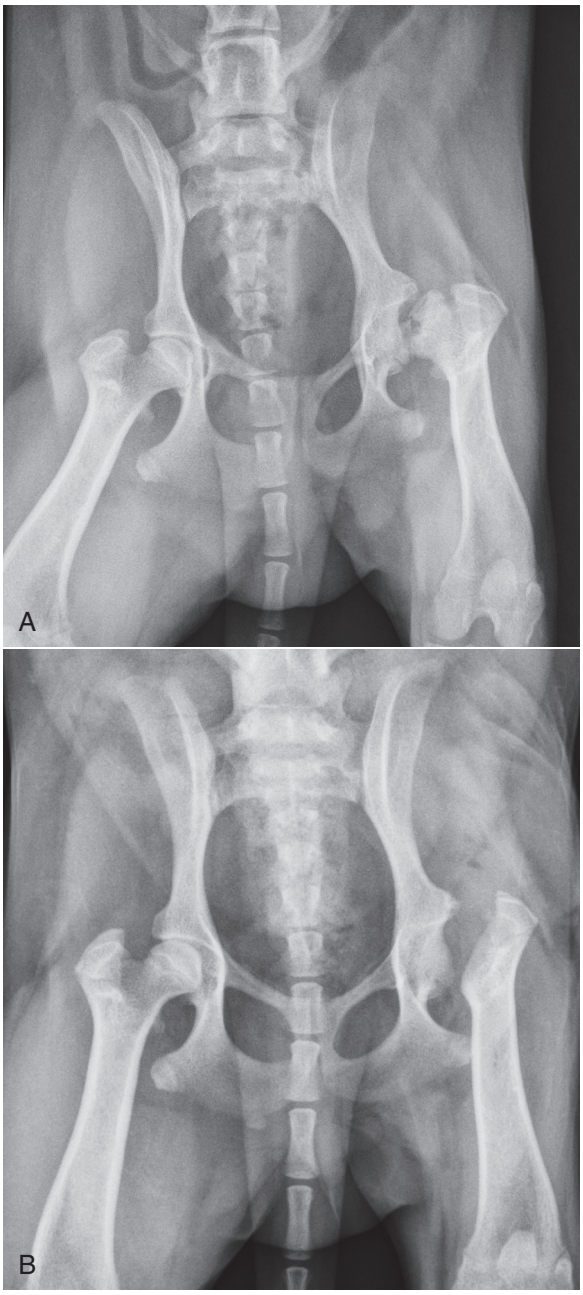


Figure 32-20 **A,** Young dog with Legg Calve Perthes disease, V-D view. **B,** Radiograph taken after femoral head and neck excision. Note the complete removal of the femoral head and neck.

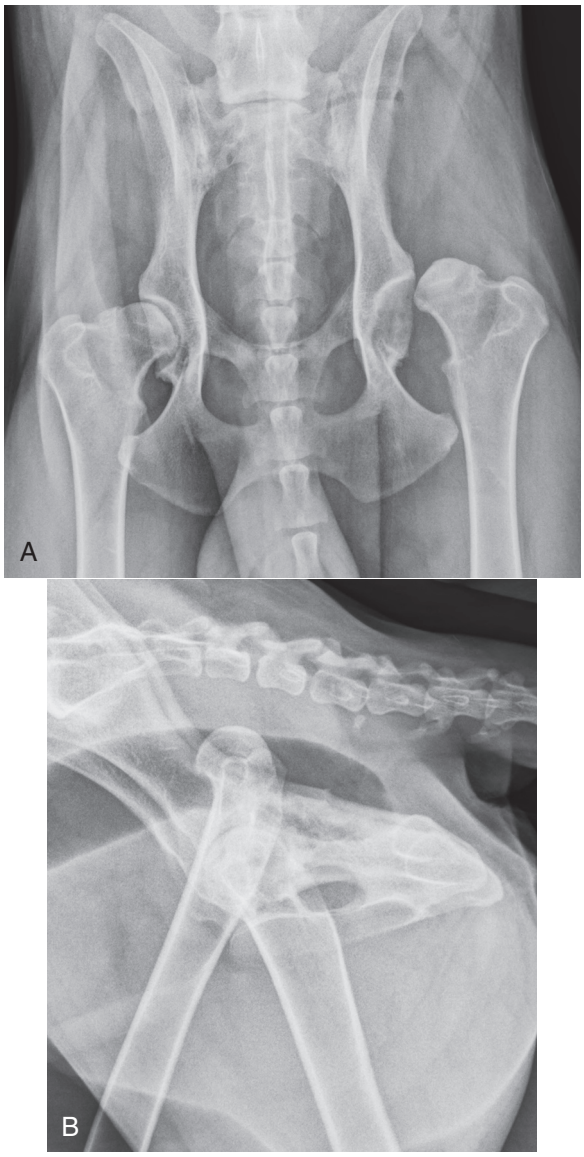


Figure 32-21 Craniodorsal hip luxation in a dog with osteoarthritis associated with hip dysplasia. **A,** V-D view, **B,** Lateral view.

dysplasia may diminish joint stability and predispose to luxation with less severe trauma. Hip luxations usually have a craniodorsal displacement. The diagnosis can often be made by palpation of bony displacement, but radiographs are indicated to evaluate for the presence of concurrent fractures or hip dysplasia (Figure 32-21). The presence of these abnormalities may affect the treatment choice and the prognosis.

If the diagnosis is made within a few days of the injury and there is relatively normal coxofemoral anatomy with no articular fractures, closed reduction and immobilization in an Ehmer sling for 1 to 3 weeks may be successful. An Ehmer sling theoretically maintains the femur in a more stable position of abduction and internal rotation. Eliminating or minimizing weight-bearing activity minimizes stresses on the periarticular tissues to allow them to heal. Open reduction and surgical stabilization are indicated if closed reduction is not successful or articular fractures are present. Common surgical repair techniques include suturing the joint capsule (capsulorraphy), creating a prosthetic capsule with suture, or toggle pin fixation (see Figure 32-16). In some cases a combination of techniques may be

used, or the greater trochanter may be relocated more distally and caudally to provide additional stability. After surgery joint healing is often protected by immobilization in the Ehmer sling for 1 to 3 weeks, depending on the degree of tissue damage and the type of repair.

If an Ehmer sling is used, the animal should be evaluated at least weekly to monitor for skin irritation and reluxation. Rehabilitation is begun after the sling is removed. Range of motion must be restored without causing the joint to reluxate. Cryotherapy and NSAIDs are used to minimize inflammation in the early postoperative period. Stretching and PROM activities are also important after hip luxations. Range of motion should be limited to comfortable flexion and extension, with external rotation, abduction and adduction not performed at all during the healing phase of a craniodorsal luxation. Muscle strengthening and re-education begins with walking on a level surface or a downhill grade. Uphill walking, stair climbing, dancing exercises, and aquatic walking may be used to help with hip extension and muscle strengthening during the latter phases of rehabilitation. Walking on an inclined treadmill may promote active hip motion, although the inclination may need to be greater than 5% to cause much increase in hip ROM.[51] In the case of ventral hip luxation, limb abduction should be avoided, and hobbles may be helpful post-reduction to prevent excessive abduction. In all cases activity is gradually increased, but aggressive activities such as jogging, swimming, or uncontrolled play are limited until the joint has healed. This may take 1 to 3 months, depending on the degree of tissue damage.

Joint preservation may not be recommended if there are irreparable fractures involving the joint, hip dysplasia with very poor joint congruency, severe OA, or significant damage to the articular cartilage. In these cases a joint salvage procedure may be a better choice. Common salvage procedures include FHO or THR.

The prognosis for traumatic joint luxations is generally good, although OA is a common sequela. If damage to the articular cartilage is noted at the time of surgery, long-term problems with OA may be expected. If the OA becomes severe, a salvage procedure may be indicated.

Thigh Conditions

Femoral Fractures

Fractures of the shaft of the femur are probably the most common fractures seen in veterinary practice. As such they also have the highest incidence of complications, including delayed or nonunion healing and osteomyelitis. Most complications are related to inadequate fixation or errors in the application of the implants. The possible configurations of femoral fractures vary widely, from a two-piece spiral fracture to a highly comminuted fracture resulting from a gunshot injury. Most of the implants that are available for fracture repair in veterinary medicine may be used to stabilize fractures of the femur, including plates, screws, wires, pins, interlocking nails, and external skeletal fixators (Figure 32-22). Implant failure can result either from single catastrophic overload or from fatigue, implying a greater number of normal weight-bearing cycles than the implant can bear before fracture healing is complete. In general, plates, interlocking nails, and some external fixators tend to be more biomechanically stable than intramedullary pin and cerclage wire constructs.

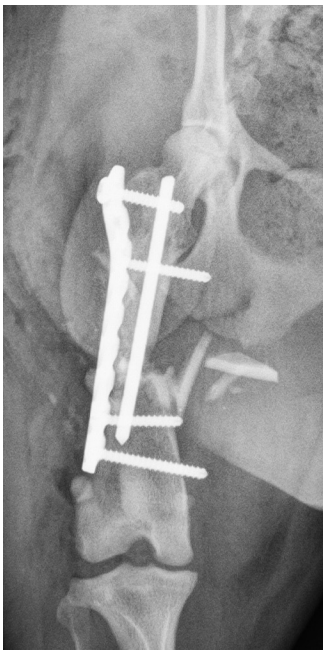


Figure 32-22 Repair of a severely comminuted femoral fracture with a plate-rod technique. A severe fracture configuration suggests a large amount of soft tissue trauma as well.

External coaptation with splints or casts is contraindicated for stabilization of femur fractures because coaptation cannot adequately immobilize the hip joint. In addition coaptation on the limb may only extend as proximally as the mid-thigh, and if so, it can create a fulcrum effect to increase movement at the fracture.

The rehabilitation plan should be based on a number of factors, including the location of the fracture (articular, physeal, long bone), whether more than one limb or bone is involved, pre-existing patient disease (obesity, OA), degree of soft-tissue injury (low energy vs. high energy fracture), the presence or absence of open wounds, and the fixation method(s) used. It is vital that the therapist understand the nature of the fracture and the type of repair that has been applied to know how much activity the dog should have, and what types of complications may occur. For example, if a puppy with a two-piece spiral fracture stabilized with an intramedullary pin and cerclage wires has a sudden loss of limb use with knuckling on the dorsum of the paw and severe pain, the therapist should realize that

the pin may have migrated and entrapped the sciatic nerve. This dog must be treated immediately if there is hope of preventing permanent nerve damage.

Ideally rehabilitation of the fracture patient begins preoperatively, beginning at the time of presentation. Cryotherapy (cold packing) is an essential and simple modality that can be applied in any practice setting. Cryotherapy generally consists of commercial or homemade ice packs that are directly applied over the closed fracture for 10 to 20 minutes, repeated every 2 to 4 hours if practical. Cryotherapy may decrease edema, hemorrhage and inflammation in the soft tissues surrounding the fracture site. Ice packs may be applied during triage of the fracture patient, assuming that body temperature is above 98° F. Temporary stabilization (cast, Robert Jones bandage, or splint) may be applied in some situations if adequate stability can be achieved. The combination of appropriate cryotherapy plus supportive bandaging can dramatically decrease soft-tissue damage and swelling, facilitating surgical repair and decreasing muscle fibrosis postoperatively. As soon as the patient is stable, analgesics should be administered both on a humane basis and to facilitate postoperative rehabilitation by preventing hyperesthesia associated with "wind-up" phenomenon caused by untreated preoperative and intraoperative pain.[52]

After surgery, the surgeon's opinion regarding risk of implant failure should be communicated to the rehabilitation team (Figure 32-23). In general NSAIDs, cryotherapy, PROM, and controlled activities are initiated following surgery. More active forms of rehabilitation such as treadmill walking, UWTM, leash walking, stairs, and swimming are added according to the nature of the injury, method used for fixation, stability of the repair, and temporal aspects of wound and fracture healing. The ROM achieved by the hip joint is affected by the severity of the fracture and the amount of periarticular fibrosis that occurs. Although most dogs develop some degree of OA with fractures of the femoral head or neck, most dogs do reasonably well, although some are candidates for THR or FHO. In addition, if concurrent neurologic injuries are present, appropriate balancing, proprioceptive, and strengthening activities should be initiated.

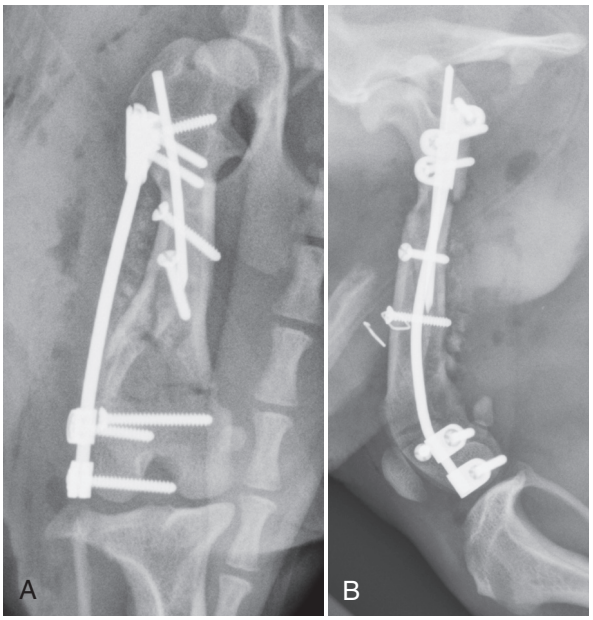


Figure 32-23 Repair of a severely comminuted femoral fracture that involves the stifle joint. **A,** A-P view, **B,** Lateral view. Good communication between the therapist and surgeon are important to understand the estimated stability of the surgical repair. In this case, the presence of a screw in the caudal aspect of the stifle joint may also affect the therapeutic plan.

Femoral Capital Physeal Fractures and Femoral Neck Fractures

Proximal femoral fractures may occur through the capital femoral physis of immature dogs or across the femoral neck of skeletally mature dogs. Rarely do fractures of the articular surface of the femoral head occur. The most serious of the fractures of the proximal femur is the capital femoral physeal fracture, especially in dogs less than 5 months of age. In this fracture the physis may close prematurely, causing a short femoral neck and a small femoral head that results in an incongruent hip joint. In spite of anatomic reduction and internal fixation, these fractures result in OA because of this incongruity.

Femoral capital physeal fractures are typically repaired with either lag screws or divergent K-wires (Figure 32-24). Early repair and minimal soft tissue dissection result in fewer complications. Although they are not generally associated with the muscle contracture that can occur with distal femoral fractures, because of the very small epiphyseal segment and possible tenuous repair, surgeons may occasionally elect to place the limb in an Ehmer or 90-90 sling for 7 to 14 days after surgery. Ideally such slings should be removed as early as possible, and cryotherapy, PROM, and early weight-bearing active ROM should begin within a few days. Collapse of the fracture around the pins can occur. If crepitus, loss of ROM, or increase in pain occur during rehabilitation exercises postoperatively, the surgeon should be contacted immediately or radiographs obtained to assess for loss of reduction or implant penetration into the articular space.

Distal Femoral Physeal Fractures

Although many different repair techniques are described for distal femoral fractures, the most common is cross-pinning. Distal physeal fractures can be difficult to reduce, particularly if they are more than 48 hours old. In addition, distal femoral physeal fractures are predisposed to a devastating postoperative complication characterized by

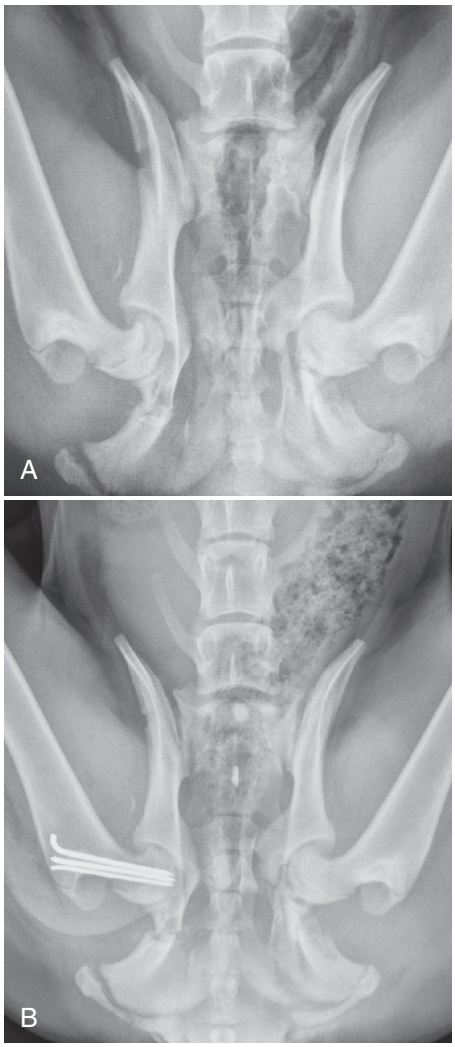


Figure 32-24 Capital femoral physeal fracture in a skeletally immature dog. **A,** V-D view prior to surgery, **B,** V-D view after surgery.

quadriceps contracture. A frequent scenario is that the surgical repair is performed, immediately postoperatively the animal seems to have good ROM of the stifle joint, but by the time of suture removal there is excessive stifle extension and severe loss of ROM. Rehabilitation following distal femoral fracture repair is focused on maintaining stifle flexion and hip extension (see Quadriceps Contracture for more detail on rehabilitation). With a stable repair and good rehabilitation, distal femoral physeal fractures are associated with a good prognosis.

Quadriceps Contracture

Quadriceps contracture, also called quadriceps tie-down, is characterized by scarring of the quadriceps muscle group with adhesions to the distal femur, so the stifle is unable to fully flex and the limb is carried in extension (Figure 32-25). Quadriceps contracture can occur secondary to severe muscle trauma or Neospora, but it is most



Figure 32-25 Inadequate postoperative management a distal femoral fracture led to quadriceps muscle group contracture in this cat.

commonly associated with distal femoral fractures in young animals. The likelihood of quadriceps contracture is greatly increased when the limb is immobilized in extension (e.g., with a cast or splint) following repair of a distal physeal fracture. Splinting in extension for as little as 5 to 7 days may result in quadriceps contracture. Chronic disuse can lead to demineralization of the long bones within the affected limb. There may also be disuse atrophy of the articular surfaces of the stifle and periarticular fibrosis. Severe cases can exhibit genu recurvatum or stifle joint ankylosis. Advanced cases of contracture carry a poor prognosis and commonly result in a nonfunctional leg. Surgical release of the fibrous tissue in combination with intense physical rehabilitation can be attempted.[53] After fibrosis has occurred attempts to break down the scar tissue by performing ROM exercises, even under anesthesia, may result in refracture of the femur. Surgical release of the contracture without intense rehabilitation will result in reformation of scar tissue within a few weeks. This complication has been successfully managed with a combination of surgical release, dynamic stifle flexion apparatus, and an intensive passive and active ROM exercise program.[53] Generally the prognosis is poor and the end result can be an amputation after what initially appeared to be a successful fracture repair.

This problem is best addressed by preventing its occurrence, and prevention of quadriceps contracture depends on several factors. The fracture should be repaired as soon as possible, preferably within 24 hours. Gentle tissue dissection and the use of minimally invasive techniques that minimize tissue trauma (e.g., fluoroscopic guidance of pins) should be employed to facilitate early limb use. Although the ends of the cross-pins may be bent to prevent migration, excess pin protrusion into the soft tissues may cause more pain and inhibit rehabilitation exercises. Some surgeons prefer to countersink the cross pins so there is



Figure 32-26 The 90-90 flexion sling is similar to an Ehmer sling. The goal of the 90-90 is to keep both the stifle and hock joints maintained in 90 degrees of flexion. An Ehmer sling attempts to keep the hip abducted and internally rotated, and the major limb joints are typically fully flexed.

less interference with ROM. The fracture must be anatomically reduced. For any distal femoral fracture, rehabilitation must begin the day of or the day after surgery. If there is any question about the possibility of performing appropriate rehabilitation (e.g., the animal is going home the day after surgery and it is difficult for the owners to manage), the limb should be bandaged in a flexed position to maintain the quadriceps muscle in a stretched position during the acute postoperative inflammatory phase. This can be accomplished by use of a 90-90 flexion sling for 72 hours postoperatively, and this may greatly decrease the risk of contracture (Figure 32-26).[54] If feasible the sling should be removed on a daily basis and PROM and stretching exercises performed. Cryotherapy can be applied with the sling in place.

If rehabilitative care is readily available, a more physiologic program may be employed. Immediately postoperatively, before anesthetic recovery, cryotherapy is performed and followed by gentle passive ROM exercises. Aggressive treatment of pain, often achieved using a combination of NSAIDs and opioids, is mandatory to successful rehabilitation efforts and this should begin before recovery from anesthesia. After the animal has recovered, cryotherapy is continued three to six times daily. Immediately after cryotherapy, gentle massage is used, working from the distal aspect of the limb toward the proximal limb for 2 to 3 minutes. Although the stifle is the primary area of interest, it is important to treat the whole limb. Therefore gentle flexion and extension of the digits with some stretching is done first, moving up to the hock, stifle, and hip, using 15 to 20 repetitions per joint. For the stifle, it may be helpful to use some gentle stretching (hold the joint at maximal comfortable flexion or extension for 30 seconds). Patella mobilization and tissue mobilization techniques may be useful to help prevent fibrosis of the various tissue layers to one another. It is important that the process not be painful for the animal and vigorous manipulations are avoided. Cryotherapy is generally continued for at least 72 hours. If the animal resents ROM or tends to hold the limb in extension, a 90-90 flexion sling may be applied intermittently for the first 3 days.

Many animals begin to bear some weight on the limb within 1 to 2 days after surgery, and short, frequent leash walks should be encouraged. Cryotherapy may be replaced with heat therapy after the acute inflammatory phase of wound healing is over, which is typically about 3 to 5 days. Heat causes vasodilation of the cutaneous blood vessels, improves muscle and connective tissue extensibility, and increases the pain threshold. Because of these tissue effects, heat can be very useful before stretching, ROM, and exercise sessions. Some therapists recommend using heat before exercise and cryotherapy after.

Early active exercises should be performed, primarily with very slow leash walks and balancing exercises that encourage active contraction and relaxation of the muscles, and flexion and extension of the stifle. Gentle tug-of-war may encourage limb use, but caution should be used to ensure that excessive force is not placed on the limb. If the animal is leash trained, treadmill walking may cautiously be attempted. It is recommended that an attendant help support the animal and be immediately available in case of resistance on the part of the patient. A syringe cap placed under the contralateral rear foot is also useful to encourage limb use. Finally, swimming is useful to encourage active flexion and extension of the stifle joint, but the incision should be sealed with no drainage or discharge. If the animal is released from the hospital within a few days of surgery, a recheck evaluation should be performed within a few days to ensure that stifle ROM is not becoming reduced. If ROM is preserved and limb function is improving, the animal should be rechecked weekly until the time of radiographic healing, usually by 4 weeks.

Muscle Strain

A strain is a tear or rupture of some part of the muscle-tendon unit, which is caused by excessive stress on the tissues. The injury may occur in the muscle belly, musculotendinous junction, tendon, or teno-osseous junction, but typically affects the musculotendinous region. Mild muscle strain (grade 1) may cause low-grade inflammation with minimal alteration in strength, ROM, or gait. There may be no obvious swelling or palpable pain. Healing may occur within days. With moderate muscle strain (grade 2) there is more damage to muscle and fascia with diminished strength and ROM. Moderate strains generally require one to three weeks to heal. Severe muscle strains (grade 3) are characterized by complete rupture of muscle fibers and fascia, with total loss of muscle function. Healing may take

months, and in some cases a chronic recurring tightness may restrict full athletic function.

Muscle strains occur commonly in human athletes, so it is likely that they are underdiagnosed in canine athletes.[55] It can be difficult to diagnose a muscle strain, especially because dogs with muscle strains often have concurrent orthopedic problems. Diagnosis may be made by eliciting pain when the affected myotendinous unit is directly palpated or passively moved into a stretched position. Imaging techniques such as diagnostic ultrasound or magnetic resonance imaging may be necessary to establish a diagnosis.

Management of acute muscle injuries varies with the severity of injury. Large areas of muscle disruption or total muscle rupture should be surgically repaired.[56] Grade 1 or 2 injuries are typically treated conservatively. This includes the use of analgesic agents, cryotherapy, and rest or controlled activity. Stretching is typically avoided during the acute phase of healing. Within the first week after injury or surgical repair, pulsed therapeutic ultrasound and laser may be used for their nonthermal effects on tissue repair.[3] If muscle is surgically repaired, the area may be immobilized by splinting the limb for several weeks. During this time, PROM of adjacent joints is indicated. After the muscle has healed, muscle lengthening may be promoted by the use of passive stretching and active ROM exercise. Additionally, exercises may be chosen to strengthen the affected muscles. However, there are no established treatment guidelines for muscle strains in dogs and the effectiveness of physical rehabilitation has not been established.[55]

Almost any muscle can sustain injury. Common muscles injured in racing greyhounds include the gracilis, tensor fascia lata, pectorals, and triceps.[56] Gracilis muscle rupture, also known as "dropped thigh" in racing greyhounds, occurs from a single traumatic event and dogs often recover after surgical repair.[29] The iliopsoas, pectineus, adductor, and gracilis have been reported to be affected by muscle strain in the hindlimb of dogs.[55]

Eccentric muscle contraction may play an important role in the development of acute muscle strain injury because a weightbearing force exceeding the force of muscle contraction is applied to the muscle, resulting in muscle lengthening while it is contracting against the load. This leads to muscle damage. Inadequate warm-up, fatigue, or overtraining may also be factors involved in muscle strains.[29] In addition, after a muscle has been injured it may be at risk for reinjury, particularly if the previous muscle strain has not completely healed. Light activity is recommended to increase blood supply and muscle temperature before vigorous exercise to reduce the risk of injury. Stretching may also be performed before exercise, but stretching without an appropriate warm-up may be detrimental.

Fibrotic Myopathy of the Thigh Muscles

Fibrotic myopathy is a condition in which the muscle fibers are replaced with fibrous connective tissue, resulting in a dense fibrous band within the muscle. It has been reported in the semimembranosus, semitendinosus, biceps femoris, gracilis, and sartorius muscles in the dog. It has been reported most commonly in German shepherds, but is also seen in Doberman pinschers, Rottweilers, and a number of other breeds.[29,57] The etiology is unknown, but muscle trauma is suspected in some cases. The onset is insidious in some cases, leading to the speculation of chronic or recurrent muscle strain as a cause. The most common clinical sign is nonpainful lameness, although pain may be elicited on palpation of the muscles. The gait is characterized by a shortened stride with internal rotation of the stifle during the swing phase, and the stifle joint may have decreased extension. The condition can be bilateral. A fibrous band may be palpated in the affected muscle.

Surgical excision of the affected tissue may cause temporary resolution of signs, but the fibrosis typically recurs within a few months if intensive rehabilitation for several months is not performed. Rehabilitation with ultrasound, PROM, cross-fiber friction massage, and controlled exercise for several months has resulted in improvement and acceptable clinical function.[29] If contracture begins to develop despite rehabilitation, a dynamic splint may be used to apply chronic, passive stretch to the tissues. Maintaining tissue tension for 8 to 12 hours per day may help delay the progress of contracture until the fibrous tissue matures in an elongated position.[3,29] Long-term monitoring and aggressive intervention may improve the chance of a successful outcome, but the prognosis is generally considered to be guarded.

Stifle Conditions

Articular Fractures of the Stifle

Fractures of the femoral condyles occur relatively rarely. Fractures of the patella may occur as a result of falling on the stifle on a hard surface, or as avulsion fractures of the quadriceps muscle. Physeal fractures of the proximal tibial physis sometimes occur. Salter-Harris Type III and IV fractures are more serious because the fracture line involves the articular surface. As with any articular fracture anatomic reduction, rigid fixation with screws and early, gentle, diligent ROM and joint mobilization are necessary for success. Surgical fixation of articular fractures must be strong enough to withstand early PROM exercises (Figure 32-27). Rehabilitation of fractures of the stifle is similar to that of fractures of the elbow (see Table 32-1).

Osteochondritis Dissecans of the Stifle

The presence of OCD in the stifle is uncommon. The lesions generally occur on the axial aspect of the lateral femoral condyle. It is usually treated by arthrotomy or arthroscopy to remove the osteochondral fragment(s), and to curette, forage, or micropick the subchondral bone

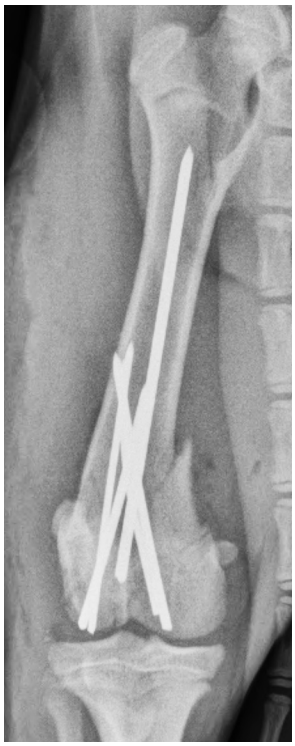


Figure 32-27 Postoperative view of an articular fracture of the distal femur. Consultation with the surgeon is recommended to determine the type and amount of exercise that can be safely done without jeopardizing the stability of the repair.

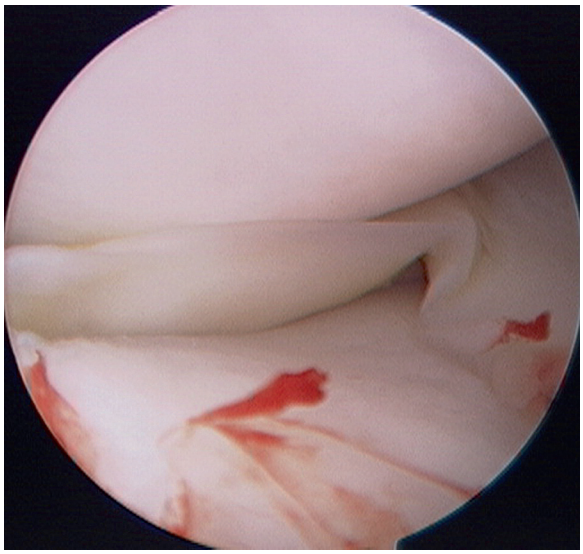


Figure 32-28 Arthroscopic view of the torn medial meniscus as it is shifted cranially within the joint. The caudal aspect of the medial meniscus is commonly damaged because of stifle instability as a result of cranial cruciate ligament rupture.

to create an opportunity for vascular ingrowth and fibrocartilage formation. Alternatively, osteochondral grafting may be an option. Nonsteroidal antiinflammatory drugs, cryotherapy, gentle PROM, and leash walks are advocated for the first 2 to 4 weeks, depending on the severity of the lesion. After that if the lameness is improving, leash walking is increased and more aggressive activities are added after 4 to 6 weeks (see Table 32-2).

Cranial Cruciate Ligament Rupture

Cranial cruciate ligament (CCL) rupture is one of the most common orthopedic conditions in dogs. Trauma-induced acute rupture of the CCL results in gross instability of the stifle, and is often accompanied by a sudden non–weight-bearing lameness, which improves gradually over the first few weeks. However, most cases of CCL rupture occur as a result of ligament degeneration. The animal may present with acute lameness or may have a more insidious lameness as partial rupture of the ligament occurs over time. In either scenario osteoarthritic changes progress with time and often result in diminished limb function.

Cranial cruciate ligament rupture is diagnosed by palpating cranial drawer motion (cranial subluxation of the tibia). Joint effusion may be evident and is best palpated adjacent to the patellar tendon. In more chronic cases drawer motion may be more difficult to elicit and the periarticular tissues may be thickened, particularly on the medial aspect of the stifle. This is termed *medial buttress*. Radiographs may be used to confirm the presence of joint effusion, assess the severity of any degenerative changes, and rule out other problems such as neoplasia.

Damage to the caudal pole of the medial meniscus often occurs as a result of joint instability (Figure 32-28). The lateral meniscus can also be damaged, but it is typically not as severely affected as the medial.[58] Damage to the meniscus rarely occurs as an isolated injury, but may be seen in approximately 45% of animals with CCL rupture.[59] In some cases the meniscus may be normal at the time of the initial surgery for CCL rupture, but become damaged at some time in the future. Clinical signs of a meniscal tear may include lameness and an audible click during joint motion. Partial or total meniscectomy via arthrotomy or arthroscopy is used to treat meniscal tear. If a meniscectomy is required the prognosis is generally somewhat worse than for stifles that have an intact meniscus. The rehabilitation program after meniscectomy is dictated by the procedure used to stabilize the joint. In some cases the meniscus is normal at the time of stifle stabilization but is damaged months or years later. This is probably the most common reason for an animal to develop acute lameness after CCL surgery.

Conservative treatment of CCL rupture by strict confinement for 4 to 8 weeks may be attempted, but the progression of OA is greater. An external brace may be used to support the stifle, but there are no studies to document their efficacy in dogs. Knee braces similar to those used in people after cruciate ligament repair may provide support and improve proprioception and limb function, but they do

Table 32-4 Rehabilitation After Surgical Repair of Ruptured Cranial Cruciate Ligament

Goals	Treatment
Control inflammation	Cryotherapy immediately after surgery and then daily until inflammation resolves, particularly after exercise
Maintain range of motion and joint health	Passive range of motion and/or joint mobilizations for 3 to 4 weeks. Consider aquatic walking. Add cavaletti walking after several weeks, if repair is stable enough.
Control pain	Nonsteroidal antiinflammatories ± other analgesics ± transcutaneous electrical nerve stimulation within first postoperative week. Decrease pain management as indicated by assessment of the dog.
Strengthen muscle	Consider neuromuscular electrical stimulation in early postoperative time to limit atrophy. Begin with controlled slow leash walking. Consider aquatic walking after incision has sealed. Progress to stair climbing and sit-to-stand exercises. Rate of progression is typically more conservative following tibial plateau leveling osteotomy as compared with an extracapsular suture. No explosive activity for 3 months.

not provide adequate stability and it is not clear that outcomes are improved.[60-63] Surgery is accepted to be the treatment of choice, regardless of dog size. Many surgical techniques have been described to treat cranial cruciate ligament rupture, but none have been proven to be superior.[65,66] Physical rehabilitation may be at least as important as surgical technique in affecting functional outcome.[67-69]

The prognosis for CCL rupture is variable. In humans a trend toward more aggressive rehabilitation has shown improved outcomes following surgery for cruciate ruptures.[70] Postoperative rehabilitation programs for CCL surgery in dogs improves joint ROM, reduces muscle spasms, and improves weight-bearing and overall joint function.[67,69] Dogs treated only with NMES after CCL surgery had improved lameness scores, increased thigh circumference, and decreased radiographic changes as compared with untreated dogs.[71] The focus of rehabilitation in most cases is on improving stifle extension and increasing mass of the quadriceps, biceps femoris, and semimembranosus muscles.[72] Some dogs recover with near normal function. However, some have moderate to severe OA at the time of diagnosis, requiring long-term management (see section on OA). Total stifle replacement surgery is available, but is not commonly performed.[73]

To simplify discussion of CCL rehabilitation, the surgical repairs are grouped into three categories. The surgical procedures have been traditionally classified as extracapsular or intracapsular, based the location of the implant or graft that is used to provide joint stability. A third approach is to change the joint biomechanics by performing some type of tibial osteotomy.

Extracapsular Stabilization

Extracapsular procedures stabilize the joint by transposition of the animal's own periarticular tissues or by securing a synthetic material (usually suture) external to the joint capsule. Most commonly, the suture is passed around the lateral (and sometimes medial) fabella and through a tunnel drilled into the proximal tibial crest. Modifications of this procedure involve stabilizing the suture with bone anchors or through bone tunnels. These procedures provide immediate extracapsular stabilization of the stifle, allowing early rehabilitation to be initiated. At some point, the prosthetic material may fatigue and break. The joint is stabilized by periarticular fibrosis within 8 to 10 weeks, so if suture breakdown occurs several months postoperatively joint stability is not significantly affected. Nevertheless, it is common for some degree of cranial drawer motion to be present weeks to months after surgery. The complication rate associated with this procedure is relatively low and most are incisional complications.[74]

Fibular head transposition is another type of extracapsular stabilization. The fibular head is transposed cranially and stabilized with a pin and tension band. The distal portion of the lateral collateral ligament inserts on the fibular head, so shifting it cranially causes it to function similar to a CCL, by limiting cranial drawer and internal rotation of the tibia. The repair provides immediate stability of the stifle joint, but some drawer motion may return with time.

Physical rehabilitation of extracapsular techniques begins in the immediate postoperative period with cryotherapy, NSAIDs, and PROM to reduce inflammation and pain (Table 32-4). The common practice of placing a compression bandage immediately postoperatively may do little to limit swelling of the joint.[75] Cold compression or microcurrent electrical therapy in combination with a compressive bandage may be effective in limiting postoperative edema.[76,77] Electrical stimulation (TENS) may help with pain management.[78] After loss of the cruciate, even with immediate surgery, the affected leg loses muscle mass within 2 weeks and bone density within 4 weeks.[69,79] It is

also important to establish normal ROM within 2 weeks to regain full stifle extension.[80] Physical rehabilitation can facilitate an increase in muscle mass after surgery.[69,72,80] Neuromuscular electrical stimulation (NMES) may also be initiated to limit muscle atrophy.[71] Controlled leash walks and active use of the limb are encouraged the day after surgery. Treadmill walking can be used to encourage weight bearing. Aquatic therapy can begin within 1 week postoperatively if the incision is sealed and has no drainage or discharge. Swimming or water walking may improve ROM, particularly stifle flexion.[38,81] Aquatic therapy, whether swimming or walking, also reduces the axial load on the joint,[39] thus potentially limiting stress on the surgical repair. Some animals may flail their limbs in water, so aquatic therapy could be counterproductive in those cases. Passive range of motion is continued with an emphasis on stifle extension. After the inflammation has subsided, topical hot packs or continuous ultrasound may be used to limit pain, increase blood flow, and increase tissue extensibility before the rehabilitation session. Cryotherapy may be performed at the end of each exercise session to limit inflammation and pain. Hindlimb muscles can be strengthened by stair climbing, incline or decline walking, sit-to-stand exercises, pulling a cart, or dancing. As strength and endurance improve, the duration and intensity of the exercises can increase to include jogging, controlled ball playing, and swimming. However, explosive activity such as jumping is not allowed for the first 3 months.

Intracapsular Stabilization

Intracapsular procedures replace the cranial cruciate ligament in a nearly anatomic position with a graft, synthetic material, or a combination of these materials. Biologic grafts are favored over synthetic prosthetics because the latter tend to degenerate and cause chronic intraarticular inflammation. Biologic grafts undergo a period of weakness until the tissue revascularizes and gains additional strength. Months may be required for the graft to regain sufficient strength to function as a normal CCL. When creating a therapeutic plan, the therapist must consider the material used for the surgery. Autografts and allografts are generally strong when initially placed, but stretch under tension. The tissue becomes weaker over the next 2 to 20 weeks while revascularization and incorporation occur. Ultimate tissue strength is achieved following biointegration of the graft. In general, the rehabilitation program is similar to, but much more gradual than, extracapsular repair, and should not progress as rapidly because of the limited graft strength.

Tibial Osteotomies

Several tibial osteotomy techniques have been described, but the most commonly used is the tibial plateau leveling osteotomy (TPLO). Other techniques include the tibial tuberosity advancement (TTA), closing wedge tibial osteotomy (CWTO), proximal tibial intraarticular osteotomy (PTIO), chevron wedge osteotomy, and triple tibial osteotomy (TTO). No one technique has been shown to be superior.[82] A variety of complications have been reported for TPLO and TTA procedures, but the overall complication rates are similar.[83] The theory behind the osteotomy techniques is to alter the joint biomechanics to eliminate abnormal cranial tibial thrust motion during weight-bearing activity. The TPLO procedure is performed by making an osteotomy in the proximal tibia, rotating the proximal tibia to level the weight bearing surface and change the joint biomechanics, and stabilizing it with a bone plate until the bone has healed (Figure 32-29). The TTA procedure is performed by making an osteotomy in the cranial aspect of the proximal tibia, allowing the tibial tuberosity to be transposed cranially and stabilized with a special wedge-shaped implant, thus shifting the alignment of the patellar tendon and eliminating cranial tibial thrust.

The most common complication following TPLO is implant-associated infection.[84] Patellar tendinitis is a common complication that is seen in the first postoperative month, and is identified by pain on palpation of the patellar tendon at its insertion on the tibial crest. The tendon is also be palpably thickened (Figure 32-30). Another complication is avulsion of a portion of the tibial crest. Fracture of the tibial crest is also a reported complication of TTA. Although dogs may become very functional after TPLO surgery, the limb may never be completely normal.[85]

The therapy of tibial osteotomy techniques is initially the same as for extracapsular techniques, and involves cryotherapy, NSAIDs, and PROM in the immediate postoperative period. Limited activity in the form of progressively longer leash walks, are begun in the initial postoperative period. Potential complications of patellar tendinopathy and tibial crest avulsion must be considered when developing the rehabilitation protocol. Excessive stress on the patellar tendon should be avoided in the early postoperative period until the tissues have a chance to adapt to the altered joint biomechanics. The quadriceps muscle-patellar tendon unit should be maintained in a relatively shortened position to reduce tensile forces on the tibial crest. Excessive flexion of the stifle during weight bearing should be avoided. This includes jumping, running, stair climbing, or walking in a crouched position. The therapist must carefully monitor the patellar tendon and tibial crest, and alter the treatment protocol if necessary. Early detection and treatment of complications should result in an earlier return to function and an improved outcome as compared with allowing the conditions to progress to the point of clinical lameness and pain. These complications usually can be managed by NSAIDs, cryotherapy, extracorporeal shock wave therapy, and more restricted activity. Use of a hinged knee brace to limit flexion of the stifle may be considered, although its efficacy has not been reported. After the tissues have healed,

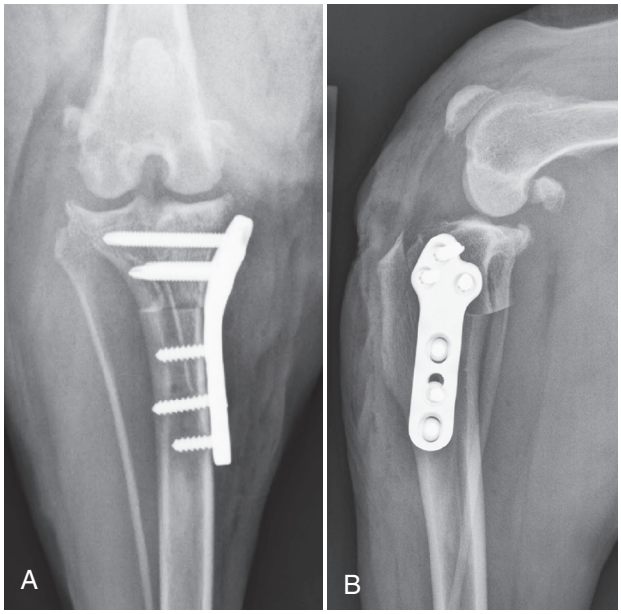


Figure 32-29 A dog with a cranial cruciate ligament injury has had the stifle stabilized with a tibial plateau leveling osteotomy. **A,** A-P view, **B,** Lateral view.



Figure 32-30 This dog became more lame several weeks after TPLO surgery. The increased soft tissue density in the cranial aspect of the joint is consistent with patellar tendonitis.

activity level can be gradually increased. The osteotomy site must be given adequate time to heal (6-10 weeks) to prevent complications related to bone healing and implant failure. As healing of the osteotomy site progresses, a gradually increasing protocol of limb use is prescribed. Aquatic therapy may be useful to reduce weight-bearing stresses on the repair.[39]

Caudal Cruciate Ligament Rupture

Isolated rupture of the caudal cruciate ligament is rare. The caudal cruciate ligament may rupture as a complication of TPLO overrotation, but it is usually caused by severe stifle trauma that results in stifle luxation. Treatment of isolated caudal cruciate ligament rupture is surgical stabilization with an extracapsular suture, and rehabilitation is carried out as for extracapsular stabilization of cranial cruciate ligament rupture. Treatment of caudal cruciate ligament ruptures that occurs in combination with damage to other stifle structures is discussed in the section on stifle luxation.

Stifle Luxation

Total derangement of the stifle, with rupture of both cruciate ligaments and one or both collateral ligaments, is a result of severe trauma. The menisci are often damaged as well. Collateral ligament trauma is usually best treated by surgical repair. The repair may involve suturing stretched or torn ligaments, or the use of prosthetic material (e.g., suture and screws or bone anchors) to replace the torn ligament(s). The cruciate ligaments are generally repaired with an extracapsular technique. If the damage was severe or injury was sustained to other limbs, there will be increased stress on the repaired stifle. The repair may be protected with a bandage or splint for the first 2 to 4 weeks. External coaptation and transarticular external fixation will cause at least a temporary decrease in ROM, but may be necessary.[86,87] Hinged external fixators allow joint flexion and extension. It is not clear whether the benefits of external fixation outweigh the potential complications. If the stifle is not immobilized, immediate PROM helps to prevent joint contracture, promote cartilage homeostasis, and stimulate scar formation along normal lines of stress, resulting in a stronger scar. If the stifle is immobilized postoperatively, the goal after bandage or brace removal is to eliminate joint stiffness and re-establish ROM by implementing passive and active ROM exercises.[87] Therapeutic ultrasound with simultaneous stretching also may be beneficial. Weight-bearing activities are encouraged with weight shifting, slow leash walks, and treadmill walking. Aquatic therapy may also be helpful. Appropriate challenges to the healing tissues enhance tissue remodeling and strengthening. Endurance and strengthening activities may be initiated between 4 and 6 weeks. Near full return to activities should be achieved by 12 to 16 weeks.

The prognosis for stifle luxation is fair. Long-term problems include decreased ROM, chronic instability, and ongoing OA. Arthrodesis is an option for a chronically unstable stifle joint, but function is poor. Activity should be restricted until there is radiographic evidence of fusion, which is usually in about 8 weeks. Increased

weight-bearing exercises and muscle reconditioning are begun after the stifle has fused (see General Physical Rehabilitation Management of Joint Arthrodesis).

Patella Luxation

Although patella luxations may be traumatic in origin, they are most commonly associated with torsional deformities and malalignment of the femur and tibia. Medial luxations are more common than lateral luxations in all dog breeds and in cats.[88] Concurrent cranial cruciate ligament rupture is present in 15% to 20% of middle-aged and older dogs with chronic patella luxation.[40] Patella luxations are classified from grade I to grade IV, with IV being the most severe. Grade I patella luxation indicates that the patella stays in the trochlear groove but can be manually luxated, whereas grade IV indicates a patella that is always luxated and cannot be reduced. The higher grades usually correlate with more severe clinical signs and severity of bony abnormalities.

Surgery is only recommended when the patella luxation causes gait abnormalities or lameness; therefore surgery is generally not performed for grade I luxations. Surgery is only recommended for grade II luxations if clinical signs are causing limb dysfunction, but is usually indicated for grade III and IV luxations. Surgical correction of patella luxations usually requires reconstruction of both soft tissues and bone. In almost all cases the tibial crest is transposed and pinned to realign the quadriceps mechanism (Figure 32-31). Some type of trochleoplasty to deepen the trochlear groove is performed if the trochlear groove is shallow. A femoral osteotomy may be performed if severe varus deformity of the femur is present. The most common complications of patella luxation repair are patella reluxation and implant loosening or failure.[89] Other reported complications include trochlear wedge migration, wound dehiscence, tibial tuberosity fracture, and septic arthritis. After surgery the tissues must be allowed to heal before vigorous rehabilitation begins, particularly if the tibial crest was transposed.

During the first few weeks cryotherapy and NSAIDs may be used for inflammation (Table 32-5). Active use of the limb is encouraged, but initial activity should be limited to short leash walks, and no running, jumping, or playing are allowed. Passive ROM is beneficial for joint resurfacing and cartilage healing, especially if a trochleoplasty was performed.[90] Small breed dogs in particular are sometimes reluctant to bear weight on the affected limb, even in the absence of apparent pain or complications. In these cases weight-shifting activities or a syringe cap placed under a

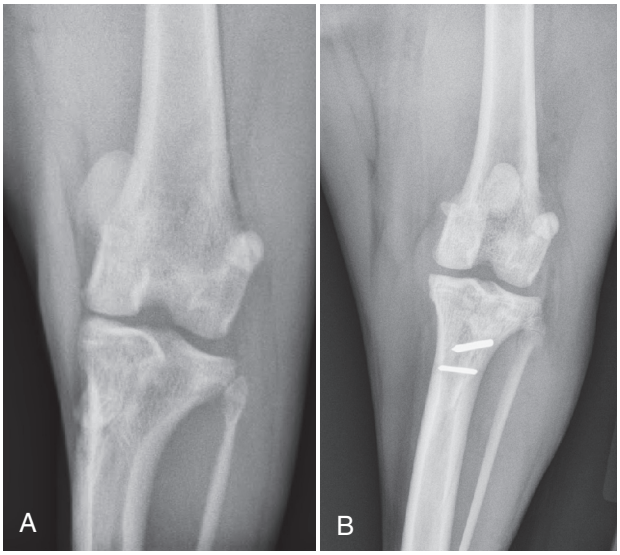


Figure 32-31 Medial luxation of the patella. (*A*) is the preoperative radiograph and (*B*) is the postoperative radiograph with pins present in the transposed tibial crest.

Table 32-5 Rehabilitation After Surgical Repair of Patella Luxation

Goals	Treatment
Control inflammation	Cryotherapy immediately after surgery and then daily until inflammation resolves, particularly after exercise
Maintain range of motion and joint health	Passive range of motion for 3 to 4 weeks. Consider aquatic walking.
Control pain	Nonsteroidal antiinflammatories ± other analgesics as needed after surgery. Decrease pain management as indicated by assessment of the dog.
Strengthen muscle	Initially, activities such as weight shifting to encourage weight bearing. Start with controlled leash walks. Limit activities that may put rotational stress on the stifle. Motion should be confined to the sagittal plane. After 2-3 weeks, focus on increased strength of hindlimb with walking, incline walking, sit-to-stands, dancing, etc.

normal foot may be instituted to encourage limb use. Walking on an underwater treadmill may encourage strengthening of the quadriceps muscles while maintaining the motion in a sagittal plane. After several weeks strengthening exercises may be initiated. Motion should be limited to the sagittal plane to avoid undue stress on the repair. The prognosis is generally fair to good for grades II and III, but guarded for grade IV luxations.

Rupture of the Patellar Tendon

Rupture of the patellar tendon is rare, but can occur with direct trauma or with forceful quadriceps contraction during forced flexion of the stifle. Patellar tendon rupture is treated by surgical tendon repair. A transarticular external fixator or splint may be placed for 2 to 3 weeks in an effort to limit tension on the repaired tendon. However, this will not prevent tension created by contraction of the quadriceps muscles. Superficial cold therapy is used in the postoperative period to reduce local inflammation. Pulsed 3-MHz therapeutic ultrasound applied directly over the tendon is appropriate to help stimulate collagen production. Therapeutic laser may also be beneficial. Therapy may include PROM and light activity such as leash walking to maintain normal tissue length and encourage normal joint movement. Standing and balancing exercises can promote proprioceptive input. However, active stifle motion should be limited to prevent disruption of the weakened tissue. Between 3 and 6 weeks controlled exercise should be initiated. The tendon gains significant strength by 6 weeks, but will not reach maximal strength for a year or more.[91] During the remodeling phase therapy is directed at maintaining tissue mobility, preventing adhesions, and improving proprioception and muscle strength.

Traumatic Injuries of the Long Digital Extensor Tendon

Avulsion of the tendon of the long digital extensor muscle is a rare condition that occurs in young dogs. Clinical signs include joint effusion and pain in the craniolateral aspect of the joint. If not treated OA and chronic low-grade lameness may result. Treatment involves reattachment of the avulsed fragment of bone, or removing the fragment and attaching the tendon to the proximal tibia or joint capsule. If the bony fragment is reattached, time is allowed for union before starting aggressive rehabilitation. In the initial postoperative period, pain and inflammation are treated with NSAIDs and cryotherapy. Limited weight bearing with muscle re-education and proprioceptive training are instituted as the fracture heals. If the fragment was excised and the tendon reattached, weight bearing, controlled activity can be started soon after surgery. The prognosis is good if surgical treatment is performed before OA is apparent.[92]

Crural Conditions

Avulsion of the Tibial Tuberosity

This condition most often occurs in immature dogs before the crest fuses with the main shaft of the tibia. Avulsion results in proximal and cranial displacement of distal portion of the tibial crest, or of the entire crest. This results in patella alta and the dog is unable to fully extend the stifle. This fracture is best treated with a pin and tension band technique to reattach the bony fragment, although minimally displaced fractures may be treated conservatively with rest and a lateral splint to limit stifle flexion. Cryotherapy, NSAIDs, and limited weight bearing are initiated after surgery. Follow-up radiographs are made 3 to 4 weeks after surgery to monitor the progress of fracture healing and guide the initiation of a more aggressive weight-bearing and muscle reeducation program.

Tibial and Fibular Fractures

Fractures of the tibia and fibula are common in the dog. Depending on the fracture type and configuration, they may be treated with closed reduction and external coaptation, or open reduction with internal or external fixation. Nonsteroidal antiinflammatory drugs, cryotherapy, and PROM are used to decrease pain and inflammation and maintain ROM for the first 2 to 3 weeks. Massage may be performed initially to help resolve edema. Active weight-bearing exercises are then initiated. The rate and progression of activity are dictated by the severity of the fracture and stability of repair. Because relatively little soft tissue covers the medial surface of the tibia, open fractures are quite common. In these cases, wound management and bandaging also may be necessary. Osteomyelitis tends to occur in the tibia more commonly than other bones and is probably related to the high incidence of open fractures. Aquatic therapy is contraindicated in patients with open wounds.

Tarsal, Metatarsal, and Phalangeal Conditions

Tarsal, Metatarsal, and Phalangeal Fractures

Fractures of the distal fibula, tibia, or tarsal bones may occur as a result of trauma, such as athletic injury or automobile accidents. Repair of medial or lateral fractures of the malleolus are important because these serve as the origins of the collateral ligaments. Metatarsal and phalangeal fractures usually occur as a result of trauma, and phalangeal fractures are often open fractures. Tarsal, metatarsal, and phalangeal fractures may be treated using closed reduction and immobilization in a coaptation device, or with a variety of implants, including intramedullary pins, wires, and plates and screws. A splint or light cast is usually

applied to the limb for several weeks after internal fixation to protect the relatively small implants that must be used.

As is the case for all articular fractures, it is important to achieve anatomic reduction and rigid fixation with screws, pins, or small bone plates. Also, early ROM is important following surgery. If a cast or splint is used, stifle ROM can be preserved while the external coaptation is in place. If possible, the cast or splint should be removed on a weekly basis to enable PROM exercises to be performed to maintain joint and tendon mobility. After performing PROM exercises, the coaptation is reapplied to protect the fracture. Following permanent removal of the splint or cast, remobilization of the joints is important, along with weight-bearing therapeutic exercises and muscle reconditioning. Sagittal-plane ROM exercises and joint mobilization work well to restore ROM to the area.

Osteochondritis Dissecans of the Tarsus

Tarsal OCD occurs less commonly than OCD in the shoulder. Lesions can occur on the medial or lateral ridge of the talus, and can be difficult to see radiographically and may be more readily diagnosed by CT or arthroscopy. The pathophysiology is the same as for OCD in other joints. Dogs with hock OCD may present with signs of OA such as joint effusion, thickening of periarticular soft tissues, decreased ROM, and crepitus. It is usually treated by arthrotomy or arthroscopy to remove the osteochondral fragment(s), and curette, forage, or micropick of the subchondral bone to allow vascular ingrowth and fibrocartilage formation. Large cartilage flaps in the hock are sometimes fixed in place in an effort to preserve joint congruency, but OA will progress. Nonsteroidal antiinflammatory drugs, cryotherapy, gentle PROM, and leash walks are advocated for the first 2 to 4 weeks, depending on the severity of the lesion. After that if the lameness is improving, leash walking is increased and more aggressive activities are added after 4 to 6 weeks. The prognosis for hock OCD is guarded because OA progresses despite treatment (see Table 32-2). In severe cases arthrodesis may be performed.

Tarsal Luxation

Most luxations of the tarsocrural joint occur with concomitant avulsion of one or both malleoli, which is where the collateral ligaments originate. Subluxation can occur with rupture or avulsion of the collateral ligaments. Traumatic shearing injuries commonly result in loss of skin, subcutaneous tissues, tendons, ligament, and bone from the medial or lateral aspect of the joint. Depending on the type of damage, surgical repair of hock luxation or subluxation may include imbricating, suturing, reattaching the ligaments, or replacing the ligaments with a prosthetic collateral ligament technique. In some cases an external fixator may be applied as the primary repair.[93] Open wounds are often present, which must be dressed or surgically repaired. If there is significant skin loss, a graft may be required after the wound is healthy. Ligament repairs are typically protected during healing by rigid immobilization in a cast, splint, or external fixator for at least 3 weeks. This prevents the sutures from tearing out and prevents elongation of the ligamentous fibers. Ligament repairs are generally not able to withstand full weight-bearing stresses for several weeks. However, in joints rigidly immobilized for 6 weeks, the ligaments fail to gain as much strength as ligaments of joints that are not immobilized beyond 3 weeks.[91] A hinged transarticular external fixation will allow some joint motion while protecting the primary repair, but it is not known whether the long-term outcome is improved over joints that are rigidly immobilized.[87] After the cast or external fixator is removed, remobilization of adjacent joints, weight-bearing exercises, and muscle reconditioning are begun. Active or passive therapeutic exercises that place varus or valgus stresses on the collateral ligaments should be avoided.

Arthrodesis may be indicated for shearing injuries if the degree of tissue loss, especially articular cartilage, makes joint reconstruction impossible. Hyperextension injuries that result in subluxation or luxation of the tarsometatarsal joint, or the proximal or distal intertarsal joints also require partial arthrodesis for a good outcome.[40] Arthrodesis is performed by application of internal or external fixation (Figure 32-32). If internal fixation is used, the distal limb is supported by a splint or cast for 6 to 8 weeks or until there is radiographic evidence of fusion. Remobilization of adjacent joints, weight-bearing exercises, and muscle reconditioning are begun after the cast has been removed (see General Physical Rehabilitation Management of Joint Arthrodesis).



Figure 32-32 Pantarsal arthrodesis as a treatment for a severe shearing injury of the hock.

Injuries of the Common Calcaneal (Achilles) Tendon

The common calcaneal (Achilles) tendon consists of three tendons that insert on the tuber calcanei: the gastrocnemius; the common tendon of the biceps femoris, semitendinosus, and gracilis muscles; and the tendon of the superficial digital flexor muscle. Rupture of the common calcaneal tendon can occur at the musculotendinous junction, the midsubstance, near the tendon's insertion on the tuber calcanei, or the tuber calcanei may fracture. The tendon may be injured by sharp penetrating trauma or chronic repetitive use. If the tendon is avulsed, the end may be palpable 2 to 3 cm proximal to the tuber calcanei. The distal end of the tendon becomes enlarged and firm as fibrous tissue develops. Diagnosis is made by physical examination, ultrasound or other imaging, such as radiographs to identify avulsion fractures or other conditions. An avulsed tendon may be sutured to bone tunnels in the calcaneus. A large bone avulsion fragment may be fixed with pin and tension band. In cases of ruptures at the musculocutaneous junction or in mid-tendon, the tendon is usually sutured primarily. Partial tendon ruptures may be managed by a cast or splint.

After tendon surgery, the hock is often immobilized in a normal standing angle to prevent hock flexion and excessive tension on the repair (Figure 32-33). However, the tendon will still be subjected to some tension from muscle contractions.[94] Therefore placing a walking ring under the foot and incorporating this into a transarticular external fixator or cast will allow weight-bearing forces to bypass the healing tendon. Three or more weeks of immobilization with a splint, cast, positional screw, or external fixator is typical. The walking ring may then be gradually raised, so that partial weight bearing is shared between the ring and foot, until complete weight bearing by the foot occurs. Some studies question which immobilization technique is superior.[94,95] Rigid fixation should be used for the shortest time necessary. Early partial weight bearing and joint movement create stress on the reconstructed tissue to promote parallel collagen alignment in the repaired tendon and increased early tendon strength.[96,97] External skeletal fixators may be applied with a hinge to allow limited motion. External fixators may also provide progressive loading of the tendon by performing a staged removal.[97] After 3 weeks controlled motion is recommended to promote remodeling of the peritendinous scar, which allows the tendon to glide. By 6 weeks the tendon has not attained full strength but should be strong enough to support partial weight.[91] The tendon may continue to slowly increase in strength for a year or more. During the remodeling phase therapy is directed at maintaining tissue mobility, preventing adhesions, and improving proprioception and muscle strength.



Figure 32-33 A transarticular external fixator is sometimes used to stabilize a repaired common calcaneal tendon.

Pulsed, 3-MHz therapeutic ultrasound may be used to stimulate collagen repair.[98] Therapeutic laser also may be beneficial. After the period of immobilization, continuous ultrasound or hot packs may be used to warm the tissues before stretching.[99] Tendon strength and joint flexion may improve over several months to a year. During this time explosive motion that places large tensile forces on the tendon (e.g., jumping) should be avoided. The long-term prognosis is good.

Luxation of the Superficial Digital Flexor Tendon

The tendon of the superficial digital flexor muscle may luxate medially or laterally from its location as it crosses the calcaneus. It is a traumatic injury, but dysplasia of the tuber calcanei may be a predisposing factor in some cases.[100] Luxation of the tendon may be palpated while the hock is flexed and extended. Treatment is surgical repair of the torn retinaculum. The healing tissues are protected by immobilization of the hock in a splint for 2 to 3 weeks. After the splint has been removed, ROM and weight-bearing exercises are begun. Tissue mobilization may be applied to the area to prevent fibrosis of the tendon, which could limit motion of the digits. The prognosis is good if the repair is done early. Chronic tendinitis and bursitis may decrease the prognosis.

Luxation of the Metatarsophalangeal and Phalangeal Joints

These conditions are the same as described for the front feet.

Table 32-6 Rehabilitation of Joints with Osteoarthritis

Goals	Treatment
Weight management	Dietary counseling and exercise program to achieve and maintain lean body weight.
Pain management	Nonsteroidal antiinflammatories ± other analgesics as needed
	± Transcutaneous electrical nerve stimulation
	± Laser
	± Shockwave therapy
	Hot or cold pack of joint
	Hot pack, therapeutic ultrasound, or massage for muscle spasms
Strengthen periarticular muscles	Neuromuscular electrical stimulation if too weak or painful for active exercise
	Controlled, low-impact exercise (walking or aquatic exercise). Ongoing patient assessments of lameness and pain are used to continually adjust the intensity of the exercise program.
Limit excessive joint stress	Lifestyle changes, such as ramps instead of steps or jumping
	Encourage controlled, low-impact activities.
Maintain or improve joint range of motion and proprioception	Passive range of motion and/or joint mobilization
	Active range of motion—therapeutic exercise (walking or aquatic)
Maintain or improve cartilage health	Passive range of motion
	Weight-bearing exercise with low-impact (walking or aquatic)
	Disease modifying osteoarthritic agents
Limit inflammation	Nonsteroidal antiinflammatories to treat chronic or acute inflammation
	Cryotherapy for episodes of acute inflammation
	Limit high-impact or uncontrolled activities
	Avoid obesity
	Diet containing omega-3 fatty acids, or fish oil supplement

Trauma of the Superficial and Deep Digital Flexor Tendons

These conditions are the same as described for the front feet.

General Physical Rehabilitation Management of Osteoarthritis

Osteoarthritis may also be termed *osteoarthrosis*, *degenerative joint disease* (DJD), or *spondylosis* if in the spine. It is a deterioration of joint health that usually occurs secondary to joint incongruity, instability, or some other disruption of the articular cartilage. Mechanical and biochemical changes that occur within the joint result in decreased cartilage resiliency, cartilage thinning, subchondral sclerosis, synovitis, and osteophyte formation. Clinical signs include joint pain, crepitus, and stiffness. Normal ROM may be lost because of joint surface incongruity, muscle spasm and contracture, periarticular fibrosis, or mechanical block from osteophytes or joint mice. The joint(s) may be enlarged because of synovitis, synovial effusion, osteophytes, or periarticular fibrosis. The pain and stiffness may lead to lameness or decreased activity, with a resultant loss of muscle mass and strength.

The goals of treating OA are to manage pain, maintain function and ROM, and maintain or regain normal activity (Table 32-6). These goals are primarily accomplished by means of weight management, therapeutic exercise, physical modalities, and medications.[101-103] Weight reduction is essential for obese animals, and a lean body weight is maintained by counseling the owners on proper diet and exercise. Weight reduction alone can cause a significant improvement in clinical signs in dogs with hip dysplasia that are more than 10% above their ideal body weight.[102] Excess weight can cause increased mechanical stresses on load-bearing joints. In addition, body fat has metabolic activity that promotes inflammatory reactions within the body, which may contribute to the pathophysiology of OA.

A controlled, low-impact exercise program alone improves pain and overall function scores in geriatric dogs with OA.[103] Regularly performed, low-impact exercises such as leash walking, walking in water, or swimming, help to maintain supporting muscle strength and joint function while minimizing joint stresses. Bursts of vigorous activity, such as running or jumping, may exacerbate inflammation and should be minimized. Certain activities may be used to target specific muscle groups. For example, repeated sit-to-stand exercises or walking up stairs may

help strengthen the hindlimbs. Exercise sessions should be challenging, but should not be followed by any increase in pain. The exercise program must be tailored to the individual and adjusted to account for the fluctuations in clinical signs that are typically seen with OA.

Other techniques may be used to enhance the exercise program. Use of a walking cart may be indicated in some cases with severe, chronic OA to allow independent function and reduce the risk of falling. In cases of severe muscle loss and weakness, neuromuscular electrical stimulation (NMES) may be used for muscle strengthening. Passive range of motion (PROM) and active range of motion (AROM) exercises are used to improve joint ROM and promote cartilage metabolism and diffusion of nutrients. Swimming can be used to improve ROM, particularly joint flexion.[81] Walking up down hills or stairs may also promote AROM of the joints.[104] Passive stretching alone can improve ROM in dogs with OA.[105] Joint mobilization may also help improve joint motion, cartilage health, and manage pain. Joint compressions, joint mobilizations, PROM, and AROM may be used to improve joint proprioception.

Physical modalities may be incorporated into the therapeutic program. Hot packs, therapeutic ultrasound, or massage may be useful to reduce pain associated with muscle spasms and decrease joint stiffness. These modalities can also be used to increase blood supply to the muscle in preparation for an exercise session. Transcutaneous electrical nerve stimulation (TENS) has been shown to be of benefit in dogs with osteoarthritic pain.[78] Laser therapy has been used to treat OA, although there is little evidence to support its effectiveness. Extracorporeal shockwave therapy also shows promise as a method to decrease pain in dogs with OA, however, the number of cases reported is limited.[106-109] Cryotherapy can be used to reduce acute inflammatory episodes associated with overuse or postexercise inflammation.

A number of drugs and other substances are promoted for the treatment of OA. Nonsteroidal antiinflammatory drugs may be used as needed for pain management and to treat chronic inflammation or episodes of acute inflammation. It is advisable to maintain the lowest effective dose, to minimize the risks of adverse side effects. The efficacy of NSAIDs varies between individuals, so if the animal is not responding well to one NSAID another may be more effective.[110] It is advisable to stop one NSAID completely before starting another because of the potential for adverse side effects. Additional analgesics such as gabapentin or amantadine may be needed in severely affected animals. Although corticosteroids have an excellent antiinflammatory effect, they also promote degenerative changes in the joint and should be avoided for chronic use.[111]

Slow-acting disease-modifying osteoarthritic agents (DMOAs), or chondroprotective agents, have the potential to improve joint health by stimulating cartilage metabolism, stimulating hyaluronan synthesis, inhibiting periarticular fibrin formation, and inhibiting catabolic enzymes. Some of these agents may also function as antiinflammatory agents or free radical scavengers. Injectable hyaluronic acid or a polysulfated glycosaminoglycan (Adequan) may be of benefit for some arthritic animals.[112,113] Several oral formulations of DMOAs have been developed. Oral glucosamine hydrochloride, chondroitin sulfate, avocado-soybean unsaponifiables, and manganese ascorbate may have a synergistic effect in dogs to reduce inflammation and lameness.[114] S-adenosylmethionine (SAMe) appears to be effective in humans with OA, but effectiveness in dogs has not been confirmed.[115] The oral forms of DMOAs, known as nutraceuticals, are not regulated as drugs, so there may be considerable variation in product content and quality. There are a number of compounds that are marketed as being effective in the treatment of OA, but have not been proved to be effective by scientific studies. Although the efficacy of most DMOAs is still unclear, the risk of adverse side effects appears to be low. Supplementation of dog food with fish oil, or feeding a diet that contains increased levels of omega-3 fatty acids can alleviate signs of OA.[116-118] Adipose-derived stem cell therapy shows some promise as an adjunctive treatment to improve the clinical signs of OA.[119]

Lifestyle modifications may be beneficial for some animals with OA. They should be housed in a warm, dry area with good footing and provided with a padded area for sleeping. Stair climbing and jumping may be minimized by the use of ramps.

The animal should be assessed routinely to alter the rehabilitation program as needed. Functional assessments should be made. Radiographs are of minimal use because they correlate poorly with limb function.[120]

Joint replacement surgery is performed in a few major canine joints. The usual indication is to treat severe OA that cannot be adequately managed medically. Total elbow and stifle replacement surgery is still in its infancy and is not commonly performed.[41,73] By contrast, total hip replacement is commonly performed in surgical practices for coxofemoral OA, articular fractures, or hip luxation. The complication rate for total hip replacement is reported to be as high as 17%, depending on the system being used and the experience of the surgeon.[121,122] However, most dogs have a successful final outcome.

General Physical Rehabilitation Management of Excision Arthroplasty

Excision arthroplasty involves the surgical removal of part of the joint, allowing a pseudoarthrosis (false joint) to form from fibrous tissue. This technique is generally limited to the shoulder and hip joints. It is done as a treatment for severe OA, irreparable fractures involving the joint, severe or recurrent joint luxation, or congenital joint deformities.

Elimination of the joint and the bony contact relieves the joint pain. The goal is for fibrous tissue to create a pseudoarthrosis with no bony contact, which provides pain-free function. However, normal function and gait are not expected due to the biomechanical changes. After excision arthroplasty early active use of the limb is encouraged to prevent excessive fibrosis and loss of motion of the pseudoarthrosis. Adequate analgesia throughout the rehabilitation program is a key factor for a successful outcome. Opioids may need to be combined with NSAIDs for pain control. Cryotherapy can also help reduce pain and early inflammation. Passive range of motion exercises are begun during anesthetic recovery and continued daily until the animal is using the limb well. Assisted weight bearing activity is initiated the day after surgery. Once the acute inflammation has subsided, hot packs or therapeutic ultrasound are used to facilitate PROM and stretching. Massage may also be helpful before passive or active exercises. In some cases weight shift exercises are used in the early rehabilitation period to encourage weight bearing. Active weight-bearing activities are progressed to higher levels as tolerated by the animal, to improve limb use and muscle strength.

Femoral head and neck ostectomy (FHO) is the most common excision arthroplasty (see Figure 32-20, *B*). After FHO the femur tends to be located more dorsally than normal so there is a functional shortening of the limb, which can cause gait abnormalities. In addition the hindquarters may appear to be asymmetric. Rehabilitation after FHO follows the same guidelines as for any excision arthroplasty, but there is an emphasis on regaining hip extension (Table 32-7). In addition to PROM for hip extension, ground treadmill or underwater treadmill exercises may be used to promote active hip motion. Sit-to-stand exercises may be used soon after surgery to strengthen hindlimb muscles, without encountering the pain associated with full hip extension. Walking on an inclined treadmill may promote active hip motion, although the inclination may need to be greater than 5% to induce much increase in hip ROM.[51] Walking up hills or steps can be initiated to promote hip extension[104] and improve muscle strength after the animal is ambulating well. Dancing exercises encourage muscle strengthening and hip extension, especially dancing backward. Although swimming is a good conditioning exercise, it does not promote hip extension.[81] Walking in an underwater treadmill may promote more functional activity if the water level is at the stifle or lower. Cryotherapy may be used at the end of each therapy session to reduce inflammation. Most animals will toe-touch consistently within 1 to 2 weeks, partially weight bear in 3 weeks, and be actively using the leg by 4 weeks. The animal should regain near-normal walking and trotting gaits, but full ROM is rarely achieved, with hip extension being the most limited. Prognosis for return to daily function is generally good but varies with the surgical technique and the chronicity of the preexisting lameness.[123,124] Large animals tend to have more difficulty with recovery than small animals, and obese animals tend to have more difficulty than athletic animals. Adherence to a rehabilitation program may also affect the outcome, so it is important to manage pain during the rehabilitation period to facilitate animal compliance.

General Physical Rehabilitation Management of Joint Arthrodesis

Arthrodesis is the surgical fusion of a joint that is done to salvage limb function when there is severe joint damage or in certain cases of nerve damage to a distal limb. To create bony fusion the joint must be rigidly immobilized after removing the articular cartilage and placing a bone graft. The joint is usually fused in a functional standing angle (see Figures 32-13 and 32-32). Goniometric measurement

Table 32-7 Rehabilitation After Femoral Head and Neck Ostectomy

Goals	Treatment
Control inflammation	Cryotherapy immediately after surgery and then daily until inflammation resolves, particularly after exercise
Maintain range of motion	Passive range of motion from immediately postoperatively until using the limb well. After the inflammation of surgery has subsided, daily hot packs or ultrasound in conjunction with stretching. Particular attention is paid to hip extension and also abduction. Incorporate dancing and incline walking into exercise program after the first week or two.
Control pain	Nonsteroidal antiinflammatories and additional analgesic medication for first the few weeks, as needed. Modalities as needed.
Regain limb function	Encourage weight bearing the day after surgery. Consider neuromuscular electrical stimulation. Begin with controlled slow leash walking and sit to stand exercises. Walk in aquatic treadmill initially to decrease weight bearing on the limb.

of the contralateral joint may be used as a guide if this limb is normal. The joint is most commonly stabilized with a bone plate, but pins, screws, or an external fixator may be used. Arthrodesis of joints that are located more distally on the limb are associated with better limb function and prognosis than arthrodesis of more proximal joints. Arthrodesis is performed most commonly on the carpal and tarsal joints.

Because of the massive inflammation and edema that can result from the surgical procedure, a pressure bandage is maintained during the immediate postoperative period. A cold-compression bandage or continuously circulating cold pressure wrap is preferred. Nonsteroidal antiinflammatory drugs are also used to minimize the inflammatory reaction. The repair is often supported by external coaptation (cast or splint) if internal fixation was used. This rigid support is usually applied in 3 to 5 days, after the edema and swelling have resolved. The external coaptation is usually maintained for 6 to 8 weeks or until there are radiographic signs of fusion. During this time, PROM or joint mobilization may be performed on adjacent joints. Muscle strengthening exercises may be instituted. Although other joints in the limb can compensate to some degree, the gait will never be normal. As the arthrodesis heals, gait training should be performed to restore the gait to the most normal gait pattern possible.

General Physical Rehabilitation Management of External Fixators

Rehabilitation is very important in patients with external fixators. During surgical planning the surgeon should place pins through safe corridors, without crossing large muscle masses, neurovascular bundles, or entering joints. A pin inadvertently placed through a large muscle mass (as can occur with fixation of the femur) or major tendon (e.g., Achilles tendon), may cause enough pain or mechanical difficulty that the patient will not tolerate ROM exercises or be willing to bear weight on the limb. It may be necessary to penetrate some muscle mass, so analgesia and careful attention to pin tract maintenance is important. The frame itself can present a problem in that it may be difficult to apply cryotherapy. Some clinicians apply ice packs to the frame itself, allowing it to act as a thermal conductor.

Passive range of motion, stretching, and massage can be applied to the soft tissues associated with an external fixator. Patients with external fixators may be reluctant to fully flex and extend the joints in the initial postoperative period. Active exercises that encourage limb flexion include walking over cavaletti rails or other objects, and walking on a land treadmill. Fixators are often used because there are open wounds that make internal fixation less desirable, and patients with open wounds should not be exercised in an aquatic environment.

If a fixator must cross a joint, but is not intended to fuse the joint, it is preferable to incorporate hinges in the fixator frame. The hinges can be locked into a rigid position for the animal's daily function. However, the hinges can be unlocked daily to allow the therapist to perform PROM on the immobilized joint.

General Physical Rehabilitation Management of Amputation

Limb amputation may be performed as a treatment for neoplasia or for severe soft tissue, orthopedic, or neurologic disorders. Dogs and cats typically do well after forelimb or hindlimb amputation. In many cases, the animal is already ambulating on three legs as a result of the condition of the affected limb. The other limbs should be thoroughly evaluated for the presence of orthopedic or neurologic conditions. If desired the affected limb may be placed in a sling before surgery to allow functional assessment of the remaining limbs. Some animals can adapt to amputation of more than one limb. They may require the assistance of a custom cart or prostheses, but some animals learn to walk with two legs (Figure 32-34).

The animal should be encouraged to stand on the first postoperative day, but may need supportive assistance. Therapeutic exercise may begin with standing and walking, and progress to more challenging activities that help the animal adapt to the new center of gravity. Uneven terrain or balance boards may be used to improve limb strength and proprioception. Most animals do well after amputation, whether it is a forelimb or hindlimb.[125] Animals that are overweight or have impaired function of other limbs may have more difficulty adapting to an amputation, especially forelimb amputation.



Figure 32-34 Double amputees can sometimes learn to walk on two legs. However, use of a cart allows more normal body posture, and may help prevent compensatory problems.

Historically limb prostheses have been used rarely in companion animals. This is partly owing to the fact that animals quickly adapt to walking with three legs. It may be argued that a three-legged quadruped will develop compensatory problems, but the average pet-quality animal functions with no obvious clinical impairments. In recent years there has been more demand for animal prostheses to replace the distal extremities. Most prostheses are designed with a sleeve that is custom fit to the residual limb. It is critical that the prosthesis fit appropriately and that keen attention is paid to skin contact sites on a twice-daily basis to help prevent skin breakdown and wounds. It is vital that an experienced animal prosthetist be involved with construction of a prosthetic. It is also usually necessary to remove the prosthesis during periods of rest to help prevent excess stress on tissues. Despite these precautions, wounds over the stump are common and require diligent care. Another method of creating a prosthesis involves surgical implantation of a prosthetic device into the bone of a limb. Bone grows into the porous metal of the prosthesis to form a more stable, osseointegrated prosthesis. There have been a few case reports involving the use of an osseointegrated prosthesis for the tibia.[126,127] Common complications include infection, which generally necessitates removal of the prosthesis.

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33 Common Conditions and Physical Rehabilitation of the Athletic Patient

Sherman O. Canapp Jr. and Deborah Gross Saunders

The diagnosis and treatment of common conditions of athletic and sporting dogs are discussed in this chapter. Most of these conditions involve injuries of the soft tissues such as muscles, tendons, or ligaments. Bone and cartilage can also be involved (e.g. subluxations or traumatic fragmentation of the medial coronoid process.

Muscle Strains

Muscle strains are the most frequent injury in human sports and can be minor, with tearing of a few muscle fibers with pain and local spasm, to complete muscle rupture.[1] Acute muscle injuries rarely have been reported in the canine veterinary literature, and discussion of chronic muscle disorders in dogs is limited to a handful of classic syndromes or inflammatory conditions. However, the reports of muscle strains in human sports medicine are bountiful and well documented in terms of evaluation and treatment.[2-4] It seems likely, given the similarities of the human and canine musculoskeletal system, injuries common in the human athlete would be common in their canine athlete counterpart. The low reported prevalence of muscle injury in dogs is probably owing to a failure to diagnose the condition. Inflammation and edema play a large role in muscle strains. Supportive care to treat the inflammation and swelling is often provided; however, in most cases little thought is given to the damage done to the involved muscles and their subsequent rehabilitation. The strength of muscle contraction may be greatly affected by even a minor strain injury. Some muscle fibers heal, but normal microscopic structure is not restored and scar tissue persists.[1] Healing of the muscle by scar tissue predisposes it to reinjury and possibly to muscle contracture or permanent shortening.[1]

Regenerative Medicine Therapy for Musculoskeletal Injury

Regenerative medicine therapy for treatment of injuries and defects is not a new concept. However, its application for musculoskeletal injuries has received significant attention in recent years because of its use in several prominent sports figures. In many human sports medicine clinics, regenerative medicine therapies are offered as "off-label" treatments for injuries involving muscles, tendons and ligaments. Regenerative medicine has been used for several years to treat these types of injuries in horses and more recently in dogs.

Regenerative medicine therapy encompasses several therapies, including platelet-rich plasma (PRP) and stem cell therapy (SCT). These therapies aim to induce healing through self- regeneration rather than scar tissue formation.

Tissue repair is achieved through a complex response predominately mediated by growth factors and cytokines. PRP, an autologous blood concentrate of platelets above the physiological baseline, contains bioactive proteins and several growth factors including:

Platelet-derived growth factor (PDGF)
Transforming growth factor-B1 (TGF-B1)
Transforming growth factor-B2 (TGF-B2)
Vascular endothelial growth factor (VEGF)
Basic fibroblastic growth factor (bFGF)
Epidermal growth factor (EGF)

In addition, PRP contains factors that include antibacterial and fungicidal proteins, metalloproteinases ADP, ATP, calcium ions, histamine, serotonin and dopamine, that may also affect tissue inflammation.[9]

Stem cells, specifically mesenchymal stem cells (MSC) found in bone marrow and adipose tissue, can differentiate into multiple cell lines including bone, cartilage, and fibrous connective tissue.[10] In addition, MSC secrete cytokine and growth factors that reduce inflammation, inhibit programmed cell death of the cells within the tissue, recruit circulating stem cells to the area, and integrate and reform tissue.[11]

Synergy between stem cells and PRP has been reported.[12-18] Certain growth factors and cytokines released from platelets bind to receptors on the surface of the stem cell and initiate a cascade involving signal transduction, gene expression and stem cell proliferation. PRP also provides a delivery vehicle or scaffold to support cell survival and proper differentiation.[19]

Anecdotal reports of faster recovery, improved function and reduced recurrence have been encouraging, however, there have been relatively few high-level clinical studies

evaluating efficacy. Studies are currently underway to validate use of regenerative medicine therapy for these types injuries in humans, horses and dogs. Regenerative medicine therapy should be considered for treatment of some conditions in this chapter. Rehabilitative therapy modalities to promote the activity of and prevent damage to the injected cells is paramount to successful treatment.

Forelimb Conditions

Most forelimb injuries sustained by performance dogs involve the shoulder. The shoulder joint is able to withstand large forces and provide extraordinary mobility while maintaining the stability and control necessary to enable precise function of the forelimb during activity. More than 25 muscles are required for shoulder motion, which involves components of flexion, extension, rotation, abduction, and adduction.[20] Given the muscular contribution and complexity of this intricate network of interlinked support mechanisms, the potential for muscle injuries causing performance issues and lameness is significant (Figure 33-1). When muscle strains occur during activities, muscle fibers can be disrupted, most commonly near the muscle-tendon junction.[20] These injuries are typically characterized by initial inflammation and pain, followed by healing with marked scar tissue, impeding the ability of the tendon to stretch.[21] Muscle strains have been reported in every major muscle group of the forelimb, but the muscles at particular risk in performance dogs are the biceps brachii and the supraspinatus.

Bicipital Tenosynovitis

One of the most common shoulder conditions seen in performance dogs is bicipital tenosynovitis (BT), which involves the biceps brachii muscle and its tendon that crosses the shoulder joint (Figure 33-2). The biceps flexes the elbow and extends and stabilizes the shoulder joint during standing or during the weight-bearing phase of locomotion.[22]

Cause

Repeated strain injury appears to be the primary cause of BT in the performance dog. This includes two-on/two-off contacts, landing vertically on the forelimbs from a misjudged jump, overstretching of the muscle, quick turns, and repetitive contractions of the muscle with the shoulder flexed and/or the elbow extended. Injury to the tendon can occur in a number of ways, including strain from overloading, degeneration, or disruption.[23] A single less than maximum load may injure some of the fibers without complete failure of the tendon, but the blood supply to the tendon proper is poor, leading to a longer healing time.[21] Repetitive strain injury may initiate actual degeneration of the tendon.[21] As the area continues to be reinjured, the tendon may weaken sufficiently for inflammation or microtears (tendinopathies) in the connective tissue in or around other tendons to form, ultimately leading to shoulder joint instability.[24]

Diagnosis

Performance dogs with BT commonly have difficulty with quick turning to the affected side and are reluctant to jump. Agility dogs may also have issues performing two-on/two-off contacts and knocking bars with their forelimbs. On gait analysis of dogs with BT often have a shortened stride and a weight-bearing lameness (subtle to severe) that becomes worse with activity. Pain may be elicited by direct

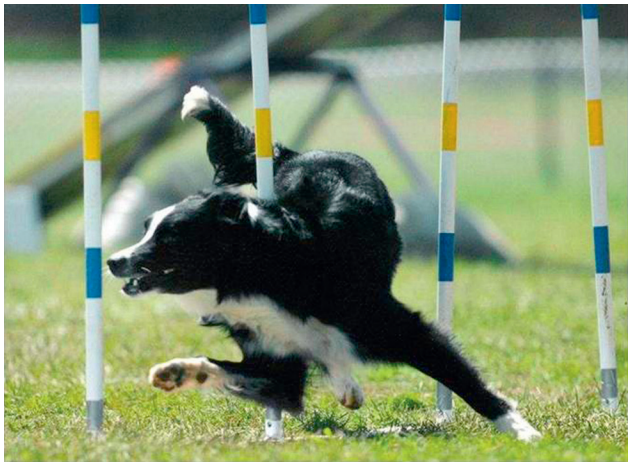


Figure 33-1 Dog going through weaves demonstrating biomechanical forces acting on the shoulder.

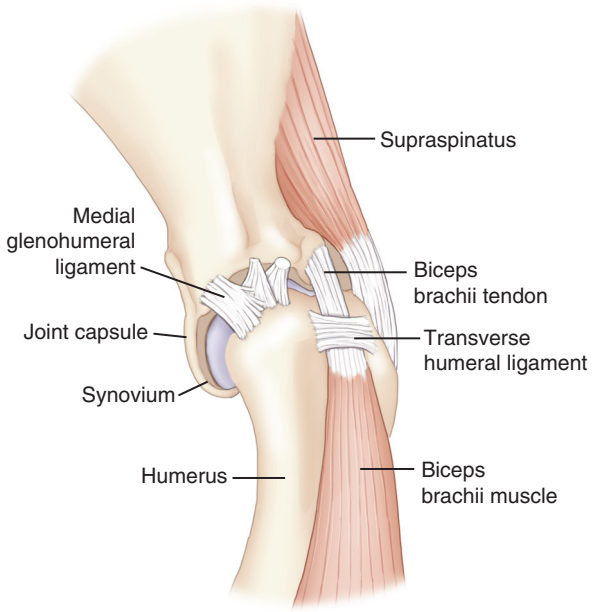


Figure 33-2 Normal canine shoulder anatomy (medial view).



Figure 33-3 Biceps stretch test.

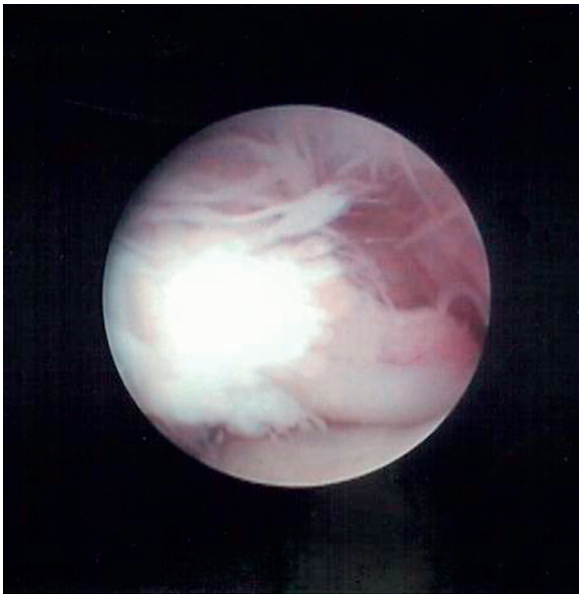


Figure 33-4 Normal arthroscopic image of the canine biceps tendon.

palpation over the biceps tendon. Pain and spasm may be noted when flexing the shoulder while at the same time extending the elbow (Figure 33-3).[3] Radiographs are of minimal assistance when the injury is in its acute phase, but may reveal mineralization of the tendon when the condition is chronic.[24] Magnetic resonance imaging (MRI) and ultrasound may be used to identify the condition in acute and chronic situations.[23,24] Shoulder arthroscopy is recognized as superior to other diagnostic procedures for diagnosis of bicipital tenosynovitis (Figures 33-4 and 33-5).[23]

Treatment

Acute cases of bicipital tenosynovitis may be treated by conservative medical management and rehabilitation therapy. Components of conservative medical management

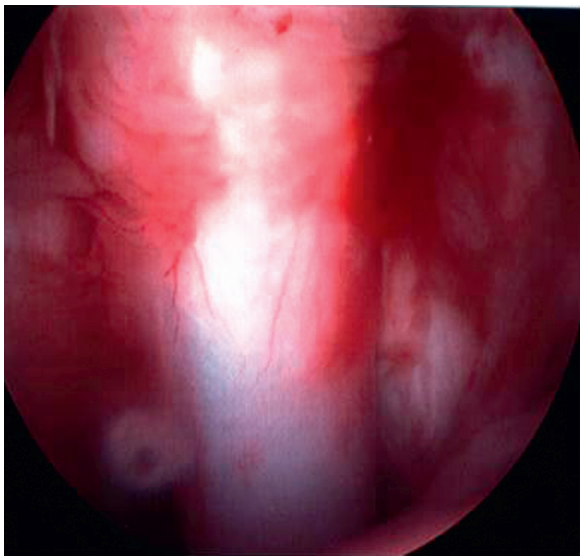


Figure 33-5 Arthroscopic image demonstrating severe bicipital tenosynovitis.



Figure 33-6 Laser therapy of the biceps tendon.

should include controlled activity, nonsteroidal antiinflammatory drugs (NSAIDs), and cryotherapy. Intraarticular injections of hyaluronic acid or steroids, or intra-tendinous injections of platelet rich plasma (PRP), stem cell therapy (SCT) or a combination of these may be considered.[20,23-25] Regenerative medicine therapy should be considered if a core lesion is present. To promote healing and decrease adhesion formation, laser therapy or pulsed ultrasound may be of use to assist healing to (Figure 33-6).[20] Initiating low-load, pain-free, high-repetition exercise and range of motion (ROM) movements early in the healing process is important. Pain relief may be achieved with transcutaneous electrical nerve stimulation.[20] Acute tendon injuries should not be stretched because of the potential for producing more microtears caused by the chemical changes in the tissue.[1,20] Pain-free, passive ROM movements (flexion and extension of the limb while the dog is not weight bearing)

and gentle wobble board activities (all directions, front to back and side to side), and alternating leg lifts are recommended first, with progression to active ROM exercises (weight-bearing exercises that cause flexion and extension of the limb). All exercises in the acute stage are aimed at increasing the strength of the shoulder and scapular stabilizers. Balance and proprioceptive exercises are required and are imperative to the healing process. An active dog requires stabilization of this region and strength of the slow twitch muscle fibers before resuming activity. Deep friction massage is used at this stage if it is not too painful.[25,26] Deep friction massage involves a sweeping motion made across the bicipital tendon (perpendicular to the fibers) for 5 minutes, daily if possible. The goal is to prevent adhesion formation and help realign the fibers of the inflamed tendon. After the cross-friction massage, cryotherapy and laser therapy are recommended before further treatment. A strengthening program consisting of controlled leash walking, stepping over cavaletti rails, wobble board use, and stabilizing exercises should follow. Examples of stabilization exercises include balancing on a well-controlled rocker board, standing on a therapy ball, weight shifting exercises, wheel-barrowing the dog so its weight is on the forelimbs, walking in figure eights, and walking in circles. Leash walks of increasing duration and end-stage eccentric exercises, such as walking and trotting down hills may then be introduced. Short periods of off-lead activity are allowed after leash walks have substantially increased without the return of lameness. In the acute stages, it is imperative to reinforce the owner or handler not to perform any activity that produces lameness. Many owners of performance dogs are eager to return to sports and often push their dogs too quickly.

With chronic BT the inflammatory process must be reinitiated to break down scar tissue and remodel the tendon fibers into the proper orientation, which increases the tendon's load capacity. Because of the need to reinitiate the inflammatory response, NSAIDs or intraarticular corticosteroids are not recommended for chronic tendinopathies. Appropriate therapy for chronic tendinopathies includes deep cross-friction massage, heat, ultrasound therapy, and pain-free ROM/stretching. At this stage cross-friction massage is applied for approximately 10 minutes, two to three times per week. Acupuncture and laser to increase circulation and for pain control may be beneficial as well. The exercise progression is similar to that recommended for acute tendinopathies, but stretching after treatment is advised. Active ROM exercise such as walking for longer periods also is recommended. End-stage eccentric exercises, such as walking and trotting down hills, should be added followed by short periods of off-lead activity provided lameness is completely resolved.

Because of the chronic nature of the tendon changes, the dog usually takes longer to recover and progress through the healing and exercise stages. It is again imperative to convey to the owner and handler that the progression of conservative management of BT may be lengthy and nothing should be done to aggravate the condition. The dog should be pain free and not exhibit any lameness.

Regenerative medicine therapy may also be considered in the treatment of chronic BT, however, modification of the rehabilitative therapy protocol is necessary to promote healing and prevent damage to the injected cells.

Surgical treatment is recommended for biceps avulsion-tears and chronic cases of BT that are not responsive to medical management or rehabilitation therapy. Surgical options include tenodesis (releasing and reattaching the tendon at a distal location on the proximal humerus) or an arthroscopic tendon release.[27] Controlled activity is an important component during the postoperative period and should include leash walks of increasing duration and end-stage eccentric exercises, such as walking and trotting down hills. Short periods of off-lead activity are allowed after the lameness has resolved. Nonsteroidal antiinflammatory drugs may be used to decrease pain and inflammation following surgery. Modalities as described for the acute treatment of BT may be used postoperatively to improve comfort and function and facilitate progression to the retraining phase of recovery.

Toward the end of rehabilitation therapy sport-specific retraining should begin. Examples of retraining techniques for BT may include starting with low, straight-line jumps with very wide sweeping turns that progress over time to higher jumps with tighter angles. Do not include the A-frame and weave poles until late in the retraining period. Alterations in contact controls from a two on/two off to running or four on the floor may be necessary. Be sure to start with turns toward the injured limb and progress to turns toward the uninjured limb.

Dogs that will be returning to jumping activities should start with low-level activities before returning to the sport.[4,26] In jumping activities, the biceps muscle and tendon act eccentrically to control the landing of the body. After cavaletti work has been employed, low-level jumping can be initiated. The jump heights should be set lower than the dog's normal jump height. For example, if the dog normally jumps 22 inches in competition, the jump height should be set at 16 inches. Three or four jumps should be set up and the surface should be padded or the exercise should be performed in sand. Three to five repetitions should be performed of the jump sequence—the dog going over the jumps—and then the dog should be reevaluated. The jump sequencing should not cause any lameness and should only be performed three times per week with at least a 48-hour break in between. Plyometrics should be progressed by increasing the jump height and the number of repetitions. Once the dog is up to its normal jump height on a controlled surface, additional training surfaces can be added.

Following a warm-up, make sure to perform active stretching techniques. Following agility work, use an

appropriate cool down with cryotherapy and passive stretching techniques (including shoulder flexion with elbow extension). Treadmill walking and the use of an underwater treadmill are also useful for the rehabilitation of BT. In acute stages exert caution with swimming because the shortened position of the biceps tendon with rapid joint motions may aggravate the condition.

Supraspinatus Tendinopathy

In humans and dogs several degenerative disorders of the supraspinatus tendon have been identified, including rotator cuff tears, calcifying tendinitis or tendinosis (microtears), and tendinosis as a result of overuse.[28,29] In humans degeneration of the supraspinatus tendon is believed to be a contributing factor in the development of rotator cuff tears.[29] There is good evidence that overuse is likely an important factor in this disorder. At the cellular level affected tendons contain discontinuous, disorganized fibers, and typically, no acute inflammation is detected.[29] In chronic cases a rapidly growing nodule develops that can impinge on the biceps brachii tendon and cause pain.[30] The supraspinatus muscle extends the shoulder and advances the limb. The muscle is important to stabilize and prevent collapse of the shoulder joint and is active during 65% to 80% of the time when the dog is standing (Figure 33-7).[22]

Cause

The cause of this injury in performance dogs appears to be related to repeated strain injury. This includes hitting the ground or agility contacts on an outstretched forelimb, slipping, overstretching, or overuse of the muscle, quick turns and repetitive eccentric contractions (contractions during lengthening of the muscle), as well as concentric contractions (contractions with shortening of the muscle) with the muscle in a lengthened state.

Diagnosis

Performance dogs with supraspinatous tendinopathy (ST) commonly have weight-bearing lameness on one side that becomes worse with activity and often is resistant to treatment. The supraspinatus muscle may be atrophied and direct palpation over the tendon and flexion of the shoulder may cause pain (Figure 33-8). Concurrent BT or shoulder instability may be present, so a thorough shoulder examination is required. Magnetic resonance imaging readily demonstrates the condition in the acute phase (Figure 33-9). Plain radiographs and computed axial tomography (CT), which reveals a cross-section of an internal body structure) may reveal mineralization in chronic cases. Musculoskeletal ultrasound is this author's preferred diagnostic tool for this condition. This technique provides a non-invasive definitive diagnosis of ST and allows for serial evaluations to assess response to treatment. Arthroscopic exploration is recommended and may demonstrate impingement of the biceps tendon secondary to supraspinatus tendon swelling as well as concurrent pathologies, including medial shoulder instability in severe cases.

Treatment

Controlled activity, retraining, and preventive techniques for acute and chronic ST is similar to that used for bicipital tenosynovitis. Similar to the rehabilitation of BT, rehabilitation should be initially aimed at reducing the inflammation and increasing the strength of the shoulder and scapular stabilizers.[4] After the inflammation has decreased and the core strength of the shoulder complex has improved, more dynamic activities may be initiated.

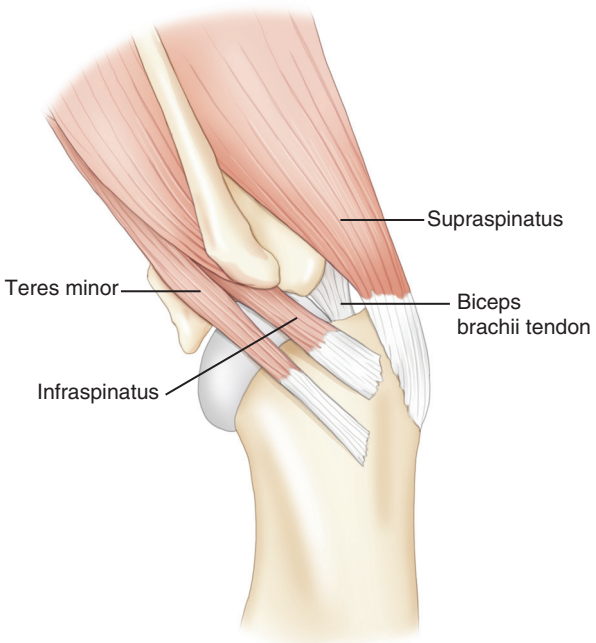


Figure 33-7 Normal canine shoulder anatomy (lateral view).



Figure 33-8 Palpation of the supraspinatus tendon point of insertion.

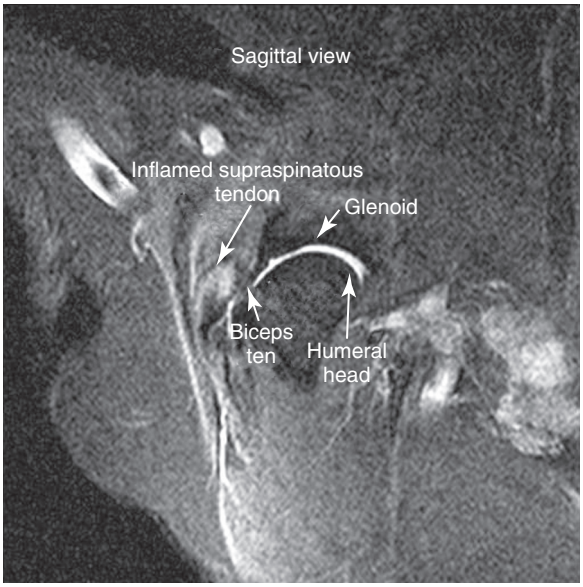


Figure 33-9 Magnetic resonance image of a supraspinatus tendinopathy.

Treatment for acute cases consists of conservative medical management (controlled activity and NSAIDs) and rehabilitation therapy (manual therapy, modalities, ROM with progression to stretching and strengthening exercises), and acupuncture as described for BT.[20] Cross-friction massage may be performed to the supraspinatus tendon in the acute phases for 5 minutes daily and 10 to 15 minutes in chronic cases.

As with BT the inflammatory process should be reinitiated in chronic cases and follow a similar rehabilitation protocol. Shockwave and ultrasound therapy have also been described as valuable modalities for the treatment of chronic calcified supraspinatus tendinopathies.[31]

Regenerative medicine therapy should be considered, especially in cases in which a core lesion has been identified. Under ultrasound guidance, cells can be directly injected into the affected tendon. In more severe cases a second injection may be needed. This therapy can be used as the primary treatment of ST, however, arthroscopic evaluation of the shoulder is recommended as concurrent pathologies of the joint are common. In these cases, arthroscopic treatment of concurrent pathologies can be combined with intraarticular regenerative medicine therapy to promote healing and speed recovery. Appropriate modification of the rehabilitative therapy protocol is necessary to prevent damage to the injected cells.

Results of a pilot study of 185 dogs with a confirmed diagnosis of supraspinatus tendinopathy (ST) by ultrasound or MRI performed by one of the authors (SC) provides support of regenerative medicine therapy for treatment of ST. Performance and sporting dogs accounted for just over one third (67) of the population, while companion animals accounted for the remaining two thirds (118). Of the performance/sporting dog population, 48% participated in agility. At presentation, a majority of dogs had a history of failed response (unresolved discomfort or lameness) to rest and NSAID therapy (134). Seventy-five failed to respond to rehabilitation therapy and 6 received intra-articular injections (hyaluronic acid – 2; prednisolone sodium succinate – 4), which also did not resolve lameness. A unilateral forelimb lameness presentation was more common than bilateral (124 and 61, respectively). Lameness was graded from 0 (sound) to 6 (non-weight bearing lame). The average lameness score was 2.9 (median 3). Duration of lameness ranged from one week to greater than a year. 29.7% of dogs had a chronic (>1 year) lameness. Of those with lameness less than one year, the average duration was 15.2 weeks (median 13 weeks). Pain on direct palpation of the supraspinatus was found in 59% of dogs. Pain or spasm on shoulder extension and abduction was noted in 63.1% and 71.3%, respectively. Pain or spasm on flexion was recorded in 74.6% of dogs and pain on biceps stretch (shoulder flexion with elbow extension) was recorded in 47.5% of dogs. Shoulder radiographs were performed on 161 dogs. Of these, mineralization was noted in 35 cases. Of dogs diagnosed with bilateral disease on presentation (61), mineralization was noted in 8 cases. MRI of the shoulder was performed in 30 cases. Findings indicative of a supraspinatus tendinopathy on MRI include hyperintensity of signal on T1 and STIR sequences of the ST at its insertion on the greater tubercle. Diagnostic ultrasound was performed in 140 cases. In most cases the supraspinatus appeared enlarged (44.3%) with irregular fiber pattern (46.4%) and mixed echogenecity (80.7%). Shoulder arthroscopy was performed in 131 cases. A supraspinatus bulge was noted in 90.8% of dogs. Of these, impingement of the biceps tendon was noted in 59.5% of dogs. While 43.5% of dogs had bicipital tenosynovitis, only 13% had actual pathology of the biceps tendon. Pathology of the subscapularis was observed in 91.6% of dogs. MGL pathology was present in 51.1% of dogs. A tear of the labrum was noted in 29.8%, cartilage defects were noted in 32.8% and bone lesions in 8.3% of dogs. Seventy-four cases were treated with regenerative medicine therapy. Final evaluations were performed on 64 dogs. Of these, lameness resolved in 90% of cases and improvement was noted in the remaining 10%.

Surgical treatment is warranted for chronic cases that do not respond to conservative medical management and rehabilitation therapy. Surgical treatment should include arthroscopic exploration to identify and treat possible concurrent BT or shoulder instability, or removal of abnormal tissue through an open approach.[30] Surgery has been considered the treatment of last resort in humans with tendinosis because of the small reported difference in treatment success between surgical intervention and conservative management. Rehabilitation therapy, as described for BT, following surgical intervention is essential in returning the canine athlete to performance.

The retraining protocol for this condition mirrors that of retraining following rehabilitation of BT.

Infraspinatus Contracture

Contracture of the infraspinatus muscle is a rare condition most often affecting large breed dogs, particularly working dogs (i.e., hunting dogs, herding dogs) and other very active dogs.[32] Infraspinatus muscle contracture was first reported in the Dutch literature in 1970[33] and is one of the two most frequently reported contractures in the dog, along with quadriceps contracture.[34,35] Unilateral contraction of the infraspinatus muscle is most common; however, bilateral contractures have also been reported.[33,36,37] Most cases are working or hunting dogs that present with an acute onset of shoulder lameness with work or exercise.[33,34,36-40]

The infraspinatus lies within the infraspinous fossa and extends caudally somewhat beyond the fossa. It arises from the fossa, the scapular spine, and the caudal border of the scapula, and at the shoulder joint becomes a strong tendon that crosses the caudal part of the greater tubercle. The infraspinatus is the outward rotator and abductor of the humerus and a flexor or extensor of the shoulder joint depending on the position of the joint when the muscle contracts.

Cause

Causes reported include repetitive microtrauma, blunt trauma, and osteofascial compartment syndrome.[41,42] Compartment syndrome is a disease process seen in people; however, it can also occur in dogs and horses.[43] The pathology to muscle tissue is caused by an increase in pressure in a confined, inelastic space that leads to muscle inflammation with no room to expand.[43] Muscle necrosis occurs when the pressure rises to greater than 30 mm Hg (normal, –2 to 8 mm Hg) in a single compartment with no relief.[43] A compartmental pressure greater than 120 mm Hg may lead to complete and irreversible nerve block.[44] Causes for an increased pressure within a compartment include hemorrhage, postischemic tissue swelling, external pressure (bandage), or intramuscular injection.[32] In elastic barriers that predispose to compartment syndrome there are structures such as fascia and bone.[43] Four different compartments have been described in the dog: femoral, craniolateral crus, caudal crus, and caudal antebrachial.[43,44]

It may be possible that compartment syndrome may play a role in the contracture of the muscles of the shoulder joint. If an injury is sustained to the infraspinatus muscle that leads to moderate cellular inflammation, the muscle begins to swell and has no room to expand. Inflammation within the boundaries of the deltoids, scapula, and surrounding fascia increase the compartmental pressure and inhibit venous blood flow, initially causing congestion. Then arterial blood flow and innervation are affected, as well as diminished cellular response to damaged tissue. It is the unforgiving pressure that leads to further cellular damage. Cellular swelling caused by pressures rising to greater than 30 mm Hg is what seems to lead to necrosis and/or fibrosis of the affected muscles. Early treatment is focused on surgical decompression by fasciotomy; however, if contraction has already occurred, tenectomy or myectomy may be necessary.[45]

Electrophysiologic studies of affected dogs indicate that infraspinatus contracture is a primary muscle disorder rather than a neuropathy. Histologically, affected tissues demonstrate degeneration and atrophy of skeletal muscle with fibrous tissue replacement. These findings support the hypothesis that acute traumatic events caused incomplete rupture of the infraspinatus muscle, leading to fibrosis and contracture.

Diagnosis

After an acute onset of a painful non–weight-bearing lameness, the initial pain and lameness improve over a period of 1 to 4 weeks, after which a characteristic circumducted gait abnormality develops in the forelimb. The initial injury to the infraspinatus muscle is not fully recognized or correctly interpreted in most cases, at least not with regard to its potential as a precursor of myopathy. Affected dogs most often present with mature contractures and adhesions of the tendon of insertion of the infraspinatus muscle to the adjacent joint capsule. As a consequence most or all of the internal rotation of the humerus in relation to the scapula is lost. This lack of rotation is a reliable clinical sign for diagnosis of the disorder. The humerus may also be held in slight abduction and external rotation.

Acutely the shoulder is swollen and very painful. The initial trauma usually resolves with rest and supportive treatment. However, the affected limb begins to show signs of contracture several days to weeks after the initial trauma.[40] Physical examination of a chronic case usually indicates a bright, alert dog, without signs of any systemic illness. There is usually a weight-bearing lameness of the affected forelimb and a characteristic gait. Palpation and manipulation of the contracted limb does not usually elicit a pain response.[38,40] The remaining shoulder muscles, mainly the supraspinatus[40] and deltoid muscles, may show signs of disuse atrophy or be contracted as well.[33,34,39,40] A substantial decrease in range of motion of the shoulder joint is appreciated.[40] The affected forelimb is usually held with the shoulder in abduction and the elbow in adduction against the thoracic wall[38] (Figure 33-10). The distal forelimb and carpus are externally rotated and held in abduction.[36,40] Gait abnormalities usually consist of weight-bearing lameness and circumduction[33,38-40] of the affected forelimb with a characteristic fliplike action of the paw when placing the foot.[36,40] When the patient is placed in lateral recumbency with the affected limb up, a true infraspinatus contracture will cause the distal forelimb to remain in an abducted and externally rotated position off the floor and away from the body.

Siems et al. reported a series of ultrasonographic findings of two concurrently traumatized infraspinatus muscles in the same dog; one resolved without complications, and the other resulted in contracture.[39] The primary ultrasonographic description of a contracted infraspinatus muscle is increased echogenicity when compared with a normal



Figure 33-10 Characteristic posture of a dog with contracture of the infraspinatus muscle.

supraspinatus muscle.[36,39] Magnetic resonance imaging is another diagnostic modality that may be useful for diagnosing the condition in both the acute and chronic phases.

Treatment

Physical rehabilitation of acute or subacute infraspinatus tendinopathies may be used to maintain joint motion and prevent contracture. Continuous ultrasound and stretching exercises may help maintain the length and mobility of the affected tissues. Mature contractures most likely require surgical release. Mature contractures are treated with a tenotomy of the tendon of insertion of the infraspinatus muscle and release of capsular adhesions of the tendon. Infraspinatus muscle contracture carries an excellent prognosis with tenectomy of the infraspinatus tendon.[40,46] Lateral or craniolateral approaches to the shoulder both provide good exposure of the infraspinatus tendon.[33,40,41] Complete transection of the infraspinatus tendon with removal of a length of the tendon is required to restore motion in the affected limb and most often proves curative with an immediate release of contracture and return to function of the shoulder joint.[36,40] It is common to find adhesions to the infraspinatus tendon and joint capsule during surgery; however, after they are dissected away they seldom cause further problems.[40,41] Postoperative bandages need not be applied, and rehabilitation therapy should start when sutures are removed to prevent the formation of further adhesions.[40]

The postoperative management after adhesion release includes full weight bearing and gentle, pain-free passive ROM, massage, and stretching exercises of the shoulder, elbow, and carpus two to three times daily to maintain ROM and promote normal alignment of the healing tissues. Cryotherapy may be used after exercise. Excessive activity such as unrestricted running or playing should be avoided in the first several weeks to avoid tissue damage and recurrence of fibrous tissue. If the contracture has been present for more than a few weeks before the surgery is performed, atrophy of other forelimb muscles may be present. General conditioning exercises for the limb, such as progressive walking, wheel-barrowing, and aquatic therapy, should be used to gradually return these muscles to normal size and strength. The use of neuromuscular electrical stimulation may also be considered if the atrophy is severe and the dog is having difficulty using the limb because of weakness.

Medial Shoulder Syndrome and Medial Shoulder Instability

Medial shoulder syndrome (MSS) is one of the most common causes of forelimb gait related issues and lameness in performance dogs. This condition may be considered somewhat similar to rotator cuff injuries in people (including ligament disruption; tendinopathy; labral and capsular tears or disruption). In human athletes, an overhead throwing motion involves intricate motion combined with proper stabilization. During this activity, the shoulder must be lax enough to allow extremes of motion but secure enough to provide stabilization.[4,26] The same is true for the canine athlete. Many routine maneuvers performed by dogs also involve extremes of ROM and require appropriate stabilization. In dogs with medial shoulder instability (MSI), lameness may be as subtle as performance-related problems such as refusing tight turns or as severe as a weight-bearing lameness. MSI involves multiple components of the shoulder joint. Definitive diagnosis requires a thorough orthopedic evaluation and advanced diagnostic tests. Medial shoulder instability is diagnosed when significant shoulder laxity is present. Early identification and treatment offer the best long-term result and prevent further shoulder instability and progression of osteoarthritis.

Anatomy

Anatomically, the shoulder joint is one of the least stable joints in the dog, relying on soft tissue structures for stability and function (Figure 33-11).[46] In dogs the glenohumeral (shoulder) joint has the widest ROM and is capable of movements in any direction, but its primary movements during ambulation are flexion and extension.[46] The glenohumeral joint relies heavily on capsuloligamentous support to provide stability because its anatomic conformation does not provide a stable socket.[46] The humeral head and glenoid cavity of the scapula make up the ball and socket of the shoulder joint. The glenoid provides little coverage of the humeral head. The joint capsule, ligaments, and surrounding muscles and tendons all contribute to the stability of the shoulder. Insult or injury to any portion of the capsuloligamentous support structures can potentially cause shoulder joint pathology resulting in pain and dysfunction.[47] The components that are most commonly affected by MSS and MSI include the craniomedial joint capsule, medial glenohumeral ligament, subscapularis

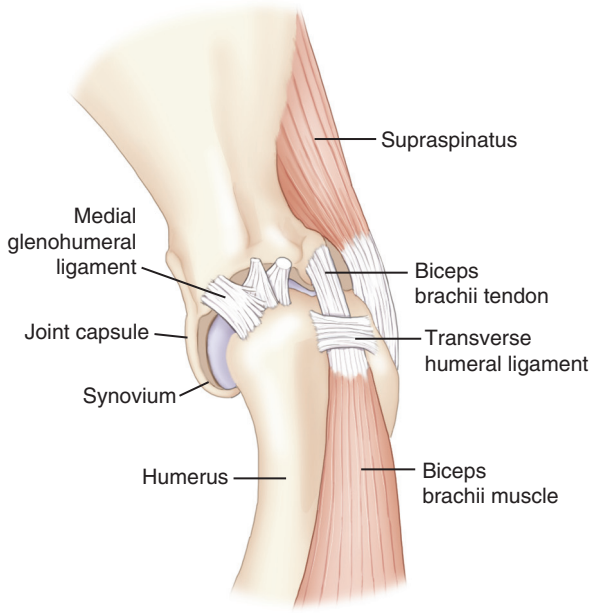


Figure 33-11 Normal canine shoulder anatomy (relevant to MSI).

tendon, supraspinatus tendon, labrum, and less commonly the biceps tendon (shown in Figures 33-12 and 33-13). Depending on the severity and chronicity of the condition, the cartilage may also be affected.

Cause

Currently, the exact cause of MSS in dogs is unknown, although it is suspected to be related to chronic repetitive activity or overuse rather than trauma. Overuse of the shoulder support structures leads to degeneration of the tissues, lowering the tensile strength of the tissues and predisposing them to fraying, disruption, and eventually complete breakdown.[1,20] Sporting athletes that participate in activities such as agility undergo extreme stresses on their muscles, ligaments, and tendons. Repetitive activities such as jump-turn combinations and weave poles performed regularly during practice and at weekend trials place the shoulder near its end range of abduction (as shown in Figure 33-14), and stress the soft tissues of the medial shoulder complex. Additionally, events such as slipping on wet surfaces, mishaps on the dog walk, teeter,

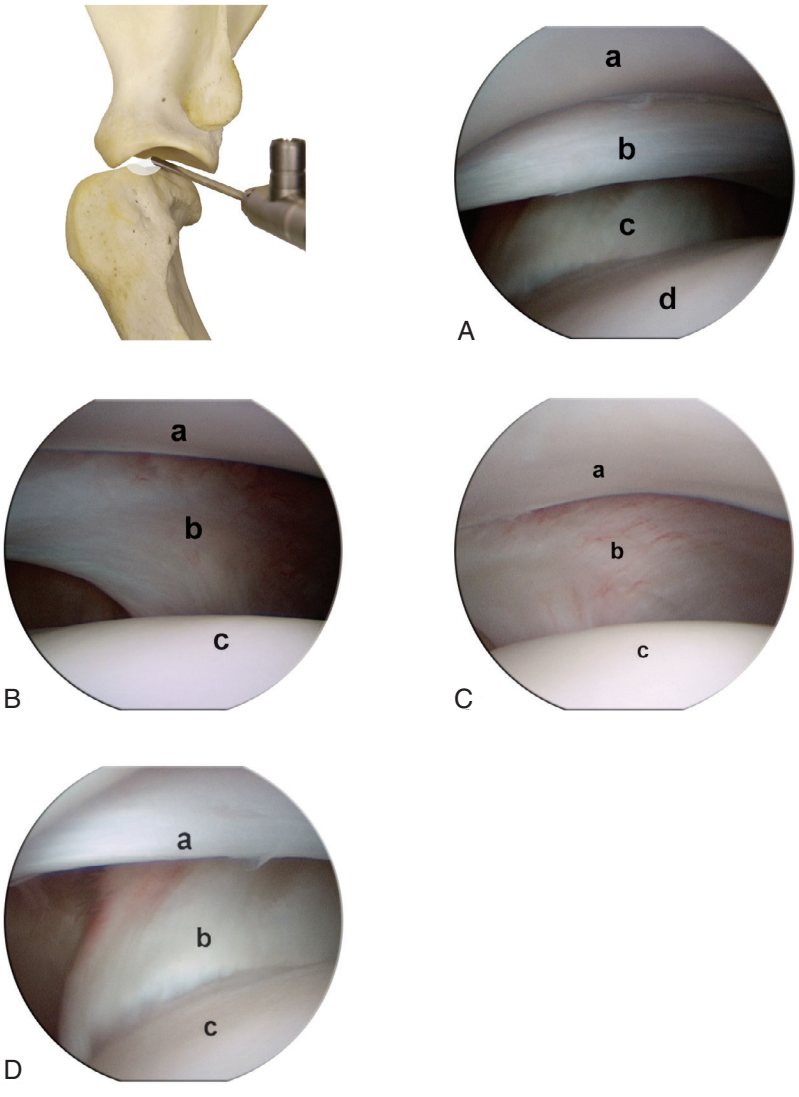


Figure 33-12 **A,** Arthroscopic view of the craniomedial compartment of the shoulder joint. Glenoid (a), cranial arm of the medial collateral ligament (MCL) (b), subscapularis tendon (c), and humeral head (d). **B,** Arthroscopic view of the centromedial compartment of the shoulder joint. Glenoid (a), centromedial area of the MCL (b), and humeral head (c). **C,** Arthroscopic view of the caudomedial compartment of the shoulder joint. Glenoid (a), caudomedial area of the MCL (b), and humeral head (c). **D,** Arthroscopic view of the cranial compartment of the shoulder joint. Cranial arm of the MCL (a), insertion of the subscapularis tendon (b), and humeral head (c). (From Beale, B.; Hulse, D.; Schulz, K.; Whitney, W.: *Small Animal Arthroscopy,* 2003, Saunders.)

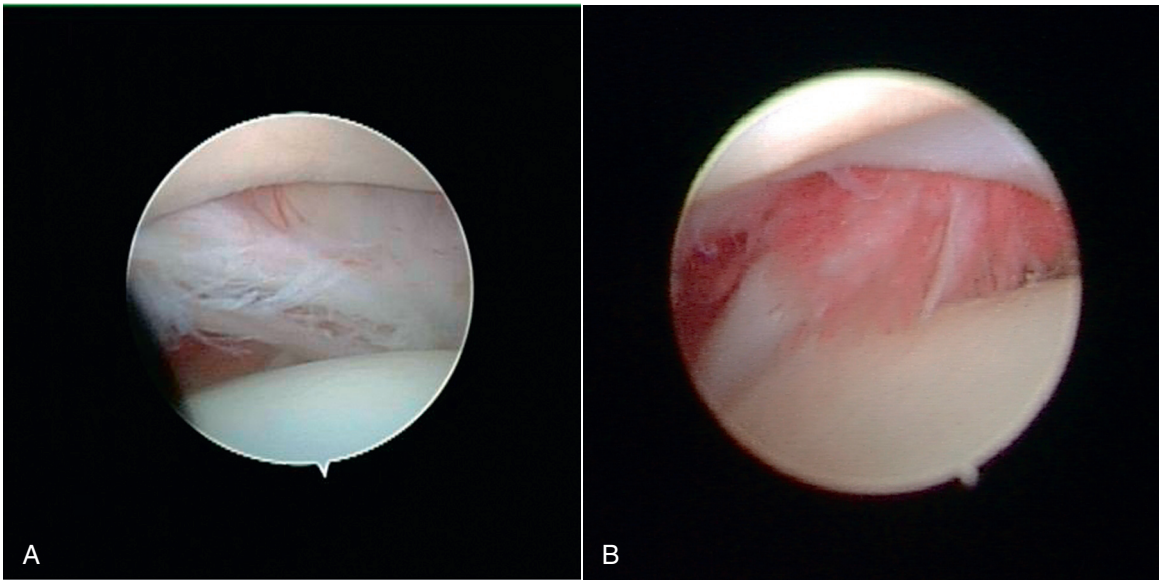


Figure 33-13 Abnormal arthroscopic images of an agility dog with MSI.



Figure 33-14 A shoulder in the end range of abduction.

or A-frame may also contribute to the trauma inflicted on the shoulder while participating in agility. Over time there may be a cumulative effect of the microtrauma occurring to the ligaments, tendons, and joint capsule, leading to a decrease in performance.

Diagnosis

History and Clinical Signs. Presenting complaints and clinical signs in dogs with MSS may vary from subtle, such as missing cues or refusing tight turns during performance, to an overt intermittent unilateral lameness. It is not uncommon for the history to include identification of restrictions, spasm, and trigger points, or mild atrophy in the affected shoulder by a therapist (veterinary rehabilitation therapist, veterinary massage therapist, or veterinary chiropractor) during routine sessions. Dogs with chronic conditions typically have a history of poor response to rest and NSAIDs, and increased lameness after exercise and heavy activity.

Orthopedic Examination. Findings from gait evaluation may range from a mildly shortened stride in the affected forelimb at a walk and a trot to significant weight-bearing lameness. Depending on chronicity, atrophy may be noted in the affected shoulder muscle(s) on physical examination. Forelimb circumference may be decreased in the affected forelimb when compared with the contralateral unaffected forelimb. Dogs with MSS typically have restriction and decreased range of motion in shoulder extension. The most consistent physical exam findings in dogs with MSS are shoulder spasm and discomfort on abduction of the affected shoulder. If a concurrent supraspinatus tendinopathy is present, pain may be noted when placing the shoulder into flexion (direct stretch of the supraspinatus) or on direct palpation of its point of insertion.

Because preoperative diagnosis of MSS is critical for clinical decision making, a palpation technique has been developed known as the *shoulder abduction test*.[48] Although this test was originally described in dogs under sedation in lateral recumbency, it is noted by the author that this test may be performed with similar consistent results on dogs awake and standing. To appropriately engage the components of the craniomedial shoulder, the elbow and shoulder must be placed in full extension with concurrent abduction of the forelimb. The scapula at the level of the acromion process must be stabilized with the evaluator's opposite hand to achieve a passive stretch of the craniomedial shoulder components. A goniometer is held such that one arm of the goniometer runs parallel to the spine of the scapula so that the center of rotation of the goniometer is centered over the shoulder joint and the free arm of the goniometer extends over the axis of the humerus in contact with the skin. With the elbow and shoulder held in extension, the forelimb is abducted to its physiologic limit and the measurement obtained as shown in Figure 33-15. In severe cases, which clinically appear to be much less common, a slight "thud" or subluxation may be felt when abducting the shoulder. The procedure should then be performed on the contralateral shoulder for comparison. Using this



Figure 33-15 Abduction test.

palpation technique, it has been reported that a normal mean shoulder abduction angle is approximately 30 degrees.[48] Although this report revealed that dogs with medial shoulder instability had abduction angles significantly larger than 30 degrees,[48] a more recent study showed an inconsistent correlation between abduction angles and medial compartment pathology identified arthroscopically.[49] This is also the author's clinical observation. Dogs with MSS rarely show evidence of "instability," but rather show signs of dysfunction and pain associated with craniomedial pathology, including varying degrees of subscapular tendinopathy; medial glenohumeral ligament (MGL) disruption; craniomedial capsular lesions; and labral tears. Because MSS is usually a unilateral problem, the contralateral shoulder usually does not show signs of dysfunction, pain, or spasm on abduction.

Advanced Diagnostics. In addition to history, signalment, gait analysis, physical examination, orthopedic and neurologic examinations, and abduction angle tests, further diagnostic tests used to differentiate causes of shoulder pathology currently consist of hematology, biochemical profile, urinalysis, arthrocentesis, imaging modalities, and arthroscopy. Considered the gold standard, arthroscopic evaluation of the shoulder provides a definitive diagnosis of MSS or MSI.[47-49] Arthroscopy of the shoulder allows for direct observation of all major intraarticular structures with magnification, "dynamic" evaluation of tissues during shoulder ROM tests, and "palpation" of intraarticular tissues using arthroscopic instrumentation.

Treatment

Patients are placed into one of three treatment categories; mild, moderate, or severe based on the results of the orthopedic examination, abduction angle tests, and arthroscopic scoring. Use of a shoulder support system/hobbles (such as DogLeggs®)[1] and rehabilitation therapy is required in all cases.

Physical therapy for orthopedic conditions has a strong scientific background in human medicine. Many studies have shown the benefits of rehabilitation following rotator cuff surgery. The canine rehabilitation therapist should be aware of treatment techniques used in people and apply similar goals, principles, and techniques to dogs. Be aware that not all exercises work for every patient; some exercises require trial and error to achieve desired results. Some dogs tolerate the shoulder support system quite well, whereas others face a difficult time with the system. Some owners become frustrated with the shoulder support system and the restraint of the dog. Home education and owner compliance play a significant role in the postoperative management of MSS and MSI stabilization; therefore it is essential to reinforce the importance of the stabilization and the compliance of the program. Behavioral activities for the dog are beneficial.

Mild Medial Shoulder Syndrome (MSI). Patients with mild MSS most commonly have arthroscopic findings consisting of mild pathology (inflammation without fraying, disruption, or laxity of the MGL, subscapularis tendon, joint capsule) are placed in a shoulder support system/hobbles, such as DogLeggs®, and entered into a rehabilitation program. The rehabilitation program is restricted and consists of stabilization exercises only during the initial period. Cessation of activities, running, and jumping are required and leash walks must be enforced. The length of leash walks depends on the dog's endurance, pain, and inflammatory level. Quality over quantity should be emphasized with leash walks, and the owner should not push the dog to lameness with daily walks. Additional rehabilitative exercises include balance board, lifting of the opposite forelimb and hindlimb, weight shifts, standing on a balance ball, walking in figure eights and circles, and scapular stabilization work on a balance ball or disk. Stretching the shoulder in extension is permissible, but any abduction movements should be avoided. Modalities such as laser, cryotherapy, and moist heat are beneficial to reduce the inflammation and muscle spasms, respectively. Neuromuscular electrical stimulation may be used if there is a significant amount of atrophy and are usually targeted at the muscle belly of the supraspinatus and infraspinatus. The teres major is often restricted and painful as a result of the compensatory stabilization and may benefit from laser therapy and massage. Compensations in the cervical and thoracic regions should be addressed if present. In addition, underwater treadmill activity may be initiated. Typically water heights should be above the elbow to decrease the stress placed on the elbow. Swimming is only advised if the dog is able to swim in a controlled fashion without shoulder abduction. As the dog progresses and the weakness in the shoulder complex has improved, a gradual return to activity may

[1]Shoulder support system, DogLeggs, Reston VA.

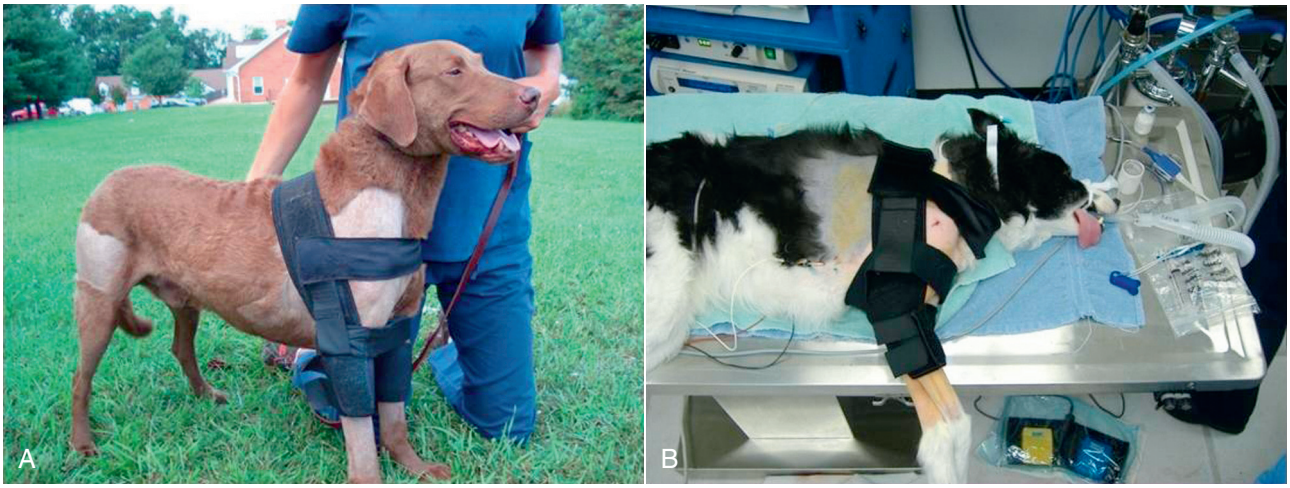


Figure 33-16 **A,** Dog wearing the shoulder support system following **B,** arthroscopic treatment for MSI.

be initiated. An increase in forelimb circumference should occur over time, along with increased active and passive shoulder extension, and decreasing lameness. This may take from 2 to 8 weeks depending on the dog, severity of injury, activity level, and commitment to rehabilitation. At this point a gradual return to activity may be initiated. The agility dog's jumping style and contact style may need to be adjusted.

Moderate Medial Shoulder Syndrome (MSS). Dogs with moderate MSS most commonly have arthroscopic findings that include subscapularis tendinopathy (disruption, fraying, core lesion), medial glenohumeral ligament pathology (fraying or disruption), craniomedial joint capsule focal synovial proliferation and/or disruption, and labral pathology (fraying, disruption, or tears). Additional findings occasionally include a bulge of the supraspinatus tendon with impingement on the biceps tendon. Depending on the tissues involved and the severity of disease, dogs in the moderate category are treated arthroscopically with a variety of techniques including radiofrequency-induced thermal capsulorrhaphy (RITC), suture imbrications (fiber wire), or stabilization using a specialized fiber band and buttons technique (TightRope®, Arthrex Co., Naples, FL).[47,49,50] The current technique for radiofrequency treatment has been altered significantly since the previous report for the treatment of MSI in dogs[50] because of the detrimental effects that can occur with aggressive application. Currently a single pass striping technique versus multipass paintbrush technique has been developed based on current application in humans.[51] In addition, a monopolar (vs bipolar) radiofrequency generator and probe using a default setting of 25 W and 70° C with high fluid flow egress has been employed to protect the surrounding tissues and cartilage. Radiofrequency-induced thermal capsulorrhaphy delivers radiofrequency energy to the capsule tissue, which causes the collagen to denature and the capsule to shrink. The optimal temperature to achieve the most shrinkage without causing necrosis of the tissue is between 65° and 75° C. This treatment causes a significant decrease in mechanical stiffness for the first 2 weeks, and then, after the tissue undergoes active cellular repair from the surrounding uninjured tissue, the mechanical properties return to near normal by 6 to 12 weeks.[52] To protect the tissues during this period dogs are placed in a shoulder support system/hobbles (such as DogLeggs®), such as those shown in Figure 33-16, for 3 months, and entered into a rehabilitation therapy program.

During the initial phases of the postoperative period, treatment is minimal and focuses on stabilization activities and PROM. Modalities aimed at reducing inflammation should not be used to maintain the inflammatory process. Active range of motion is not performed on the shoulder complex until 8 weeks postoperatively. Passive range of motion is performed on the digits, carpus, and elbow. Joint compressions may be performed to the digits, carpus, and elbow during the initial period, but the shoulder region should be avoided. If restrictions are present in the digits, carpus, or elbow, appropriate joint mobilizations to increase ROM may be performed.[4] However, joint mobilizations should not be performed to the shoulder. Restrictions in the contralateral limb, spine, and hindlimbs should be evaluated and treated appropriately with ROM, joint mobilization, massage, laser, and other modalities. Leash walks are permitted but are restricted.

The hobbles must be worn at all times. Weight-shifting exercises may be added 4 weeks after surgery, as well as stretches of the cervical region. Sit-to-stands are also beneficial at this time. The dog should remain in the shoulder support system or hobbles at all times, with the exception of rehabilitation. Typically the dog is seen every other week for the first 8 weeks to monitor progress and ROM. At that point, weekly therapy is necessary and should consist of stabilization work, ROM, balance and proprioception exercises, and endurance

work. The underwater treadmill and more dynamic activities are not introduced until approximately 12 to 14 weeks postoperatively, although standing in water before that period is beneficial if the dog remains in its hobbles and does not thrash around in the water.

At 12 to 16 weeks postoperatively full ROM of the shoulder joint should be present without any soft tissue tenderness. Underwater treadmill therapy may begin at this stage. Also, appropriate modalities may be used if needed on any region. During the final stages of rehabilitation, approximately 12 to 16 weeks, the hobbles may be removed for controlled activity. Initially, short periods of exercise (5-15 minutes) are recommended without the hobbles and the activity should be controlled. For example, the dog may be off leash in a small fenced area. The goal is to remove the hobbles completely, unless the dog is in danger of slipping on the floor or during aggressive play. This may take several weeks to achieve. During rehabilitation, activities such as swimming, side stepping, cavaletti work, and hill walking may be added. All of the activities must be monitored and controlled. The activity may progress to tight turns, crawling activities, walking through tunnels, advanced balance activities, and increased endurance activities, such as leash walking and underwater treadmill.

The goals of the individual dog should be evaluated, and if the dog is returning to a sport such as agility, specific agility activities should be added 15 or 16 weeks postoperatively. Trotting, running, and jogging should be introduced on different surfaces and different intervals. Transitioning from a walk to a trot to a jog and back is an exercise that helps with proprioception and strength. Jumping can be introduced with a low-level jump in a straight line. The dog's normal jump height should be reduced by 50% to 75% depending on the height of the dog. For example, if the dog normally jumps 26 inches, the initial jump height should be 12 inches. Begin with one jump and progress to a series of three to five jumps in a row, spaced about three times the body length of the dog from each other. The dog should be jumped three to five times for three repetitions, two to three times per week. If no problems persist, the repetitions and the height can be gradually increased. Turns in the jumps may be added—setting consecutive jumps in a semicircle or a pinwheel. As the dog progresses, tighter turns can be added with a gradual return to normal activities.

Severe Medial Shoulder Syndrome (MSS) and Medial Shoulder Instability (MSI). Dogs with severe MSS or MSI typically have signs of craniomedial compartment laxity with shoulder abduction angles greater than 65 degrees. Arthroscopic findings usually include complete tears of the medial glenohumeral ligament and severe disruption of the subscapularis tendon, joint capsule and labrum. For this type of injury reconstruction of the medial compartment by direct tissue reapposition using synthetic capsulorrhaphy, bone/soft tissue anchors, and/or TightRope® stabilization (Arthrex) may be indicated. Following surgical repair dogs are placed in a non–weight-bearing Velpeau sling for 2 weeks followed by a shoulder support system (such as DogLeggs®) for 2 to 3 months. Rehabilitation therapy is required for a longer period of time because recovery following primary reconstruction ranges from 4 to 6 months. Fortunately, severe cases are less common and are usually caused by trauma rather than repetitive activities, as seen in the mild and moderate categories.

Traumatic Fragmented Medial Coronoid Process

Traumatic fragmented medial coronoid process (TFMCP), sometimes called *jump down syndrome*, is a condition in the elbow joint of dogs that appears to occur commonly in performance dogs. Unlike the classic condition of fragmented medial coronoid process (FMCP), which affects the elbow joints of skeletally immature large to giant breed dogs, TFMCP appears to have no age or size limitations.

Anatomy

The elbow joint as a whole is a trochlea arthrosis or ginglymus (hinge) joint, allowing flexion and extension of the humerus over the radius and ulna. In dogs, the caudal aspect of the head of the radius rotates in the radial notch of the ulna during pronation and supination.[22] The normal elbow joint is characterized by a smooth transition from the ulnar articular surface to the radial surface. The medial coronoid process of the ulna is level with or slightly below the surface of the radius (Figure 33-17).

Cause

The cause of TFMCP is poorly understood. It is possible that abnormal repetitive loading, such as landing from a jump, hitting contacts or a flyball box, may lead to microfractures of the bone underneath the cartilage (subchondral fractures).[53] Additionally, increased repetitive loading can arise from contraction of the biceps/brachialis muscle

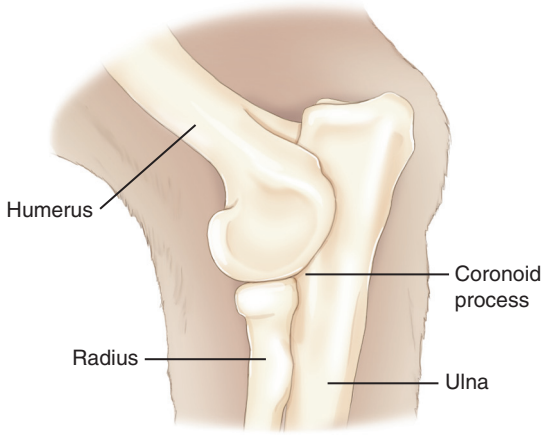


Figure 33-17 Normal canine elbow anatomy.

complex. When the biceps/brachialis contracts, a force is generated that rotates the medial coronoid into the radius. These microcracks disturb the mechanical properties of bone, and if not repaired properly through normal healing mechanisms, fatigue fractures develop. Additionally, loss of osteocytes (bone cells), indicated by decreased osteocyte density, has been strongly associated with the presence of microdamage after fatigue loading.[53] These studies imply that excess load may lead to fatigue microdamage of the subchondral trabecular bone and eventual fracture, which may play an important role in the pathogenesis of TFMCP. Dogs may be further predisposed to this condition if they have elbow dysplasia. Dogs with elbow dysplasia may have asymmetric growth of the radius and ulna during development, resulting in elbow joint incongruity. Elbow incongruity, such as radioulnar step defects, humeroulnar incongruence/conflict, and varus deformity of the humerus, causes abnormal contact patterns in the elbow, specifically at the coronoid-trochlear articulation, which is theorized to increase the load on the medial coronoid process (MCP).[54] Regardless of the etiology, if left untreated as a continual lameness, secondary osteoarthritis may progress, as noted by damage to the cartilage such as softening, fibrillation, fissuring, and erosions as well as additional subchondral bone microcracks and fragmentation. The free fragments may contribute to frictional abrasion ("kissing lesions") of the opposing surface, including the medial aspect of the humeral condyle and radial head.

Diagnosis

History and Clinical Signs. Dogs with TFMCP may be presented with a history ranging from a subtle intermittent off-loading of the forelimb to significant unilateral or bilateral forelimb lameness. This lameness is typically exacerbated with exercise and heavy activity. The onset of lameness is insidious. As lameness persists, it may increase in severity. Affected dogs often place the carpus in an exaggerated valgus position (turned out) when sitting or standing, and circle the foreleg outward and move the elbow away from the midline (circumduct the antebrachium and abduct the elbow) during the swing phase of the stride. The history of dogs with TFMCP typically includes a lack of response to rest and NSAIDs. Many dogs with TFMCP are mistakenly treated for shoulder pathology because the attending veterinarian elicits a pain response when the shoulder is extended. The authors believe the pain response actually arises from the elbow because when the veterinarian performs an extension maneuver of the shoulder, the elbow is usually simultaneously extended. Extension of the elbow causes tension in the biceps/brachialis muscle complex. Tension in the biceps/brachialis exerts pressure on the medial coronoid and overlying inflamed joint capsule causing the pain response.

Orthopedic Examination. On physical examination discomfort is usually noted on direct palpation of the medial compartment of the elbow joint, specifically the medial coronoid process. Discomfort may also be noted on hyperflexion of the elbow. Most dogs with TFMCP are reluctant to allow full elbow flexion. In chronic cases, full flexion may not be obtained. Crepitus may be noted when placing the elbow through ROM. Some investigators suggest that the carpus should be placed in a flexed, externally rotated position while the elbow is extended to exacerbate the pain response. Joint effusion may be detected as a fluctuant swelling distal to the lateral or medial epicondyle of the humerus. Depending on the chronicity, atrophy may be noted in the affected forelimb muscles.

Advanced Diagnostics. In addition to history, gait analysis, physical examination, and orthopedic and neurologic examinations, further diagnostic tests used to differentiate causes of elbow pathology currently consist of hematology, biochemical profile, urinalysis, arthrocentesis, imaging modalities, and arthroscopy. Unfortunately, radiographs may be of little value in early cases because of difficulty identifying the fragment or line of separation using standard radiography.[55] In chronic cases, however, radiographs may reveal secondary evidence of bony remodeling consistent with osteoarthritis. These changes may include sclerosis of the ulnar notch and remodeling along the anconeal process and MCP.[56] Advanced diagnostic imaging modalities such as CT scans, MRI, nuclear scans, and arthroscopy may allow confirmation of the condition.[56] Arthroscopic evaluation of the elbow joint allows direct observation of all major i ntraarticular structures with magnification, "dynamic" evaluation of tissues during elbow ROM tests, and "palpation" of intraarticular tissues using arthroscopic instrumentation.[57] Arthroscopic exploration of the elbow provides a definitive diagnosis of TFMCP when a fragment or cartilage fissure is observed (Figure 33-18). In a small percentage of cases advanced imaging (nuclear scan, CT, MRI) may indicate fragmentation of the MCP, but arthroscopic observation does not reveal a fissure or fragment. In such cases the fissures (microcracks) are believed to be within the coronoid process beneath the cartilage surface.

Treatment

Treatment of TFMCP is multimodal and includes a combination of medical and surgical management as well as rehabilitation therapy. Through a multimodal approach it may be possible to relieve pain and maintain limb function, as well as to return the dog to a near-normal level of activity and competition.

Arthroscopy is an excellent modality for diagnosing TFMCP as well as a minimally invasive means of treatment. Arthroscopic removal of the fragments is recommended not only to remove the inciting cause of lameness

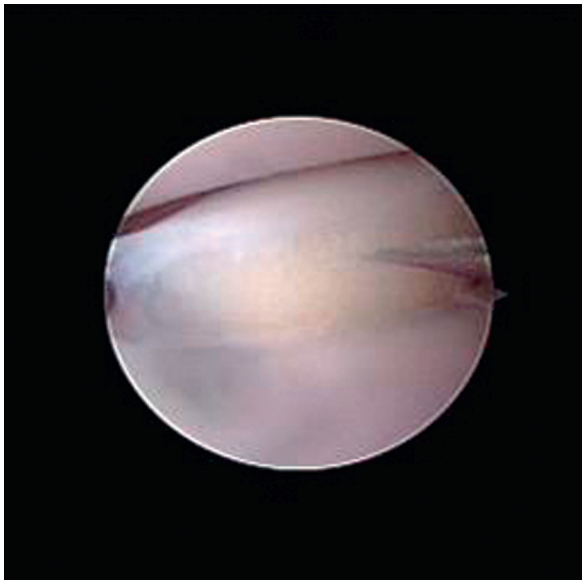


Figure 33-18 Arthroscopic image of a traumatic fragmented medial coronoid process in an agility dog.

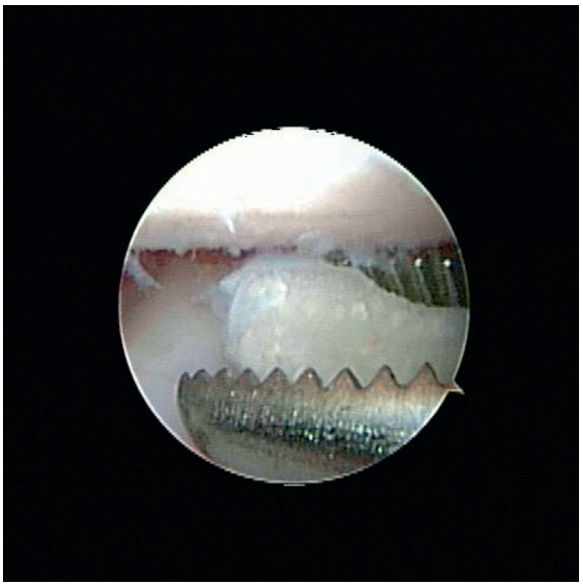


Figure 33-19 Arthroscopic treatment of a traumatic fragmented medial coronoid process.

but also to help slow the progression of osteoarthritis. Arthroscopic treatment may include a combination of techniques such as fragment removal, débridement of diseased tissues, creation of vascular access by abrasion arthroplasty, forage, microfracture, and subtotal coronoid ostectomy depending on the progression and severity of disease (Figure 33-19).[58] Arthroscopy allows for superior visualization of structures in the joint and is also believed to have less soft tissue trauma, shorter surgery and hospitalization times, decreased risk of infection, and shorter recovery times compared with the traditional surgical approach of fully opening the joint (arthrotomy).

Following arthroscopic treatment, dogs are typically prescribed NSAIDs for 14 days to help decrease inflammation and discomfort. Polysulfated glycosaminoglycan (PSGAG), an injectable joint protective agent, is also recommended following arthroscopic treatment and is administered intramuscularly twice a week for up to 4 weeks. Polysulfated glycosaminoglycans have been shown to decrease the amount of degradative enzymes (which stimulate inflammation and cartilage erosion) as well as promote repair and regeneration of cartilage.[59] In addition, an oral joint protective agent such as glucosamine, chondroitin sulfate, and avocado/soybean unsaponifiables (ASU) is recommended as a daily supplement for life. The antiinflammatory properties and pain-reducing effects of these supplements have been studied.[60,61] There is also evidence that they may have a chondroprotective effect.[60,61]

As with any rehabilitation therapy program, rehabilitation after elbow arthroscopy should follow a sequential and progressive multiphased approach.[62] The ultimate goal of elbow rehabilitation is to return the patient to its previous functional level as quickly and safely as possible. However, each case is uniquely different, and several factors—including duration of injury/lameness before surgery, secondary osteoarthritis and surgical intervention—all influence the rate at which the patient proceeds through the healing process.

The elbow is predisposed to flexion contractures because of the intimate congruency of the joint articulations, the tightness of the joint capsule, and the tendency of the cranial capsule to develop adhesions following injury. The biceps/brachialis complex also attaches to the capsule and crosses the elbow joint before becoming a tendinous structure and inserting on the ulna. Injury to the elbow may cause excessive scar tissue formation of the brachialis muscle as well as functional splinting of the elbow. Reestablishing full elbow extension is the primary goal of early ROM activities to minimize the occurrence of elbow flexion contractures.[63-65]

Additional goals of this stage are to protect healing tissue, decrease pain and inflammation, restore weight bearing, and retard muscular atrophy.[66-74] The rehabilitation therapist must not overstress healing tissues during this phase. It is important that the owner restrict the dog's activity to short leash walking only with no running, jumping, and rough play.

Cryotherapy (Figure 33-20) may be performed during the initial inflammatory period to assist in reducing pain and inflammation. The primary physiologic effects include vasoconstriction, reduced cellular metabolism and permeability, decreased motor and sensory nerve conduction velocity, analgesia, and decreased muscle spasm.[75-77] Ice



Figure 33-20 Cryotherapy of the elbow immediately post arthroscopy.



Figure 33-21 Range of motion of canine elbow following arthroscopic treatment.

may be applied around the entire elbow joint for a duration of 10 to 20 minutes, three to six times daily.

Range of motion is performed to the entire forelimb: toes, carpus, elbow, and shoulder (Figure 33-21). Slow controlled movements are performed with slight over pressure at end range. Range of motion activities are usually performed two to three times daily. Range of motion activities are performed for all planes of elbow and carpus motions to prevent the formation of scar tissue and adhesions. Grade I and II joint mobilizations (Figure 33-22) may be performed during the early phase of rehabilitation as tolerated.[78] Joint mobilizations increase circulation, proprioception, and healing and may decrease swelling. Pain-free grade III posterior glides with oscillations are performed in the midrange of motion to assist in regaining full elbow extension. Aggressive mobilization techniques are not used until later stages of rehabilitation when pain has subsided. Grade I and II mobilization techniques are also used to neuromodulate pain by stimulating articular receptors.[78,79] Grade III and IV joint mobilizations also should be performed to all joints that are hypomobile or restricted in any direction. Common regions of decreased mobility may also include the scapula, cervical/thoracic spine, or pelvis.

Manual therapy, including soft tissue massage/mobilization and stretching of the surgical forelimb, nonsurgical forelimb, cervical spine, and hindlimbs may be necessary because of compensations that occur before and/or secondary to surgery. If the dog continues to have difficulty achieving full extension using ROM and mobilization techniques, a low load, long duration (LLLD) stretch may be performed to produce a creep of the collagenous tissue, which results in tissue elongation.[80-83] The dog lies in lateral recumbency with a towel roll placed under the brachium to act as a cushion and fulcrum. Light pressure



Figure 33-22 Joint mobilization of the elbow in a dog following arthroscopic treatment.

is applied to the carpus and distal antebrachium. The amount of pressure applied should be of low magnitude to enable the dog to perform the stretch for the entire duration without pain or muscle spasm. The aggressiveness of stretching and mobilization techniques is dictated based on healing constraints of involved tissues as well as the amount of motion and end feel. If the dog has decreased motion and a hard end feel without pain, aggressive stretching and mobilization techniques may be used. Conversely, a dog exhibiting pain before resistance or empty end feel should be progressed slowly with gentle stretching.

Several modalities may be used to aid in the healing process during this stage and serve as an adjunct to manual treatments and therapeutic exercises. Laser therapy (Figure 33-23) mediates a variety of events including release of



Figure 33-23 Laser therapy of the elbow following arthroscopic treatment.



Figure 33-24 Transcutaneous electrical nerve stimulation of the elbow following arthroscopy.

growth factors, cytokine reactions, and cell replication; the ultimate effect of these events is accelerated tissue healing.[84,85] Laser treatment dosage is provided in Joules (energy). A typical treatment of 4 to 6 Joules per cm^2 is applied covering the entire treatment area. Laser therapy can be performed daily for the first week, and then decreased to every other day during week two. After week two, laser may be applied on an as needed basis.

Ultrasound therapy is a commonly used modality in human medicine.[86] Ultrasound has a direct effect on cells and in laboratory studies, to stimulate healing. A treatment schedule may include daily treatment for up to 10 days, followed by less frequent sessions as the condition improves. Continuous wave ultrasound produces heating of the tissues, which may be beneficial before stretching.

Transcutaneous electrical nerve stimulation (TENS) (Figure 33-24) is used commonly for pain relief. The primary effect of pain relief is obtained through two main mechanisms: the inhibition of the pain via the gate theory, and via descending inhibitory mechanisms (release of endogenous opiates).[87] A recent study in dogs showed significant improvement in ground reaction forces (weight bearing) immediately after treatment that lasted for up to 3 hours.[88] Treatments initially may be applied three to seven times per week and then decrease in frequency as pain and lameness subside.

After the acute inflammatory phase has passed, moist heat, warm whirlpool, and ultrasound may be used at the onset of treatment to prepare the tissue for stretching and to improve the extensibility of the joint capsule and musculotendinous structures.

The early phases of rehabilitation must also focus on retarding muscular atrophy through therapeutic exercises. Alternating rhythmic stabilization drills for forelimb flexion/extension/horizontal abduction/adduction and shoulder



Figure 33-25 Three-legged standing.

internal/external rotation are performed to begin reestablishing proprioception and neuromuscular control of the upper extremity.[89,90] Leash walking may be initiated after surgery, beginning with 5 to 10 minutes, three times a day, with increasing duration to 20 minutes as tolerated, by the end of the acute phase. Other weight-bearing exercises are initiated at this time and are performed on a daily basis. Some examples include the following:

- Leg standing—Lift nonsurgical forelimb and shift weight to surgical limb (Figure 33-25).

- Down to stand—Ask patient to lie down and then to stand. Use treats as needed.
- Rocking on all fours—Dog stands on all four limbs. Gently rock the dog from side to side and diagonally, with 3-cm perturbations in each direction. Promotes weight bearing and proprioception to the affected limb.

When the dog exhibits full ROM, minimal pain and tenderness, and improved lameness progression, the goals should be focused to enhance elbow and upper extremity mobility, improve muscle strength and endurance, and reestablish neuromuscular control of the elbow complex.

Stretching exercises are continued to maintain full elbow flexion and extension. Mobilization techniques may be progressed to more aggressive grade III and IV techniques, as needed, to apply a stretch to the capsular tissue and improve end range. Flexibility progresses during this phase to focus on the carpus, elbow, and shoulder, including—flexion, extension, internal and external rotation.

Also during this stage emphasis is placed on increasing strength and proprioception of the affected limbs. Strength exercises may include longer leash walks, including up and down hills, stepping over objects such as poles/sticks, and walking over uneven surfaces such as sand, mulch, or tall grass. Initiate walking in circles and figure eights by starting with moderately sized circles, and decrease the diameter weekly until tight circles are achieved. Walking in a figure eight pattern may be done around cones placed 5 to 6 feet apart depending on the size of the dog. Additional therapeutic exercises include the following:

- Ladder—Use an extension ladder on a level floor or cavaletti rails. Walk the dog slowly through ladder or rails to ensure individual weight bearing on all limbs.
- Wheelbarrow exercises—Elevate both rear legs and have the dog walk forward on the front limbs only. Begin for short distances and increase as tolerated.
- Hydrotherapy—The use of underwater treadmill or swimming may be effective during this stage as well. NOTE: Full ROM of the elbow should be present and there should be no palpable tenderness/inflammation of the elbow or shoulder. Hydrotherapy sessions are performed two to three times/week and are initiated slowly, beginning with 5 minutes, and increased 1 to 2 minutes per session as tolerated.

Neuromuscular control exercises are initiated in this phase to enhance the muscles' ability to control the elbow joint during athletic activities. These exercises include proprioceptive neuromuscular facilitation exercises with rhythmic stabilizations and slow reversal manual resistance elbow/carpus flexion exercises. One method includes having the dog weight bearing and manually shifting the dog back and forth from a cranial to caudal direction on varying surfaces, and ambulating on uneven surfaces while applying manual forces.

The next phase involves a progression of activities to prepare the dog for return to off-lead activity and sport participation. The goals of this phase are to gradually increase strength, power, endurance, and neuromuscular control, to achieve a gradual return to sport. Specific criteria that must be met before entering this phase include full nonpainful ROM, no pain or tenderness, and strength and muscle mass that is 70% of the contralateral forelimb.

Advanced strengthening activities during this phase include exercises emphasizing high-speed and eccentric muscle contraction and plyometric activities. Elbow flexion exercises using elastic bands (Thera-Bands™) are progressed to emphasize eccentric control. Elbow flexion can be performed with elastic bands to emphasize slow and fast speed concentric and eccentric contractions. The elastic bands are placed on the cranial aspect of the mid humerus and the practitioner stands behind the dog while ambulating to offer eccentric resistance.

Plyometric drills are an extremely beneficial form of exercise for training the upper extremity musculature.[74] The physiologic principles of plyometric exercise use an eccentric prestretch of the muscle tissue, thereby stimulating the muscle spindle to produce a more forceful concentric contraction. Plyometric exercises are performed using jumps during the later stages of this phase to train the upper extremity musculature to develop and withstand high levels of stress. Examples of these activities include the use of a single jump exercise, then a series of jump grids consisting of four jumps, initially placed in a straight line, then in an arc, and progressing to a serpentine or threaded configuration. Another example is simply jumping on and off a box at several different angles.

Increasing time, distance, speed, or terrain during leash walks aids strengthening. Steeper hills and stairs may aid in advanced strengthening. Proprioception and stabilization exercises may be advanced with the use of wobble boards, BOSU®, and/or therapeutic ball exercises (Figure 33-26). Placing the dog on an unstable surface such as these further challenges the patient's proprioception as well as improves strength of the proximal forelimbs.[91] Progression of these exercises may include gentle rocking from side to side and having the patient follow a treat in all directions. Eccentric exercises such as walking through a tunnel and walking backward also may be added. This will challenge the cranial muscles of the shoulder and elbow.

The final phase of elbow rehabilitation, the return to activity phase, allows the dog to return progressively to full off-lead activity and competition using an interval training return to sport program. Sport-specific functional exercises are performed to prepare the dog for the stresses involved with each particular activity or sport.

Before a dog is allowed to begin the return to activity phases of rehabilitation, the dog must exhibit full ROM, no pain or tenderness, have equal forelimb muscle



Figure 33-26 Proprioception and stabilization exercises may be advanced with the use of a BOSU.



Figure 33-27 Manual balance perturbations in all directions are initiated on uneven surfaces such as a wobble board.

symmetry, no signs of lameness at a walk, trot, or when performing tight circles at a trot, and a satisfactory clinical examination.

If healing of all tissues has occurred appropriately, advanced strengthening techniques may be encouraged such as jumping (on/off a couch or over a jump). Longer walking or trotting sessions may be necessary to build up strength and cardiovascular endurance. Gradual off-leash activities may be initiated at this time. The duration of exercises may be increased, and additional manual balance perturbations in all directions may be initiated on uneven surfaces, such as wobble boards (Figure 33-27), BOSU, or therapy ball. Lifting one limb while performing these activities further increases the difficulty.

After achieving the previously mentioned criteria to return to sport, a formal interval sport program is initiated.[92] For the performance dog, training is initiated three times per week with a day off between sessions. Each step increase in activity is performed at least twice on separate days before the dog is allowed to progress to the next step. Full activity should be performed without pain or significant increase in clinical signs (lameness, joint effusion, and discomfort on ROM). If the dog experiences worsening clinical signs at a particular step of the program, the owner/trainer is instructed to regress to the prior step until the signs subside. It is important for the performance dog to perform active stretching before training and performance and to do mild warm-up activity, such as jogging. This provides an adequate warm-up, but also ensures maintenance of necessary ROM and flexibility of the forelimb.

Advanced strengthening and proprioception must be accomplished if return to sporting activities is a goal. Advanced training and rehabilitation may be accomplished with activities such as plyometrics, sprinting, jumping, agility training, and sport-specific training. Greater balance perturbations on the BOSU or therapy ball; quick directional changes, pivoting, figure eight exercises at submaximal effort initially, then progressing to greater speeds; cardiovascular endurance; and a continuation of strength training should be targeted.[93]

Hindlimb Conditions

The most common hindlimb orthopedic/sports medicine conditions afflicting performance and working dogs are iliopsoas muscle strain and cranial cruciate ligament (CCL) insufficiency. Cranial cruciate ligament insufficiency is presented elsewhere in this text. Presented in the following are the most common muscular injuries/conditions of the hindlimb: iliopsoas strain and gracilis and semitendinosus/semimembranosus contracture. Iliopsoas strain is seen much more frequently than gracilis or semitendinosus/semimembranosus contracture. Because contracture of the gracilis or semitendinosus/semimembranosus is less common, it is often undiagnosed until significant pathology is established. Diagnosis and treatment in the early stages of the disease process can significantly inhibit the progression, emphasizing the need for early identification.

Iliopsoas Strains

Acute, Stretch-Induced Injuries

Iliopsoas strain is an overstretching injury. It is estimated that muscle injuries account for more than 30% of all

injuries seen in a typical human sports medicine practice, and has been reported to be the most common injury seen in human general practices.[7,94] Laboratory reports indicate that muscles that cross multiple joints or that have complex architecture have increased susceptibility to strain injury.[7] Strain injuries result from either excessive stretching or stretching when the muscle is being activated.[94] The injured muscle is weaker and has reduced tension, increasing the risk of reinjury.[7,94] Appropriate treatment and allowing for complete healing is a imperative to prevent reinjury. Rehabilitation therapy initially focuses on relieving pain and inducing healing followed by muscle strengthening, endurance and flexibility. A complete program should also include educating owners on proper warm-up and stretching techniques to further reduce the incidence of reinjury.

Anatomy

The psoas major and the iliacus muscles combine to form the iliopsoas muscle (Figure 33-28). The psoas major muscle arises from the transverse processes of the lumbar vertebrae at L2 and L3 and the bodies of L4-7, and the iliacus muscle arises from the ventral surface of the ilium. They have a common insertion on the lesser trochanter of the femur. The action of this muscle is to move the pelvic hindlimb forward relative to the trunk via hip flexion.[22]

Cause

Iliopsoas muscle strains occur as the result of excessive force acting on the muscle, and are commonly associated with highly athletic activities such as agility.[94] These injuries often occur at or near the muscle-tendon junction. Eccentric contraction, in which the muscle is contracting while it is stretched, is thought to be an important factor in the development of these acute strain injuries.[95] Traumatic incidents that result in active eccentric muscle contraction, such as slipping into a splay-legged position, jumping out of a vehicle, aggressive agility training, or roughhousing with other dogs, are often reported to precipitate acute lameness. It is not uncommon to find concurrent orthopedic problems or a recent history of surgical treatment for another orthopedic condition, such as cranial cruciate ligament rupture.

Diagnosis

The history for dogs with iliopsoas strains commonly includes a lameness ranging from a subtle intermittent offloading of the hindlimb to significant unilateral hindlimb lameness that is exacerbated with activity. These dogs commonly demonstrate performance issues such as knocking down the bars of jumps with the hindlimbs or slowing in the weave poles.

On direct palpation, tightness, discomfort, and spasm of the myotendinous unit may be noted (Figure 33-29). Pain and spasm may also be noted when stretching the myotendinous unit by either placing the hip in extension with abduction or by simultaneous extension of the hip with internal rotation of that pelvic hindlimb (Figure 33-30).

Radiographs are of little value in the early phase of the condition but may reveal mineralization just cranial

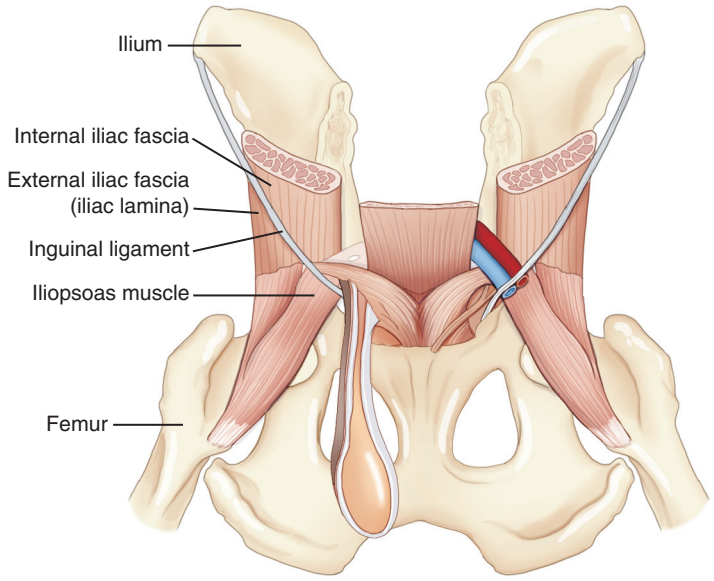


Figure 33-28 Iliopsoas anatomy.



Figure 33-29 Palpation of the iliopsoas.



Figure 33-31 Laser therapy for an iliopsoas strain.



Figure 33-30 Iliopsoas passive stretch.

to the lesser trochanter in chronic cases. The use of advanced imaging modalities to demonstrate lesions of the affected muscle and/or tendon can increase confidence in the diagnosis. Ultrasonography is a relatively inexpensive noninvasive imaging modality for canine musculoskeletal evaluation with the additional advantages that general anesthesia is not required and response to treatment can be monitored with follow-up imaging. This imaging modality depends on the expertise of the operator, which may limit its practical application in some settings. Advanced diagnostics such as CT scan and MRI may be used to identify iliopsoas strains and both are widely used to diagnose acute, stretch-induced muscle injury in human patients.[96] Although CT is valuable for imaging soft tissue lesions, the use of MRI has greatly increased the ability to detect subtler lesions.

Treatment

Acute iliopsoas strains should be treated conservatively. Skeletal muscle relaxants may be administered in severe cases to reduce pain and muscle spasms. Medical management may also include NSAIDs, cryotherapy, and controlled activity. Rehabilitation can be very effective in treating iliopsoas strains. Treatments may include laser therapy to increase circulation, remove waste products, and promote healing (Figure 33-31). Pain-free PROM and high-repetition exercise also are recommended. Acute strain injuries should not be stretched because microtears may occur. Core strengthening exercise is essential in the return of the athletic dog to competition and a pain-free lifestyle. Exercises using a therapy ball should be performed to increase the dog's core muscles, including the lower back and abdominal muscles. Because the iliopsoas originates from the lumbar spine, the lumbar spine and lumbosacral area may need to be treated in acute cases. Passive range of motion, interferential electrical stimulation, stabilization exercises, and gradual increases in weight bearing of the involved limb are recommended. Active ROM and strengthening exercises are added next. Strengthening exercises might include stepping over cavaletti poles, hemi-standing (lifting both front and hindlimbs on one side of the dog's body while balancing on the contralateral limbs), standing with the front paws on an elevated surface, and use of the wobble board. Hill walking should be added to increase the strength of the pelvic limb musculature. A steep hill of approximately 20 to 40 degrees in incline should be used to slowly walk the dog up the hill. It is important that the dog walks, and does not bunny hop or trot, up the hill. Slow walking promotes an equal balance of each hindlimb and focuses on the hamstring and the gluteal muscles. The dog should be walked up the hill approximately 100 feet and then slowly walked down.

The downward motion works the iliopsoas muscle in an eccentric fashion. This can be repeated three to five times and repeated a few times during the week. Acupuncture may be helpful to assist with pain control and to promote healing, including the lumbar and lumbosacral region. Joint mobilization and other manual therapy may also be needed to assist with lumbar spine ROM and motion of the coxofemoral joint.

In chronic iliopsoas strains it is important to reinitiate the inflammatory process to assist in remodeling of the tendon fibers. Nonsteroidal antiinflammatory drugs should be avoided with chronic iliopsoas strains as they impair the inflammatory response. Rehabilitation therapy is recommended with chronic iliopsoas strains. Modalities might include heat, ultrasound, and laser, followed by massage therapy. Be sure to check for pain or discomfort in the lumbosacral joint and lumbar region (lower spine) during manipulation. Chronic iliopsoas strains may arise from a problem with mechanics; therefore working on correcting the mechanics of movement may help to reduce the strain on the iliopsoas and contribute to its healing. If movement mechanics are not corrected, the condition may not improve. The exercise progression is similar to that for acute iliopsoas strains, but initiating stretching (hip extension with abduction) after modalities and massage is advised, as are longer walks. In chronic muscle strain injuries it usually takes longer to recover and progress through the stages of healing and exercise because of the chronic nature of the changes in the myotendinous unit. Education of owners/trainers is extremely important because they should be instructed to move the dog slowly through the phases of recovery. Core strengthening and activities focusing on strengthening of the gluteals and hamstrings, as well as the eccentric strength of the iliopsoas, should be emphasized. The underwater treadmill, hiking, and hill work are all appropriate. With regard to aquatic therapy, swimming may aggravate the iliopsoas injury because it forces the body to maintain the hip in a shortened or flexed position with more rapid movement of the limbs than occurs with walking on an underwater treadmill.

When returning to agility training, weave poles and tight turns at full jump heights should be avoided during the early stages of retraining. The risk of reinjury to a previously strained muscle is well established in human patients when a previous minor injury often predates a major strain injury. The risk of more significant strain is increased when preexisting strain injury has not completely healed. This also appears to be the case in canine patients, in whom pain from iliopsoas strain injuries may recur. Appropriate warm-ups, stretching, and retraining are extremely important in preventing injury and in returning the dog to a competitive performance level. Examples of retraining techniques for iliopsoas strains include starting with low, straight-line jumps, and later incorporating very wide sweeping turns that progress over time to higher jumps with tighter angles. Do not include higher jumps and weave poles until late in the retraining period. It has been deduced from human rehabilitation that stretching without an appropriate warm-up is detrimental to both the dog's tissues and its performance. In fact it is much more important to do an appropriate warm-up than stretching if time does not permit both. Following training and performance, make sure to implement iliopsoas stretching techniques (hip extension with abduction) and use an appropriate cool-down with ice therapy (5 minutes on, 5 minutes off, 5 minutes on).

Regenerative medicine therapy consisting of a combination of stem cells and platelet rich plasma may be considered for those cases refractory to conservative medical management and rehabilitation therapy. Injections should be performed with ultrasound guidance to ensure proper placement of cells. This author has had success with this treatment followed by an appropriate rehabilitative therapy program. In more severe cases, additional injections may be needed.

Surgical treatment may be warranted for those that do not respond to conservative medical management and rehabilitation therapy. In cases where there are irreversible changes to the myotendinous unit, such as fibrosis (forming excess fibrous tissue while healing) of the muscle-tendon junction, surgical treatment by tenotomy/tenectomy (releasing the tendon) or reattachment may be indicated.[97] Surgical intervention should also be considered when the strain injury recurs at regular intervals, although the lesion should be first confirmed with ultrasonography, CT, or MRI imaging. Good to excellent results have been reported with dogs returning to function, although performance dogs may work at a lower level.

Gracilis and Semitendinosus/Semimembranosus Myopathy/Contracture

Muscle contracture is defined as "The abnormal shortening of muscle tissue, rendering the muscle highly resistant to stretching."[98] A contracture can lead to permanent disability.[98] There is a significant difference between muscle contraction and muscle contracture. *Contraction* refers to the normal physiologic process of muscle shortening, resulting in work. *Contracture* refers to the abnormal pathologic process resulting in fibrosis and permanent damage to a muscle. It is important to remember that a muscle can have a significant amount of scar tissue from prior injury and still not be considered a contracture based on its ability to function. The pathologic process of muscle contracture is characterized by replacement of most or all of the entire muscle and/or associated tendon with fibrous connective tissue. This process can take weeks to months and leads to shortening of the affected muscle or muscle group. A muscle contracture can also severely affect the associated joints by fixing them in flexion or extension. In most cases, response to treatment and

prognosis is better for muscle contractures of the forelimb than the hindlimb.

Canine muscle contracture is reported to affect several different muscles, is associated with a number of predisposing factors, and has a varied prognosis depending upon which muscle is affected. Most patients suffer some form of trauma weeks to months before the contracture is present. The clinical signs include lameness, pain, weakness, decreased ROM, firmness noted throughout the entire muscle, and usually a characteristic gait. Predisposing factors for muscle contracture include compartment syndrome, infection, trauma, repetitive strains, fractures, infectious diseases, immune-mediated diseases, neoplasia, and ischemia. Contractures of the gracilis and semitendinosus/semimembranosus may occur individually or concurrently. History associated with a contracture of the gracilis and/or semitendinosus/semimembranosus typically consists of a single or repeated strain injury[99] with a gait abnormality developing after. Gait abnormalities usually start abruptly and progress over a period of 6 weeks to months,[40,99] at which time the condition becomes static. Locomotion following gracilis contracture is characterized by raising the affected leg in a jerky fashion with the hock hyperflexed and rotated laterally, the metatarsus rotated medially, and the overall stride shortened.[40,99] A shortened stride with a rapid medial rotation of the paw, external rotation of the hock, and internal rotation of the stifle mid-stride is noted with contracture of the semitendinosus.[99] There appears to be some breed and age predilection; however, the sex of the animal does not have an appreciable influence. Gracilis muscle and semitendinosus/semimembranosus contracture most often affects highly active German shepherd dogs and shepherd-related breeds between the ages of 3 and 7 years.[40,99,100] The suspected cause in the working and performing German shepherd is repetitive strain injury leading to secondary contracture.

Anatomy

The gracilis is a broad muscular sheet that arises from the pelvic symphysis, extends superficially along the caudal aspect of the inner surface of the thigh and tapers to its end along the entire length of the cranial border of the tibia. An aponeurosis expands out into the crural fascia. From the caudal border, a well-developed reinforcing band extends to the calcanean tendon with attachment to the tuber calcaneus. The gracilis is responsible for adduction of the thigh, extension of the hip, flexion of the stifle, and extension of the hock.

Arising from the tuber ischium, the semitendinosus inserts distally on the medial surface of the tibia, with a second insertion by means of a fascial attachment to the tuber calcaneus. It is responsible for extension of the hip and tarsal joints and flexion of the stifle joint in the free non–weight-bearing limb. The semimembranosus also arises from the tuber ischium then extends distally with insertion on the caudal surface of the femur distal to the pectineus, and to the proximo-medial aspect of the tibia. It is responsible for extension of the hip and flexion of the stifle joint in the free non–weight-bearing limb.

Cause

Skeletal muscle tissue cells have a very limited ability for regeneration.[101] Repair to damaged muscle tissue begins early in the inflammatory cascade and involves two processes: regeneration of injured tissue by parenchymal cells of the same type, and replacement by connective tissue (fibroplasia).[101] The balance of the two dictates the amount of new skeletal muscle tissue or scar tissue produced. It appears to be this limitation for skeletal muscle cell regeneration that drives the formation of scar tissue and inevitable fibrosis after skeletal muscle trauma instead of regenerating new cells to replace the damaged muscle.

The exact cause of fibrotic myopathy is usually unknown. The fibrotic myopathy may result from acute trauma, chronic repetitive trauma, autoimmune disease, drug reactions, infections, neurogenic disorders, and vascular abnormalities.[102] Fibrosis and contracture may also result from ischemia secondary to indirect trauma.[102,103] Histologic examination reveals dense, collagenous connective tissue in place of muscle.[102]

Muscle strains can be characterized by tearing of muscle fibers, most often at or near the musculotendinous junction, that result from excessive force or stress to the muscle.[104] Indirect muscle injuries in humans often occur subsequent to rapid acceleration during athletic activities.[104] The type and severity of injury directly correlates to how healing will occur; predominantly by regeneration of functional myofibrils or scar tissue formation.[97] Fibrosis and contracture with minimal regeneration results when muscle damage is severe. Fibrous scar tissue provides tensile strength and is involved in the natural healing process of muscle tissue, however, when excessive, scar tissue limits regenerative healing and interferes with muscle contraction and relaxation resulting in mechanical lameness of varying degrees.[99,100,103,105,106]

Susceptibility of this disorder appears greater in active dogs. Therefore it is reasonable to suspect the same mechanism of injury may play a role. Findings from a 2002 study of canine athletes appears to support this theory suggesting that excessive activity can lead to muscle strains with resulting inflammation, edema, localized hemorrhage, and eventually fibrosis.[100]

Muscle injuries in dogs may be underestimated because of the failure to establish a definitive diagnosis, poor recognition of muscule damage when accompanied by concurrent, more severe trauma, and problems with the classification of muscle trauma.[99,101,105] Muscle injuries are said to account for only 5% of reported musculoskeletal disease.[107] Lameness is usually more intense in the acute phase and improves with time. When injured muscle

undergoes fibrous contraction, mechanical lameness may remain.[100,105,108]

Diagnosis

The unique and consistent presentation of gracilis and/or semitendinousus/semimembranosus contracture allows for effective diagnosis by way of a thorough history, observation of gait and physical examination.

Most contractures have a history of acute injury or lameness weeks to months before the onset of contracture. The lameness and initial swelling usually subside with supportive treatment. However, clinical signs of the contracture occur a few weeks later. In most cases there is a distinctive hind limb gait abnormality in affected dogs. The gait is characterized by a shortened stride with a rapid, elastic medial rotation of the paw, internal rotation of the hock with external rotation of the calcaneus and internal rotation of the stifle during the mid to late swing phase of the stride.

On physical examination a firm mass within the gracilis, semitendinosus, or semimembranosus muscle(s) and a taut, firm band along the caudomedial aspect of the thigh are typically palpable. A stretch of the gracilis-semitendinosus muscle complex (hip flexion and stifle extension with limb abduction) may elicit pain and spasm. Stifle extension may be significantly limited in severe cases.

A complete chemistry panel and CBC are usually within normal limits, although creatinine phosphokinase (CK) may be elevated. Radiographs of the hindlimb are typically normal; however, an increased radiopacity may be observed between the muscle and the lower tendon junction in some cases. A slightly echogenic muscle may be noted on ultrasound; however, established fibrosis tissue will appear hyperechoic. Magnetic resonance imaging also may be useful for diagnosing this condition in both the acute and chronic phases. Histopathologic examination of the contracted muscle reveals replacement of degenerating myofibers with connective tissue. The findings are consistent with primary myopathy caused by polyphasic muscular damage (degeneration, segmental necrosis, phagocytosis, and fibrosis).

Treatment

Medical management before or in lieu of surgery was attempted in a previous report with no apparent response.[6] Fifteen dogs had one or more surgical procedures. Although transection, partial excision, or complete resection of the affected muscle resulted in resolution of lameness following surgery, lameness recurred 6 weeks to 5 months (mean, 2.5 months; median, 2 months) following surgery. Adjunctive medical treatment did not prevent recurrence. Variable replacement of the affected muscle with fibrous connective tissue (predominantly along the caudolateral border of the muscle) was evident grossly, and replacement of myofibers with fibrous connective tissue was confirmed histologically. A definitive etiology could not be established. Myectomy of the entire gracilis muscle still has a poor prognosis because the gait abnormality may return within 3 to 5 months due to semitendinosus involvement.[6,40]

Aggressive rehabilitation following surgery is necessary to achieve optimal results. Rehabilitation therapy may consist of continuous ultrasound therapy, manual therapy and a home therapeutic exercise program including massage and ROM/stretching. Although rehabilitation therapy may significantly improve the gait abnormality and function of the hindlimb in chronic cases, it may not lead to complete resolution of clinical signs. The sooner the condition is recognized, and treatment is initiated, the better the outcome. Working shepherds with this condition may continue to remain on active duty; however, a continued rehabilitation and home maintenance program is crucial. Several alternative therapies are currently under investigation including shockwave therapy and botox injections. Stem cell therapy is also being considered due to its anti-fibrotic benefits.

Education for all German shepherd and shepherd-related breed owners and handlers is an important component to prevention. Appropriate warm-up, active stretching followed by cool-downs and passive stretching and massage should be performed whenever possible before working and events.

Conclusion

Sporting dogs can have a wide range of sport-related conditions and injuries. A sound knowledge base of structure and biomechanics and of the sport aids in successful rehabilitative management. In addition, rehabilitative needs may change as the patient progresses through therapy and returns to activity. Therefore appropriate and successful rehabilitative management requires constant reevaluation of the patient and modification of the rehabilitation therapy program to address those changing needs.

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34 Neurologic Conditions and Physical Rehabilitation of the Neurologic Patient

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General Considerations

Typical objectives in rehabilitating the patient with neurologic disease are to minimize pain, reestablish normal neural pathways, prevent secondary complications, and ultimately return the animal to independent function. It is also essential not to worsen neurologic function or pain in patients with spinal instability.

Neurologic conditions are often very painful. Adequate analgesia is important to achieve a successful outcome. Pain-free animals are relaxed and cooperative, recover faster and more completely, and owners are much happier and more compliant with recommendations when their pet is comfortable. Pain originates from multiple sources. Nociceptors respond to a variety of stimuli, including mechanical (e.g., pinching a toe), thermal (heat, cold), and chemical. Activation of superficial mechanical or thermal nociceptors initiates sharp, pricking pain that is precisely localized and ceases when the stimulus is no longer present. Increased intensity of stimulus activates slower conducting pain felt as burning or throbbing that is poorly localized. With tissue injury and inflammation, this type of pain persists even after the initial injury. Patients with neurologic disease can develop neuropathic pain, including spontaneous pain, paresthesia (tingling, prickling), and dysesthesia (paroxysmal shooting, shocklike pain or continuous burning pain). One of the most common causes of neuropathic pain is intervertebral disk herniation that compresses or stretches a nerve root.[1]

Analgesic and antiinflammatory drugs are important in managing pain in neurologic patients. Rehabilitation modalities are used to supplement medications and reduce drug requirement. Cryotherapy (see Chapter 18) reduces pain through the gate control theory described by Melzack and Wall that is governed by balance between excitation and inhibition in the dorsal horn, slowed nerve conduction velocity and increased threshold to nerve stimulation. Duration of analgesia lasts at least 30 minutes posttreatment providing an opportunity to perform passive manipulation. Transcutaneous electrical nerve stimulation (see Chapter 20) also works under the same gate control theory (Figure 34-1). Laser therapy (see Chapter 21), and massage (see Chapter 27) may also reduce pain and enhance relaxation.

Restoring normal function entails a number of exercises, including assisted standing, walking, and swimming. The therapist supports the patient as necessary to give the animal confidence and security when standing and walking. The center of gravity is just behind the scapula, meaning more weight is borne by the thoracic limbs than the pelvic limbs in normal animals. The best way of supporting the animal is to relieve weight from the affected limb(s) and maintain the center of gravity in the correct place by supporting the patient under the sternum and under the pelvis. This minimizes stress on the lumbar vertebrae and allows the cervical vertebrae to move freely. Patients are not supported under the abdomen because this places pressure on abdominal organs and stresses the lumbar vertebrae. Similarly a neck collar is not used to support the patient because this puts pressure on the trachea and cervical vertebrae. A harness that supports the thorax and another to support the pelvis are helpful in larger patients.

When the patient is standing, the therapist helps place the limbs in a normal position. The normal stance is a rectangular configuration in which the feet are at each corner of the rectangle, with each thoracic limb directly under the shoulders and each pelvic limb under the pelvis. The spine should be in a straight line from the head to the tail. With thoracic limb pain, the patient redistributes the weight to the other three limbs, shifting weight and center of gravity caudally, resulting in a stance in the configuration of a triangle. The affected limb is placed laterally, cranially, or caudally and the spine curves toward the unaffected side to maintain equilibrium. This posture overloads the hips and knees. When a thoracic limb is paretic and not painful (e.g., brachial plexus avulsion), the affected limb may be placed caudally and the neck and head may drop ventrally, shifting the center of gravity cranially.

If one pelvic limb is affected, the other pelvic limb is placed medially, the thoracolumbar spine curves to the unaffected side, and the head and neck drop ventrally, shifting the center of gravity cranially. This increases stress on the biceps tendons, elbows, and carpi. When both pelvic limbs are paretic they tend to be placed medially and the thoracic limbs are placed laterally and caudally, with the paws pointing laterally. The head and neck drop ventrally head to shift the center of gravity cranially. This overloads

From Olby N, Halling KB, and Glick TR: Rehabilitation for the Neurologic Patient, Vet Clin North Am Small Anim Pract 35(6):1389-1409, 2005.

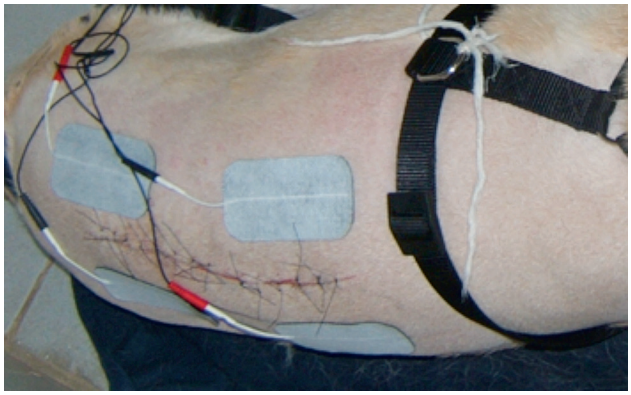


Figure 34-1 Transcutaneous electrical nerve stimulation applied to a patient for pain control.



Figure 34-2 While the patient is eating, the stance may be corrected and gentle weight shifting exercises may be performed.



Figure 34-3 Dry, well-padded bedding and frequent repositioning of recumbent patients help prevent pressure sores.



Figure 34-4 Patients with loss of sensation or abnormal sensation are at risk of damaging their limbs from dragging or self-mutilation. The affected limb(s) should always be protected to prevent trauma.

the shoulders, biceps tendons, elbows, and carpi. The thoracolumbar spine is curved dorsally (kyphosis).

A good time to correct the stance is meal times. Although the patient is occupied with eating, gently correct the stance to a normal position (Figure 34-2). Repeat this as needed to maintain a proper stance.

Neurologic patients are at risk of several complications including shortening of ligaments and tendons, muscle atrophy and contracture, pressure sores, urinary tract infections, and respiratory tract infections that can develop secondary to immobility, recumbency, and altered biomechanical stresses. Modalities such as passive range of motion (PROM) and stretching exercises decrease the risk of contractures. Dry, well-padded bedding and frequently repositioning recumbent patients help prevent pressure sores (Figure 34-3). Bony prominences such as the ischiatic tuberosity, greater trochanter, and xiphoid are inspected frequently to detect early skin lesions. Urine retention is prevented by urethral catheterization or manual bladder expression (see Chapter 16). Helping the patient maintain a normal, upright posture as much as practical minimizes many of the adverse effects of recumbency. Patients with loss of sensation or abnormal sensation are at risk of damaging their limbs from dragging or self-mutilation (Figure 34-4). In these patients, the affected limb(s) should always be protected to prevent trauma.

Specific neurologic diseases and their management are discussed in the following sections.

Intervertebral Disk Disease

Intervertebral disk (IVD) disease is a syndrome of pain or neurologic deficits, resulting from displacement of part of the disk. Disks are interposed between each vertebral body except the first and second cervical vertebrae and each of the fused sacral vertebrae. The IVD is situated between adjacent vertebral end plates and is composed of an outer fibrous ring, the annulus fibrosus, which surrounds an eccentric amorphous gelatinous center, the nucleus pulposus.[2]

Intervertebral disk herniation is a general term that refers to displacement of a portion of the disc. Although a normal disk can herniate as a consequence of major trauma, most herniations are secondary to preexisting degeneration of the disk. Disk degeneration is associated with decreased water and proteoglycan concentration. This reduces the ability of the disk to function as a hydraulic cushion and predisposes to displacement of a portion of the disk. There are two types of disk degenerations, chondroid and fibrous metaplasia. Chondroid metaplasia is most common in chondrodystrophic breeds and causes a progressive transformation of the gelatinous nucleus pulposus to hyaline cartilage. This type of degeneration predisposes to disk extrusion where disk material is displaced beyond the outer edge of the annulus (also called Hansen Type I).

Fibrous metaplasia is an age-related degenerative process that occurs in any breed but is more common in nonchondrodystrophic dogs 7 years of age and older. It is characterized by a fibrous collagenization of the nucleus pulposus and degeneration of the annulus fibrosus. Fibrous metaplasia predisposes to disk protrusion, in which disk material is displaced from the disk space but is contained within an intact annulus (also called Hansen Type II). Nonchondrodystrophic breed dogs can also suffer extrusions and less commonly chondrodystrophic breeds suffer protrusions.[2,3]

The lifetime prevalence for intervertebral disk disease is approximately 3.5% and overall mortality rate attributed to disk disease is about 1%. Miniature dachshunds have the highest risk, with a lifetime prevalence of 20% and other chondrodystrophic breeds are also at increased risk.[4]

Diagnosis is based on clinical features and imaging studies, including radiography, myelography, computed tomography (CT), and magnetic resonance imaging (MRI). Survey radiographs are useful at ruling out other diagnostic possibilities, such as fractures/luxations, vertebral tumors and diskospondylitis, but are usually not accurate enough for surgical planning for intervertebral disk disease. Myelography is more accurate than survey radiographs but carries a risk of complications such as seizures and worsening of neurologic signs. Computed tomography is accurate for acute extrusions in chondrodystrophic breeds and is noninvasive and usually faster than myelography. However, CT is not as sensitive for noncalcified herniations. Magnetic resonance imaging is generally considered the best diagnostic modality and is accurate and noninvasive, but is also expensive.

Cervical Disk Disease

Cervical disk disease commonly affects small, middle-age to older chondrodystrophic breeds, with beagles and dachshunds having the highest risk.[4,5] The C2-C3 disk space is most commonly affected, with the frequency decreasing at progressively more caudal disk spaces. Cervical disk disease in large-breed dogs is usually associated with cervical spondylomyelopathy and is discussed separately. Neck pain is the most common and often the only sign of cervical disk disease and is present in approximately 90% of affected dogs.[5] This is manifested as low head carriage, stiffness or decreased motion of the neck, vocalizing, and spasms of the neck muscles. Some dogs exhibit lameness of one or both thoracic limbs, owing to nerve root or spinal nerve attenuation (root signature). In severe cases, there is ataxia, conscious proprioceptive deficits, weakness, or paralysis of all limbs. Spinal reflexes are usually normal to exaggerated, although reflexes may be weak or absent in the thoracic limbs with caudal cervical disk extrusions and occasionally with more cranial lesions.[6] Other causes of cervical lesions include trauma, neoplasia, meningitis, discospondylitis, cervical spondylomyelopathy, and atlantoaxial instability.

Treatment options include surgery and nonsurgical therapy, with the decision based on the duration and severity of clinical signs. Nonsurgical therapy is indicated as the initial treatment in patients with no or mild neurologic deficits. The most important aspect of treatment is strict cage confinement to minimize stress on the damaged disk to allow the disk time to heal. A harness is used instead of a neck collar and leash. A short course of anti-inflammatory doses of corticosteroids or nonsteroidal anti-inflammatory drugs (NSAIDs) is administered if necessary for pain control. Muscle relaxants may be beneficial in some patients with spasm of the cervical muscles. Analgesic drugs without cage confinement are contraindicated because they may lead to increased patient activity and risk further disk extrusion. If the patient does well, cage confinement is continued for at least 2 to 3 weeks. About 50% of dogs with neck pain caused by disk disease recover with nonsurgical therapy. Recurrence of signs occurs in about one third of patients.[7]

Surgery is indicated for patients with substantial neurologic deficits, neck pain unresponsive to appropriate nonsurgical therapy, or recurrent bouts of neck pain. Removal of extruded disk material by a ventral slot to decompress the spinal cord and nerve roots is the procedure of choice in most patients. Dorsal hemilaminectomy may be necessary for lateral or far-lateral extrusions. In chondrodystrophic dogs, prophylactic fenestration of the other disk spaces at the time of ventral slot is indicated to decrease the risk of extrusion of another disk. Fenestration alone of the affected disk is not indicated because it does not allow removal of extruded disk material.[8] In dogs with neck pain and mild neurologic deficits, the success rate with surgery is about 95%. Nonambulatory dogs have a good outcome in 62% to 100% cases, with small dogs (≤15 kg) having a better prognosis than larger dogs.[5,8,9] Preoperative, intraoperative, and postoperative management included drugs described for medical management. Postoperative physical rehabilitation is an integral part of the overall care of the patient.



Figure 34-5 Whirlpool with hydraulic hoist and sling.



Figure 34-6 Life preserver used for flotation.

Physical Rehabilitation for Cervical Disk Disease

The most severe cervical injuries cause tetraplegia with a compromise of respiratory function. It is imperative that respiratory function is evaluated in any tetraplegic animal and that respiratory complications (such as pneumonia or hypoventilation) are identified and understood before any exercises that may exacerbate the problem.

Physical rehabilitation as part of medical management includes massage, heat therapy, electrical stimulation (ES), therapeutic ultrasound (US), therapeutic laser for muscle spasms and pain relief. Whirlpool therapy may also be cautiously used in patients carefully supported in a sling as long as there is no struggling (Figure 34-5).

Postoperative therapy is staged and begins immediately or within 48 hours of surgery to relieve pain and muscle spasms. Modalities such as cryotherapy, ES, carefully administered US, laser and massage may be used based on the clinical needs of the patient. In ambulatory patients, neck leashes should not be used, and leashes should be attached to a harness or across the chest and under one thoracic limb.

Methods of actively eliciting cervical motion begin in the first few days after comfort is achieved and there is no pain on movement of the neck. Active range of motion can be achieved using toys or treats to motivate the patient to turn the head and neck in all directions. Begin with smaller movements and progress to the point that the patient is able to take a toy or treat from near the rear limbs for lateral excursion, between the forelimbs for cervical flexion, and high above the head for extension. Pain-free PROM, stretching, and massage are used to help treat muscle spasms.

Resistive exercises are used in tetraparetic, paraparetic, and hemiparetic patients. Pushing on the bottom of the foot of an extended extremity elicits a flexor withdrawal reflex causing flexion of the limb that stimulates muscles, tendons, ligaments, joints, and bones of the limb. While maintaining limb extension and force on the foot, move the limb slightly in all directions. As the reflex causes the limb to retract, active, resistance can be achieved by the therapist holding the foot, creating a gentle "tug-of-war" in which the patient is pulling more forcefully to withdraw the limb from the therapist's grip. This exercise should be performed for three to five repetitions per limb, three to four times per day.

Assistance should be provided to patients that are able to stand and support weight to attain correct limb placement. Careful use of a sling and hoist may be useful in larger nonambulatory patients. Necessary support through the chest is provided as needed to invoke as much independent weight bearing as possible. Patients are assisted in slow ambulation by placing and moving any limbs with motor deficits. Assisted walking (tail, sling), proprioception stimulation, and limb placement exercises facilitate neuromuscular re-education and proprioceptive functioning, as well as muscle strengthening.

Spinal walking is a phenomenon that occurs in cats following surgical transection of the spinal cord and occurs in some dogs after an injury that causes severe spinal cord injury or transection. While these dogs do not regain normal motor function, spinal walking may be a functional mode of ambulation for short distances.

For strengthening, underwater treadmill walking or swimming may be initiated after the incision site is healed. The time of suture removal is generally a safe time to start hydrotherapy. Some therapists start some form of aquatic therapy earlier, with caution to avoid submersion of the incision, especially an incision in the ventral cervical region. In any case, the incision should be sealed, with apposed edges, no discharge or drainage, and no separation of the incision with gentle manipulation of the edges. In small dogs, a whirlpool may be used for aquatic therapy. The duration of aquatic therapy depends on the fitness, endurance, and ability of the patient. Assistance or life preservers are provided as required (Figure 34-6). If the patient does not actively move its limbs through an

adequate range of motion, assistance is given either by passively moving the limb(s) or by providing resistance to motion to stimulate the patient to engage the limb against the applied resistance.

Patients can be placed on exercise rolls to strengthen proprioception and joint stability. They may be further challenged with mild bouncing and slight movements of the ball to create an unstable surface (Figure 34-7). Requiring patients to walk on foam, bubble wrap, or an inflatable object or challenging them with a rocker board (Figure 34-8) will also help them to develop stability and proprioception. To assist in regaining kinesthetic awareness, patients can be challenged to step over objects of varying shapes and sizes (Figure 34-9). Small objects are initially used; the height and width are increased as the patient progresses. Strengthening the thoracic limbs can be achieved by ambulating on land, a dry treadmill (Figure 34-10), and deep water and underwater treadmill hydrotherapy. Additional strengthening exercises include walking down inclines with variable pitch, wheel barrowing (Figure 34-11), and increased weight bearing with an exercise roll beneath the pelvic limbs (Figure 34-12). Analogous exercises may be used to strengthen the rear limb muscles, such as walking up inclines, dancing, and weight bearing with the forelimbs on an exercise roll.

Therapeutic modalities are provided for a minimum of 3 weeks. Owners are instructed further in performing strengthening exercises after discharge. Continued rehabilitation modalities are often prescribed after discharge on a recommended weekly schedule.



Figure 34-7 Therapy roll used for improving conscious proprioception.

Thoracolumbar Disk Disease

Thoracolumbar herniations are most common from T12-T13 to L1-L2, although other areas may also be affected. dachshunds, cocker spaniels, and beagles and other chondrodystrophic breeds are at increased risk.[4] Type I extrusions are most common in these breeds. Type II protrusions are more common in large, nonchondrodystrophic dogs, but large dogs can also suffer acute, Type I extrusions.[10]

Onset of signs can be sudden or gradual and depend on the force, quantity, and location of the disk material and the duration of disk herniation. About 10% of affected dogs have back pain with no neurologic deficits. They are often reluctant to run, jump, or climb stairs and may have kyphosis. Neurologic deficits range from mild ataxia of the pelvic limbs to complete paraplegia with urinary and fecal incontinence and absent deep-pain perception. The neurologic deficits may be symmetric or worse on one side. Spinal reflexes are usually normal to exaggerated, but are weak to absent if the extrusion occurs caudal to L2-L3. In the most severe cases, progressive ascending-descending myelomalacia may result. In this syndrome, the spinal cord is damaged progressively cranial and caudal from the initial site. This is recognized in patients with an initial thoracolumbar lesion as a loss of pelvic limb reflexes, loss of abdominal muscle tone, cranial migration of the level of anesthesia and the cutaneous tunic reflex, thoracic limb paresis, and ultimately respiratory arrest. This is a grave condition that the therapist should be aware of if the neurologic status deteriorates.

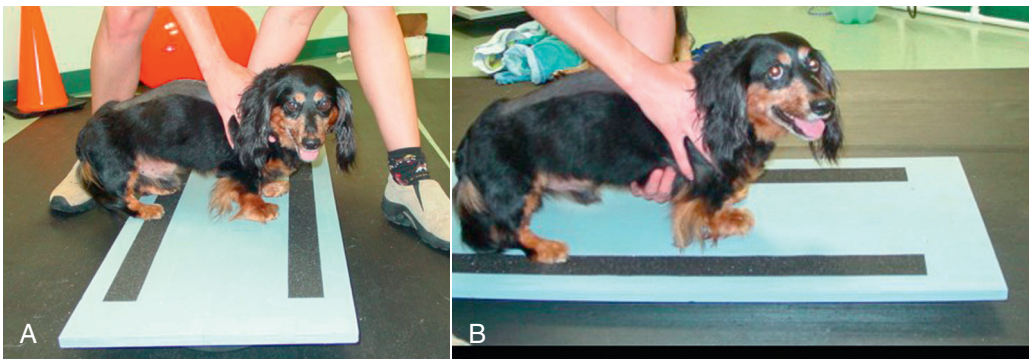


Figure 34-8 Rocker boards for rehabilitation. **A,** Side-to-side motion. **B,** Forward and backward motion.



Figure 34-9 Various-sized rails used as obstacles.



Figure 34-10 Land treadmill.



Figure 34-11 Wheelbarrow exercises.

Other diagnostic considerations for thoracolumbar spinal lesions include trauma, fibrocartilaginous embolism, degenerative myelopathy, discospondylitis, degenerative lumbosacral stenosis, neoplasia, meningitis, and orthopedic conditions such as severe hip dysplasia or bilateral cranial cruciate ligament insufficiency.



Figure 34-12 Facilitating patient standing using a therapy roll.

About 85% of dogs with back pain and no neurologic deficits will improve with nonsurgical therapy, as described for cervical disk disease. Surgery is indicated in patients with substantial neurologic deficits, or back pain refractory to nonsurgical therapy. Patients with a sudden onset of severe neurologic deficits need urgent surgery. Removal of extruded disk material via hemilaminectomy is the procedure of choice in most cases. Fenestration of the affected intervertebral disk space is routinely performed. Prophylactic fenestration of other thoracolumbar disk spaces reduces recurrence in chondrodystrophic breeds.[11] The prognosis depends in large part on the severity and duration of the neurologic deficits. The recovery rate for nonambulatory dogs with acute extrusions and intact deep pain perception is 85% to 95%, with most patients regaining ambulation in 1 to 4 weeks. For dogs with loss of deep pain perception, the recovery rate is about 50% if surgery is performed within 24 hours of the loss of deep pain. This decreases further if surgery is delayed for longer than 24 hours. The recovery rate for large breed dogs with chronic protrusions is much lower, ranging from 22% to 52%.[4]

Physical Rehabilitation for Thoracolumbar Disk Disease

Although physical rehabilitation has been used with pharmacologic intervention in the conservative management of acute and recurrent thoracolumbar disk disease with some success, it is important to perform minimal intervention and avoid motions that might risk further herniation of disk material for one month; the major application of therapeutic modalities is in the postoperative period. The initiation of therapy and protocols used are similar to those used for cervical disk patients, but the focus is on the pelvic limbs. Patients with thoracolumbar disk disease are more likely to have proprioceptive and motor deficits, requiring greater attention to balancing and strengthening activities. Additional nursing care is required such as

bladder management, extending the skills of the veterinary therapist and dictating close interaction between the therapist and veterinarian.

Therapeutic modalities and exercises prescribed depend on the stage of the postoperative period and the neurologic status of the patient. Control of pain and swelling and the beginning of exercise are the focus of the initial postoperative period. Swelling of the surgical site is addressed with cryotherapy. Electrical stimulation is applied for pain relief using interferential or premodulated interferential waveforms. Therapeutic ultrasound must be used with care over the thoracic and lumbar epaxial musculature to avoid generating excess heat over the laminectomy site and possibly damaging nerves and the spinal cord.

Depending on the neurologic deficits, the patient may be unable to sit, stand, ambulate, or urinate independently. Appropriate supportive care, including bladder management, must be carefully performed, and there must be close communication between the therapist and veterinarian. Occasionally patients have significant hyperesthesia along the thoracolumbar areas. In these cases, light massage and desensitization procedures may be useful. Patients with significant paraparesis or paraplegia may benefit from therapeutic exercises designed to help gain trunk control. One exercise is to assist patients to a sitting position with decreasing amounts of support until they are able to sit independently. After this is achieved balance can be challenged using gentle perturbation exercises on firm surfaces initially, and then progressing to unstable surfaces such as an exercise roll, foam mats, bubble wrap, or pillows. It may be helpful for the therapist to be positioned caudal to the patient and place the hands on the cranial stifle to help keep them extended to maintain a weight bearing position. Russian or biphasic ES to the muscles of the pelvic limbs may be used to induce stifle and hock extension and flexion, as tolerated by the patient.

Standing exercises may begin as soon as treatment is initiated. Appropriate support is provided while placing the patient in a standing posture to ensure some loading of the pelvic limbs and correct positioning of the feet. To promote correct stance, support the patient under the chest and pelvis and place the limbs in the correct stance twice daily while the patient is eating. This fosters continued extension of the limbs by proprioceptive neuromuscular facilitation and allows the patient to remain in the standing position for a longer period of time. This can be performed on a firm surface or with the patient supported by an exercise roll. Gently allow the dog to intermittently bear weight. If the patient tends to sink down, tickle the perineal region to stimulate extension of pelvic limbs. After the patient is able to maintain a standing position, pressure is applied to the pelvic limbs by pressing on the dorsal aspect of the pelvis. Gentle perturbations are applied from all angles while the patient is standing so that it is challenged to maintain stability and a standing position. Early ambulation may be assisted by moving the pelvic limbs in a reciprocal walking pattern.

Ambulation is allowed at controlled paces in patients with motor control. Assisted tail or sling walking and underwater treadmill hydrotherapy may be used to unload weight before attaining complete ambulatory independence. Strengthening exercises are initiated as independent ambulation improves (Figures 34-13 through 34-17). Activities include ascending and descending inclines, walking in circles or figure eights, walking on terrain with varying textures (carpet, grass, sand), stepping over objects of varying size to regain proprioceptive awareness, and practicing sit-to-stand activities for coordination. Nonslip flooring is ideal to encourage proprioception and appropriate limb placement. Commercially available booties may provide protection for dogs that tend to knuckle over on the dorsum of the paw or drag the paws while ambulating.

After the incision has healed, deep water hydrotherapy may be initiated. Before active movement of the pelvic



Figure 34-13 Facilitation of weight bearing on rear limbs using a therapy roll.



Figure 34-14 Platform used to facilitate limb placement.



Figure 34-15 Platform used to facilitate weight bearing and limb strengthening.



Figure 34-16 An individual limb exercise.

limbs, swimming benefits the trunk muscles, aiding the patient's overall stability. Active movement of the pelvic limbs may be elicited by applying resistance to the paw, stimulating the medial aspect of the stifle, or flexing the hock.

In patients that are unable to urinate voluntarily, preventing urine retention is essential. Initially, this may require intermittent or continuous urethral catheterization or bladder expression. After the catheter is removed the dog is taken outside three times daily to see if the patient can urinate voluntarily. Taking several dogs together will sometimes induce urination as the patient sees and smells other dogs urinate. If the patient does not urinate completely after an adequate amount of time, palpate to check bladder size. If full the bladder is manually expressed. Urine samples are periodically obtained to identify and treat any infection.

A minimum of 3 weeks of physical rehabilitation is recommended. Frequently this is extended to continue facilitating recovery. The degree of success in rehabilitation of thoracolumbar disk disease varies greatly and may take several months of dedicated treatment by the therapist and owner.



Figure 34-17 Dancing exercise.

Degenerative Lumbosacral Stenosis

Degenerative lumbosacral stenosis is a degenerative disorder resulting in stenosis of the vertebral canal causing compression of the lumbosacral nerve roots or cauda equina. In most adult dogs the caudal end of the spinal cord is located at the level of the L6-7 disk. The nerve roots originating in the L6, L7, sacral, and caudal spinal cord segments course from the distal end of the spinal cord to exit their respective foramina. This collection of nerve roots is called the cauda equina.

Degenerative lumbosacral stenosis occurs when soft tissue and bony changes, possibly in conjunction with abnormal motion of the lumbosacral joint, impinge on the nerve roots of the cauda equina. The most common change is protrusion of the L7-S1 disk, but osteophytes of the articular facets and hypertrophy of the interarcuate ligament and articular facet joint capsule may also cause nerve root compression.

Degenerative lumbosacral stenosis is most common in middle-aged, large-breed dogs. German shepherd dogs are the most commonly affected breed. Working dogs and other large-breed dogs, including boxers, rottweilers, and Doberman Pinschers, are also at increased risk.[12]

Pain and lameness are usually more prominent than frank neurologic deficits because stenosis affects the cauda equina, which is more resistant to damage from compressive forces compared with the spinal cord. The most common sign is lumbosacral pain, characterized by reluctance to work or participate in normal activity, run, jump, or climb stairs. Affected dogs often maintain a characteristic posture, keeping their lumbosacral joint flexed, which decreases nerve root compression. Tail weakness, urinary or fecal incontinence, and pelvic limb lameness or weakness can also occur. On examination, palpation or extension of the lumbosacral joint and elevation of the tail usually induces pain. Other findings may include a weak perineal reflex, proprioceptive positioning deficits in one or both pelvic limbs, and decreased hock flexion when eliciting a flexor withdrawal reflex. The differential diagnosis includes other lesions of the lumbosacral region (discospondylitis, neoplasia), degenerative myelopathy, fibrocartilaginous embolic myelopathy, orthopedic diseases (hip arthritis, cruciate rupture), trauma, and urogenital diseases, such as prostatic disease.

Diagnosis is based on clinical features and imaging. Survey radiographs may be normal or reveal chronic nonspecific changes to the lumbosacral region, including sclerosis of the vertebral end plates, ventral spondylosis deformans, and arthritis of the articular facets. Myelography is useful in assessing the caudal region of the lumbar spine, but will not identify lumbosacral stenosis when the dural sac ends cranial to the lumbosacral junction, which happens in about 25% of large-breed dogs. Although contrast radiography may provide evidence of static and dynamic compression, definitive diagnosis and localization are best made on CT and MRI[13] (Figure 34-18).

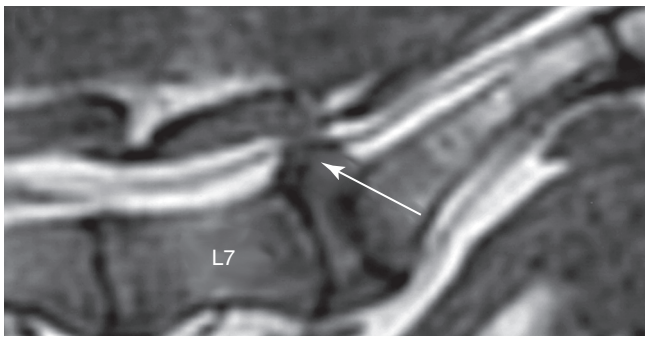


Figure 34-18 Sagittal plane MRI showing protrusion of the L7-S1 disk (*arrow*) with compression of the cauda equine. *L7*, The L7 vertebral body.

Nonsurgical treatment is appropriate initially for patients with mild pain and entails strict rest, antiinflammatory or analgesic medications, and weight loss if necessary. Epidural injections of glucocorticoids improve signs in many patients and avoid the complications associated with long-term administration of oral steroids.[14] Physical rehabilitation also may be useful in the medical management of lumbosacral disease.

Surgery is indicated when there is persistent pain or neurologic deficits. The most common procedure is dorsal laminectomy of L7-S1 with excision of the protruded disk and foraminotomy if necessary. Some surgeons also include fusion of the L7-S1, but it is unclear in which patients this is necessary. Resolution of pain is good, with 80% to 95% of patients improving postoperatively.[15-18] In working dogs, 67% to 80% of patients are able to return to work.[19,20] Only about 25% of patients with incontinence have a return of continence, and more than a 2-week duration of incontinence carries a poor prognosis.[16] Recurrence of clinical signs occurs in about 18% of patients.[15]

Physical Rehabilitation for Degenerative Lumbosacral Stenosis

Physical rehabilitation is beneficial as part of the medical and postoperative management of lumbosacral disease. In combination with medication, therapeutic modalities focusing on pain management, such as ES protocols and therapeutic US, massage therapy, and aquatic therapy are prescribed. Surgical patients are provided routine care of the incision site with icing and limited ES for pain management while hospitalized. Therapists should be informed of the type and location of any implants. Continued care consists of cage or crate confinement with assisted walking for 3 weeks after discharge. Physical rehabilitation then resumes following reassessment of the patient and radiographs if fusion was performed. Standard protocols are prescribed for strengthening and muscle re-education, including ES, passive range of motion, standing exercises, leash walking, proprioceptive and gait training, core muscle strengthening, and aquatic therapy (Figure 34-19). As with most protocols, therapy continues for a 3- to 6-week period and is lengthened if necessary.

Cervical Spondylomyelopathy

Cervical spondylomyelopathy, also called caudal cervical stenotic myelopathy or wobbler syndrome, is a disorder caused by abnormal development of the cervical vertebrae resulting in compression of the spinal cord. Middle-aged (7-9 years) Doberman pinschers and young (1-4 years) giant-breed dogs (Great Danes, mastiffs, rottweilers, Bernese, and Swiss mountain dogs) are most commonly affected.[21] The specific pathologic changes vary, but may include stenosis of the vertebral canal, bony enlargement

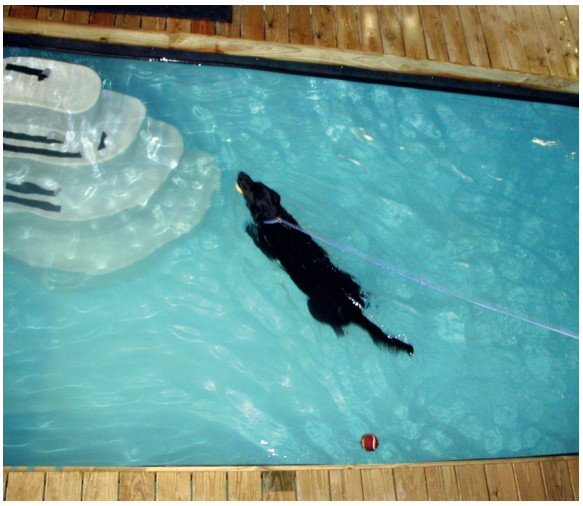


Figure 34-19 Deep water hydrotherapy.

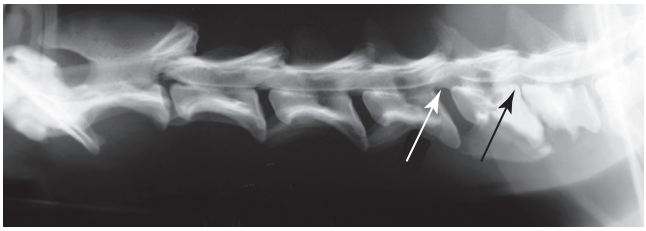


Figure 34-20 Myelography of cervical spondylomyelopathy. There is compression of the spinal segments dorsal to the C5-C6 disk space (*white arrow*). There is a second, less severe lesion dorsal to the *C6-C7* disk space (*black arrow*), which is narrowed.

of the articular facets, disk protrusion, capsular and ligament proliferation, synovial cysts, and vertebral instability. The C5-C6 and C6-C7 vertebrae and disk spaces are most commonly affected.

Clinical signs may be acute or slowly progressive. Mild cases are characterized by subtle ataxia of all limbs, often evident as swaying and increased stride length in the pelvic limbs, with a short-strided gait in the thoracic limbs. Because of the anatomic arrangement of the long spinal tracts to the pelvic limbs, the clinical signs are usually more pronounced in the pelvic limbs. Gait deficits are typically most evident at a slow walk and are less obvious at a fast walk or trot. Additional signs vary and may include low head carriage and neck stiffness. In severe cases there is paresis or paralysis of all limbs. Neck pain is variable, but is more likely in acute cases. The differential diagnosis includes congenital anomalies, trauma, meningomyelitis, discospondylitis, neoplasia, and degenerative, ischemic, and embolic conditions.

Definitive diagnosis is based on spinal imaging. Survey radiographs may be normal or show abnormally shaped vertebra, narrowed disk space(s), and osteoarthritis of the articular facets. Myelography, CT-myelography, or MRI is necessary to fully characterize the specific changes and is essential if surgery is considered (Figure 34-20). Hypothyroidism, von Willebrand disease, and cardiomyopathy are relatively common in middle-age Dobermans, so thyroid testing, buccal mucosal bleeding time, and chest radiographs are indicated in affected Dobermans.

Treatment is based on the severity of the neurologic signs, the results of diagnostic imaging, quality-of-life issues, and the wishes of the owner. Nonsurgical treatment consists primarily of exercise restriction to minimize vigorous activity that would exacerbate the dynamic component of spinal cord compression. Dogs are walked on a leash, and unsupervised activity is avoided. A body harness is worn instead of a neck collar. Antiinflammatory doses of corticosteroids such as prednisone are often helpful. In one study, 54% of patients treated medically improved and 27% remained unchanged over the long term.[22]

Surgery is indicated in patients with substantial neurologic deficits and those that do not adequately respond to nonsurgical treatment. The goals of surgery are to relieve any spinal cord compression and stabilize vertebral instability. The particular technique chosen is based on the specific changes evident on imaging and surgeon preference, and may include ventral slot decompression, dorsal laminectomy, or distraction and fusion of affected vertebrae. Overall 70% to 80% of patients improve after surgery, with a mean time to reach maximum improvement of 2.6 months. About 20% suffer recurrence. There is no clear difference among different surgical techniques.[22,23] Aftercare is a major part of the treatment, requiring significant intervention and effort of the caregiver.

Physical Rehabilitation for Cervical Spondylomyelopathy

Physical rehabilitation plays a significant role in patient care. Modalities directed toward decreasing muscle spasms, such as US and ES, passive range of motion of limbs, and support with a soft sling to provide assisted standing and weight bearing, may be used in nonambulatory patients. Assisted sling walking and assisted aquatic therapy are useful to help patients regain unassisted ambulation. Neuromuscular ES for muscle building and re-education is performed in selected cases. As ambulation improves and strength is gained, unassisted deep-water and underwater treadmill therapy may be started. Gait training and strengthening exercises are incrementally implemented. Modalities used must be compatible with the patient's neurologic status and the stage of wound healing in the postoperative period. Extended time in rehabilitation is often necessary for proper management of these cases because of the difficulties in managing large and giant breeds in a home environment.

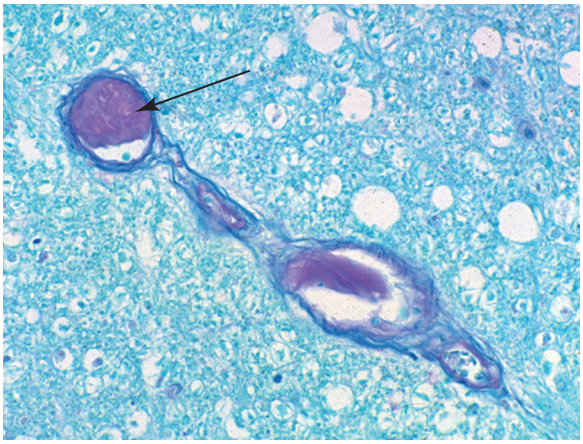


Figure 34-21 Photomicrograph of fibrocartilaginous embolic myelopathy. There is a piece of fibrocartilage (*arrow*) occluding one of the vessels in the spinal cord. There is necrosis of the adjacent portion of the spinal cord (hematoxylin eosin with PAS stain).

Fibrocartilaginous Embolic Myelopathy

Fibrocartilaginous embolic myelopathy (FCEM) is an acute infarction of the spinal cord caused by a vascular embolus of fibrocartilage, probably originating from the intervertebral disk (Figure 34-21). Adult large- or giant-breed dogs and miniature schnauzers are most commonly affected. This disease is less common in small dogs, chondrodystrophic dogs, and cats.

The onset is sudden and often associated with activity such as running or playing. Neurologic deficits rarely progress beyond the first few hours. Although patients sometimes yelp as if in pain at the onset of signs, spinal pain is rarely evident by the time of examination. This lack of focal spinal pain is helpful in ruling out other causes of acute spinal diseases, such as fracture/luxation and disk extrusion. Any region of the spinal cord may be affected, and the spinal cord segments involved dictate the specific neurologic deficits. Ataxia, paresis, or paralysis may affect the pelvic limbs or all limbs. Asymmetric or unilateral deficits are common. Lower or upper motor neuron deficits may result. In severe cases there is loss of deep pain perception caudal to the lesion.[24,25]

Diagnosis is based on the clinical features and exclusion of other causes. Essential features of FCEM are the signalment, sudden onset, nonprogressive course, lack of focal spinal pain, and often the asymmetry of deficits. The differential diagnosis includes intervertebral disk disease, trauma, neoplasia, and inflammatory disease. Radiographs and myelography are typically normal, although myelography occasionally shows focal spinal cord swelling.[24,25] Cerebrospinal fluid analysis is nonspecific. Magnetic resonance imaging is the best imaging modality and usually shows signal changes suggestive of focal spinal cord infarction.[26]

There is no specific treatment for this condition. Corticosteroids are sometimes used although there is little evidence that any drug is beneficial. Nursing care and physical rehabilitation play an essential role in promoting recovery and preventing complications. About 85% of patients recover, depending on the severity of the deficits and owner's commitment.[26]

Physical Rehabilitation for Fibrocartilaginous Embolic Myelopathy

Modalities and techniques used are similar to those used for patients with intervertebral disk disease. Rehabilitation techniques vary depending on the location of the lesion and the degree of damage to the spinal cord. Depending on the severity of the condition, the patient may have increased muscle tone and spasticity, or decreased muscle tone and flaccidity. In general, ES, passive range of motion of affected joints, aquatic therapy, and assisted ambulation are early modalities that may be used. Continued ES, assisted sling walking, deep water aquatic therapy, and gait and strength training are performed as the patient improves.

Nonambulatory patients require increased nursing care, including appropriate bladder care, assistance with adequate nutrition and fluid intake, and appropriate monitoring to ensure cleanliness and comfort. Patients should be repositioned every 4 to 6 hours to prevent decubital sores. A positioning protocol should be initiated immediately. If increased tone to the limb(s) is present, at-risk muscles are placed in elongated positions. Passive range of motion to joints and muscles at risk of developing contracture and adaptive shortening is performed regularly, typically three to six times daily. Passive range of motion or active assisted range of motion and stretching using the body weight and movement of the body in closed-chain activities can be effective. Slow rhythmic bouncing on a therapy ball assists in decreasing hypertonia and allows for more effective muscle stretch and more normal movement patterns.

If the patient is unable to sit or stand independently, assistance to these positions, with proper joint alignment, should be promptly initiated. Placing the patient over a small therapy ball with the feet still in contact with the floor assists in maintaining a standing position (see Figure 34-12). It is important to provide weight bearing on the affected limbs while maintaining a standing position. Weight shifting in functional planes of movement also may be initiated in this position. If working only with the pelvic limbs, the torso and front limbs may be placed on the ball while the pelvic limbs are worked in a weight-bearing position.

Firm, deep pressure through manual contacts activates the tactile receptors, neuromuscular proprioceptive pathways, and sensory awareness. Proper hand placement provides security and support to unstable body segments and may stimulate contraction of the muscles under the hands of the therapist (Figure 34-22). Applying approximation



Figure 34-22 Assisted standing exercise.

and compression joint mobilization techniques facilitate postural extensor and stabilizers and enhances body awareness. Approximation of the affected joints can be achieved by bouncing the patient while it is sitting on a therapy ball or weight bearing over a ball, and through manual compression of the joints. Providing intermittent pressure through the pelvis and legs (bouncing) stimulates approximation of affected joints in patients with pelvic limb dysfunction that are able to assume a standing position.

Deep water hydrotherapy is also used to stimulate muscle activity and motor learning (see Figure 34-19). Depending on the functional level of the patient, life preservers or manual assistance in the pool may be necessary to ensure safety (see Figure 34-6). This assistance also allows the therapist to more easily provide reciprocal movements to the affected limbs. Assisting the patient to stand in shallow water or weight bearing on the pelvic limbs with the forelimbs elevated are also useful treatments in the pool.

Assisted ambulation with manual reciprocal placement of the patient's feet may stimulate motor memories and initiate motor learning. Sling or tail walking aids ambulation. As the patient improves, the use of an underwater treadmill or land treadmill further benefits strengthening and gait training. Standard 3-week rehabilitation protocols may be lengthened in those patients experiencing a slower recovery.

Neoplasia

Spinal tumors are classified by their location as extradural, subarachnoid (intradural-extramedullary), and intramedullary. Extradural neoplasia is most common and includes

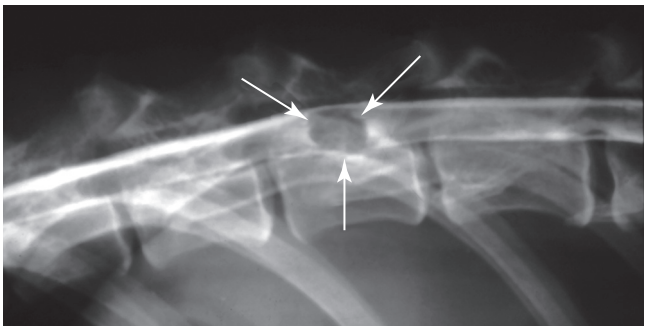


Figure 34-23 Myelography showing a spinal tumor (meningioma) at the level of L1. There is a well-defined filling defect (*arrows*) outlining the mass.

osteosarcoma, multiple myeloma, fibrosarcoma, chondrosarcoma, hemangiosarcoma, and lymphosarcoma. Extradural lymphosarcoma is the most common spinal tumor in cats and may be secondary to infection with the feline leukemia virus. Subarachnoid tumors include nerve sheath tumors, meningioma, lymphosarcoma, and neuroepithelioma. Intramedullary tumors include glioma, lymphosarcoma, and hemangiosarcoma. Metastatic tumors, including metastatic carcinomas and sarcomas, may occur in any location. Spinal neoplasia is more common in middle-aged or older animals. However, neuroepithelioma affects young, large breed dogs, and lymphoma may affect a cat at any age.

Clinical signs are typically insidious and progressive but can be acute. Focal spinal pain is the most common initial sign. Later ataxia and paresis may develop as the spinal cord becomes progressively affected. Systemic signs, such as weight loss, may occur with metastatic cancer. Nerve sheath tumors often present as chronic lameness of one limb that can be difficult to differentiate from orthopedic disease. Eventually, muscle atrophy and neurologic deficits develop. Intramedullary tumors may present with progressive neurologic signs without evidence of pain.

Diagnosis is based on clinical features, and diagnostic imaging. If neoplasia is suspected, chest radiographs are indicated to screen for metastasis. Spinal radiographs may show abnormalities in cases of vertebral tumors, although initial changes may be subtle. Myelography is helpful in identifying spinal cord compression (Figure 34-23). Computed tomography, CT-myelography, and MRI are more sensitive. Definitive diagnosis often requires surgical exploration and biopsy.

Medical management may provide temporary relief of pain and weakness to maintain a reasonable quality of life for a short period of time. Generally this consists of judicious activity, analgesics, and corticosteroids. Chemotherapy may be appropriate for specific tumor types. Definitive treatment usually consists of surgery. The goal is to obtain tissue for histopathologic diagnosis, relieve compression

on the spinal cord, decrease the tumor volume before radiation therapy, or completely remove the tumor, depending on the location, size, and type of tumor. Radiation therapy may be used as a primary treatment or as an adjunct to surgery, depending on tumor type. The prognosis depends on the tumor type.

Physical Rehabilitation for Neoplasia of the Spinal Column

Postoperative physical rehabilitation protocols are generally similar to those used in the rehabilitation of patients after intervertebral disk decompression. One essential difference, however, is that protocols must generally be stepped down rather than stepped up as the disease progresses and the condition and ability of the patient diminish.

Trauma

Major trauma can result in various head and spinal lesions, depending on the anatomic location. A thorough history usually documents the presence and mechanism of trauma, although the cause is sometimes unknown or surmised by concurrent findings if the accident is not witnessed. Automobile accidents, gunshot wounds, falls, and dog fight injuries are common. Spinal injuries may consist of vertebral luxation, vertebral fracture, vertebral fracture/luxation, or traumatic IVD herniation.

In any trauma patient, full assessment of all body systems is essential to diagnose and treat shock and any other injuries. A neurologic examination is performed to localize the injury and determine the severity of the deficits. It is vital that the examination be carefully performed, minimizing movement of the patient to avoid further damage to the spinal cord as a result of instability of the spinal column. Assessment of mentation, cranial nerves, voluntary movement, and spinal reflexes, as well as head and spine palpation, is performed, but moving the patient to test gait and postural reactions is avoided until unstable spinal injuries are ruled out with radiographs. The presence or absence of deep pain caudal to the injury is the most important prognostic sign; lack of deep pain sensation after spinal trauma carries a poor prognosis for neurologic recovery.

Following initial assessment and stabilization, appropriate analgesics are administered. Methylprednisolone (30 mg/kg IV, then 1 hour later 5.4 mg/kg per hour IV infusion for 24 to 48 hours) may improve the chance of recovery from severe spinal cord injury if started within 8 hours of injury. However, conclusions from human clinical trials are controversial and there are no published controlled studies in veterinary patients.[27] A lateral-view radiograph of the affected region of the spine is obtained when the patient is stable. If dorsoventral views are necessary, a horizontal beam view is safest. The purpose of radiography is to confirm the localization, classify any fracture/luxation as stable or unstable, and try to determine if there is any persistent spinal cord compression that may require surgery. The two components of the spine that maintain stability are (1) the dorsal compartment, consisting of the articular facets and lamina, and (2) the ventral compartment, consisting of the vertebral bodies and intervertebral disk. Damage to one of these components usually does not result in severe instability, whereas damage to both the dorsal and ventral components usually requires fixation to prevent further displacement. Myelography or CT is indicated if survey radiographs are normal or inconclusive. Routine chest and abdominal films also may be indicated to determine other body systems affected by the trauma.

The decision to treat the patient is based on the severity of the neurologic deficits, the type of vertebral injury, the severity of any concurrent injuries, and the owner's understanding of the risk, prognosis, and expense of the injury and treatment. Animals with mild neurologic deficits caused by cervical injuries or stable thoracolumbar injuries often recover with nonsurgical therapy. This consists of 4 to 6 weeks of complete cage rest, with very cautious movement of the animal to assist it with posturing to urinate and defecate. Weight bearing is generally not encouraged, unless the vertebral column is stable. Sometimes an external splint is used in conjunction with cage rest.[28]

Indications for surgery include (1) unstable fracture/luxation, usually evident on imaging by disruption of the dorsal and ventral components of the vertebrae; (2) persistent compression of the vertebral canal, evident as a substantially displaced fracture/luxation or extradural compression of the spinal cord on myelography, CT, or MRI; and (3) progressive deterioration in neurologic status despite conservative treatment, usually owing to an unstable fracture/luxation or progressive spinal cord compression from hemorrhage or disk extrusion.[28]

Surgery usually involves decompression of the spinal cord and fixation of unstable vertebrae. The choice of fixation technique depends on the size of the animal, the type and location of the injury, and the surgeon's preference and experience. In general, dorsal techniques (spinal staples, dorsal spinal plates, and modified spinal instrumentation) are indicated if the ventral components of the vertebrae are intact. Ventral techniques (vertebral body plates, pins/screws, and bone cement) are stronger and can be used with dorsal and ventral component injuries (Figure 34-24). A laminectomy to decompress the spinal cord from intervertebral disk material, ligament, or bone may be necessary before fixation. External splints of light casting material or moldable thermoplastic materials provide some stability and are most applicable to thoracic, cranial lumbar, and cervical regions. Splints may create sores and have the potential for creating complicated wounds that require additional care. Splints may also be used as an adjunct to internal fixation.[28]

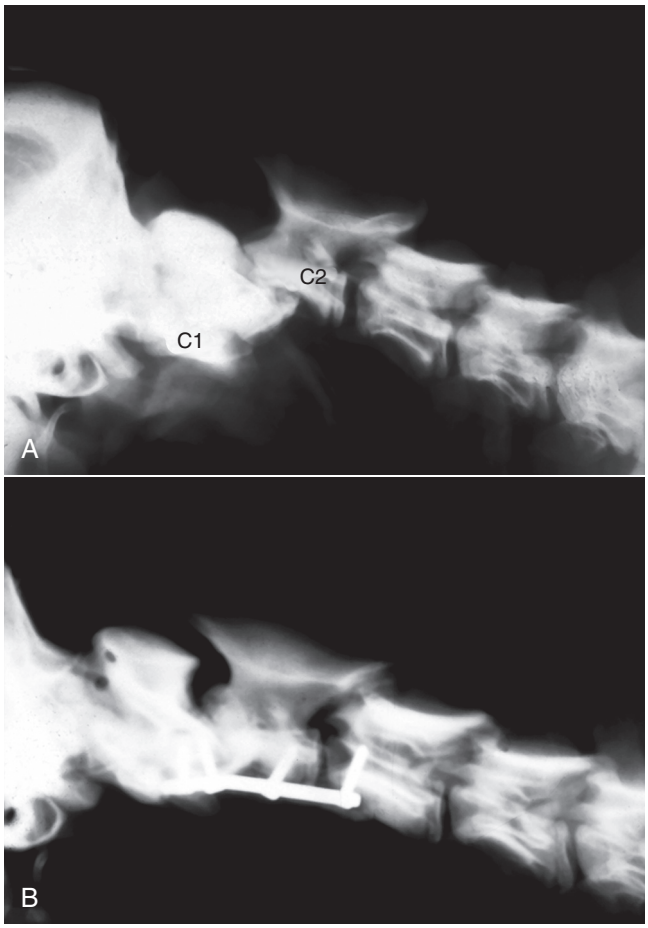


Figure 34-24 Surgery for a spinal fracture/luxation. **A,** There is a fracture of C2 with dorsal displacement of C2 relative to C1. **B,** Postoperative view. The injury has been repaired with a plate and screws affixed to the ventral aspect of C1, C2, and C3.

Physical Rehabilitation for Traumatic Injuries

Physical rehabilitation protocols and modalities are similar to those used for rehabilitation of lumbosacral decompression/stabilization and cervical spondylomyelopathy patients. Wound care is discussed in detail in chapter 36 for those patients that develop cast wounds. Extended care and therapy beyond the standard 3-week rehabilitation protocol may be required in some cases.

Peripheral Nerve Injury

Common causes of peripheral nerve injury include fractures (e.g., femoral fracture that damages the sciatic nerve), intramuscular injection (usually affecting the sciatic nerve), traumatic brachial plexus nerve root avulsion, and poor surgical technique. Vascular injuries can also occur, the most common of which is iliac thrombosis in cats causing a distal sciatic neuropathy, but thrombosis of the brachial artery can also cause thoracic limb monoparesis. Axons in peripheral nerves regenerate at rates as fast as 1 mm a day. Nerves must be in a Schwann cell environment for this regeneration to occur. Peripheral nerve injuries have three levels of severity. In neurapraxia, axonal conduction is lost without disruption of the axon. This injury usually results from compression, transient ischemia, or blunt trauma. Loss of conduction may be a result of myelin damage or insufficient energy to maintain axonal resting potential. In axonotmesis the axon integrity is lost, but the endoneurium and Schwann cell sheath it lies within are still intact, providing the opportunity for regeneration back to the correct target. Successful regeneration may occur, particularly if the axon is damaged close to its target. In neurotmesis the entire structure of the nerve is disrupted. The axon has the ability to regenerate but needs to find a Schwann cell sheath to do so, making it much more difficult.

Peripheral nerve lesions can cause unpleasant abnormal sensations (paresthesia) and hyperesthesia, both of which can result in self-mutilation. Another sequel to denervation of a muscle is severe muscle atrophy, which over time may lead to muscle contracture and, in growing animals, to skeletal deformities.

Localization of the lesion is based on the neurologic examination, which indicates the pattern of muscle denervation and sensory loss. It is useful to refer to references depicting the cutaneous sensory zones of peripheral nerves.[29,30] Severity of the lesion is determined by assessing the level of motor function and assessing for deep pain sensation. Electrophysiologic evaluation of the muscles and nerves using electromyography and nerve conduction velocity studies allows a more detailed description of the severity and course of the injury. Muscles that are completely denervated develop spontaneous electrical activity when at rest, although such changes do not appear for at least a week after denervation of a muscle. Nerve conduction studies should be interpreted with care. Immediately after an injury, conduction may be lost across the site of injury, whereas the distal portion of the disrupted nerve can continue conducting for a period of hours to days. As nerves regenerate and sprout to innervate denervated muscles, the size of motor units increases; therefore the size of motor unit potentials on the EMG increases.[31,32]

As a rule neurotmesis carries a poor prognosis unless immediate surgical intervention to reconnect the severed nerve occurs. Animals with axonotmesis or neurapraxia carry a better prognosis. Neurapraxia usually reverses within 2 weeks of injury, although damage to myelin slows recovery to 4 to 6 weeks.

The recovery from axonotmesis is governed by the proximity of the injury from the target muscle, the severity of muscle atrophy, and the development of contractures. If the damage has occurred far from the target muscle (e.g., at the brachial plexus), by the time the axon has regrown, severe muscle atrophy and contractures could limit recovery.

Brachial plexus injuries tend to involve the caudal two thirds of the plexus (radial, median, ulnar, and lateral thoracic nerves and the sympathetic innervation of the head) or the complete plexus, although cranial plexus injuries have been reported.[31] It is easy to be misled when evaluating animals with caudal plexus injuries, because there is preservation of musculocutaneous function and elbow flexion. This function is not useful for recovery of the ability to bear weight and should not be used to determine the prognosis. Instead, it is important to test deep pain perception, particularly in the lateral digit.[33] The absence of deep pain in this digit implies severe radial nerve injury. If it does not reappear within 2 weeks of injury, the prognosis for recovery of useful motor function in that limb is guarded.

In peripheral nerve inures, sensation must be considered. Dogs experiencing anesthesia, paresthesia and hyperesthesia may injure their limbs by not recognizing when the limb may be caught on an object, dragging the limb and causing damage, or by self-mutilation caused by the unpleasant sensations.

Rehabilitation for Peripheral Nerve Injury

The goals of a rehabilitation program for patients with lower motor neuron injury include restoring and maintaining joint range of motion, improving muscle strength, restoring neuromuscular function, and preventing self-mutilation and trauma to the affected limb. The lack of spinal reflexes and corresponding muscle tone in these patients poses a challenge to their rehabilitation, and emphasis is placed on restoration of muscle and joint function.

Because of the dysfunction of the spinal reflex arc, passive exercises are performed until a near-normal gait is established. Passive range of motion and stretching are used in the same manner as in patients with other neurologic problems. Patients with lower motor neuron deficits may benefit from stretching of affected and antagonist muscles. Loss of tone to antagonist muscle groups predisposes patients to joint contractures. Massage of a mildly contracted muscle group may also be beneficial in restoring its function and should be performed two to three times per day after warming the region.

In patients with a sciatic nerve deficit, elicitation of a flexor withdrawal reflex may not be possible. Nevertheless, progress should be monitored by serial evaluations of the spinal reflex arcs. In patients with weak or intact flexor withdrawal reflexes, stimulation of the flexor reflex helps to improve muscle tone and neuromuscular coordination. Patients with femoral nerve injury require much assistance to maintain normal posture. A balance ball (Swiss ball) may be used to support the trunk while slowly lowering the pelvic limbs to the ground. The patient's hind limbs are then gently raised (enough to lift the toes off the ground) and lowered, such that the animal is required to support its body weight as the hind limbs are lowered to the ground.

Patients with mild radial nerve deficits benefit from being challenged to bear weight on their forelimbs. Patients who lack elbow or carpal extension (e.g., brachial plexus avulsion) have great difficulty or are unable to perform this activity until some extensor muscle tone is present. The exercise is performed by placing the patient in a standing position while supporting the trunk and thoracic limbs. With the animal's front feet placed squarely on the ground, the amount of support is gradually reduced. When the patient starts to collapse in the thoracic limbs, support is provided to the animal and it is returned to a standing position. A balance ball or custom orthotics may similarly be used to support the patient. Orthotics are described in more detail in Chapter 17. The exercise is repeated 5 times, two to three times per day.

Active exercises are designed to improve muscle strength, neuromuscular balance, and coordination in patients who have at least some voluntary movement of their limbs. Patients with peripheral nerve disease affecting more than one limb may not be able to do some of these activities. Patients with sciatic nerve deficits can often perform sit-to-stand exercises because these exercises require active stifle extension but only passive stifle and tarsal flexion. Balance and coordination exercises benefit patients with peripheral nerve injuries and are performed as described previously.

The application of neuromuscular electrical stimulation in patients with peripheral nerve disease may delay the onset of neurogenic muscle atrophy and recondition the affected muscles.[34] When a muscle is completely denervated, electrical muscle stimulation is the modality of choice. Affected muscle groups should be stimulated once a day for 15 minutes each.

Discospondylitis

Discospondylitis is an infection of the intervertebral disk and adjacent vertebral bodies. It is usually caused by hematogenous spread of organisms from sites elsewhere in the body, such as the urinary tract, skin, or mouth. Penetrating wounds, surgery, or plant material migration can cause direct infection of the disk space or vertebra. In certain geographic regions migrating plant material such as grass awns is a relatively common cause of vertebral osteomyelitis and discospondylitis of the L2-L4 vertebrae. *Staphylococcus intermedius* is the most common etiologic agent identified in dogs with discospondylitis. Other frequently documented pathogens include *Streptococcus* spp., *Brucella canis*, and *Escherichia coli*, but virtually any bacterial agent can be causative. Some infections are caused by fungal organisms, including *Aspergillus*, *Paecilomyces*, and *Coccidioides immitis*. Vertebral infections caused by

grass awn migration are often associated with mixed infections, with *Actinomyces* being a common isolate.[35]

Discospondylitis most commonly affects large, middle-aged dogs. Small dogs and chondrodystrophic breeds are uncommonly affected, and the disease is rare in cats. The most common initial sign is spinal pain, which varies in intensity from mild to extreme. The duration of signs noticed by the client usually ranges from several days to several weeks. Neurologic deficits ranging from ataxia to paralysis are seen in some patients, although usually not as the initial abnormality. Signs suggestive of systemic illness, such as fever, lethargy, depression, anorexia, and weight loss occur in about 30% of affected dogs. Concurrent urinary tract infection, including prostatitis in male dogs, is common.[35]

Diagnosis is based on clinical features and diagnostic imaging. Radiographs of the involved region are usually diagnostic, although they may be normal early in the course of disease. The earliest radiographic sign is subtle irregularity of the vertebral end plates. As the infection progresses the erosion of the end plate becomes more pronounced, and there are osteolytic and osteoproductive changes of the intervertebral disk space, vertebral end plates, and in severe cases, the entire vertebral body (Figure 34-25). Computed tomography and MRI are more sensitive and are helpful when radiographs are normal or inconclusive. Myelography or MRI is indicated only in patients with substantial neurologic deficits when decompressive surgery is considered. Complete blood count, urine culture and sensitivity, blood cultures, and serology for brucellosis are performed in patients with radiographic evidence of vertebral infection. The differential diagnosis includes chronic intervertebral disk disease, inflammatory disease, and neoplasia.

Initial treatment consists of antibiotics, cage rest, and analgesics. Selection of antibiotics is based on culture and sensitivity of blood and urine and serology for *B. canis*. Pending these results or if these tests are negative, treatment is started with a bactericidal antibiotic effective against beta-lactamase–producing *Staphylococcus*. First-generation cephalosporins are effective against the majority of isolates from affected dogs and are a good choice for initial therapy. Clindamycin and amoxicillin with clavulanic acid are also usually effective. Patients with fever, neurologic deficits, or rapidly progressing signs are treated with intravenous antibiotics for 3 to 5 days, followed by a course of oral antibiotics. Initial oral therapy is appropriate in patients with mild, slowly progressing spinal pain.[35]

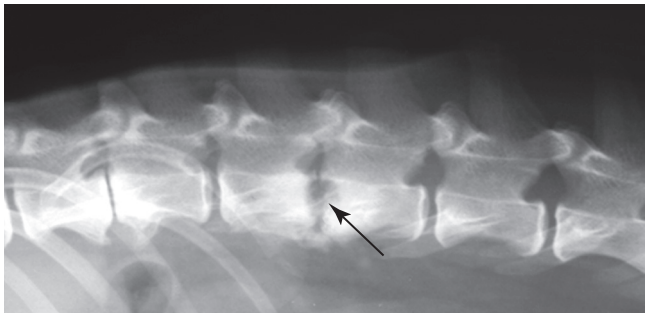


Figure 34-25 Discospondylitis. The radiograph shows lysis of the vertebral end plates (*arrow*) adjacent to the L2-L3 disk space.

Clinical signs such as spinal pain and fever usually resolve within 5 days of starting effective therapy, although neurologic deficits usually resolve more slowly. Patients should be treated for at least 8 weeks in an attempt to completely clear the infection and prevent relapse. Patients that fail to improve within 5 days of starting treatment are reassessed. Options include treating with a different antibiotic or culture of the affected disk space by needle aspiration, guided by fluoroscopy or CT.[36] Most animals recover with appropriate medical treatment. Surgery is rarely indicated to obtain biopsy when there is no response to initial therapy, or to decompress the spinal cord in patients with substantial neurologic deficits, evidence of spinal cord compression on imaging, and no response to medical therapy.

Physical Rehabilitation for Discospondylitis

Physical rehabilitation is indicated in more severely affected patients after a positive response to medical management is achieved. Protocols used are similar to those used in dogs with IVD, specifically aquatic therapy, and strength and gait training. Particular care should be exercised because affected patients generally have significant weakness of the affected bones of the vertebral column.

Atlantoaxial Luxation

The anatomy of the atlas (C1) and the axis (C2) is distinctly different from the rest of the vertebral column. There is no intervertebral disk and very little flexion at this site. The anatomical relationship between C1 and C2 is primarily maintained by ligaments. Atlantoaxial luxation occurs when instability of the joint allows C2 to luxate dorsally, relative to C1, and compress the spinal cord. Associated lesions can include congenital absence of the dens, fracture of the dens, and absence or rupture of atlantoaxial ligaments.

Atlantoaxial luxation is most common in toy-breed dogs with malformation of the dens or ligaments. It can occur in any animal as a consequence of trauma. In dogs with congenital instability of the atlantoaxial joint, clinical signs usually occur within the first few years of life and are often precipitated by relatively minor trauma. Yorkshire terriers, Chihuahuas, miniature and toy poodles, Pomeranians, and Pekingese dogs are most commonly affected. Clinical signs include neck pain and gait dysfunction, ranging from ataxia to tetraplegia, with normal or exaggerated reflexes. Severe cases may be fatal due to

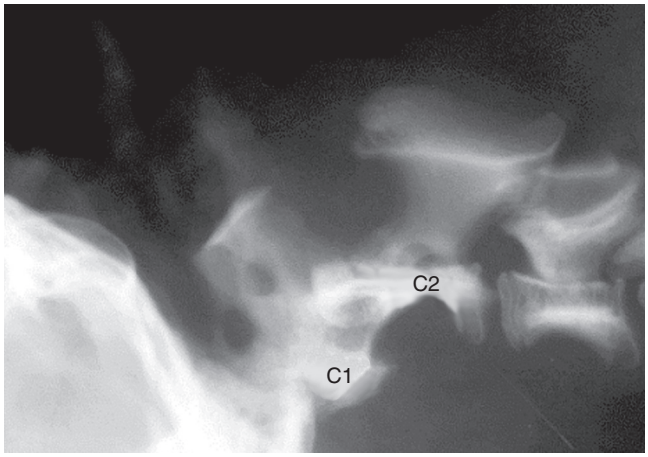


Figure 34-26 Atlantoaxial luxation. The radiograph shows dorsal displacement of C2 relative to C1.

respiratory paralysis. It is critical to avoid flexion of the neck because this exacerbates spinal cord compression. The differential diagnosis includes vertebral fracture or luxation, intervertebral disk disease, discospondylitis, neoplasia, and meningomyelitis.

The diagnosis is based on history, physical and neurologic examination, and radiographs. Typical radiographic findings include dorsal displacement of C2 relative to C1 and an increased space between the arch of C1 and spine of C2 on the lateral view (Figure 34-26). The size and shape of the dens are best evaluated on a ventrodorsal view. The dens may be normal, hypoplastic, aplastic, or dorsally angulated. If survey radiographs are equivocal, the atlantoaxial joint may be slightly flexed during fluoroscopy to assess stability. CT or MRI is also useful if the diagnosis is uncertain, or to identify coexisting lesions. Cerebrospinal fluid analysis is helpful in identifying or ruling out inflammatory disease. Lumbar puncture for fluid collection avoids flexion of the cervical spine that is required for collection of fluid from the cerebellomedullary cistern.

Nonsurgical treatment is indicated in patients with mild signs. This consists of cage rest and application of a neck brace that prevents neck flexion for 6 weeks. Analgesics are administered as needed. Conservative treatment may be successful in patients with traumatic luxations, but dogs with congenital instability can suffer relapse because of the inherent instability. Surgery to reduce and stabilize the atlantoaxial joint is the definitive treatment. Doral techniques achieve fixation by securing the spinous process of the C2 to the dorsal arch of C1 using the nuchal ligament, orthopedic wire, nonmetallic suture, or an implantable device (Kishigami atlantoaxial tension band). Cross-pinning of the atlas and axis with polymethylmethacrylate has also been described. Ventral techniques can be performed using transarticular screws or pins, with or without polymethylmethacrylate, or a plate. Implants may also be placed into the pedicle of the atlas and the body of the axis. The articular cartilage may be removed and bone grafts placed to encourage fusion. A theoretical advantage of ventral techniques is bony fusion although incomplete bony fusion is apparent on follow-up radiographs in many patients with a good outcome.[37] Potential complications include implant failure, worsening of neurologic deficits, and upper respiratory problems associated with the ventral surgical approach. Successful outcome is reported in 62% to 91% of patients. Older age (greater than 24 months) at onset of signs, longer duration of clinical signs (greater than 10 months), and more severe neurologic deficits increase the risk of poor outcome.[37]

Physical Rehabilitation for Atlantoaxial Subluxation

Postoperatively, a cervical splint fashioned from light casting material may be applied. Periodic removal allows assessment of underlying tissues. External coaptation is continued for 4 weeks along with strict confinement. Physical rehabilitation begins after cast removal. Whirlpool aquatic therapy combined with ES and therapeutic US is appropriate in the early phases of rehabilitation. Assisted aquatic therapy is beneficial until ambulation begins. Strengthening and gait training exercises and underwater treadmill aquatic therapy may be incrementally added.

Degenerative Myelopathy

This is a slowly progressive degenerative disease of the central nervous system that most severely affects the thoracic spinal cord segments. It is inherited as an autosomal recessive trait. The cause is a mutation in the superoxide dismutase 1 (SOD1) gene, similar to the familial form of amyotrophic lateral sclerosis in human patients (Lou Gehrig's disease).[38]

Degenerative myelopathy affects older dogs, usually more than 8 years of age. German shepherd dogs, Rhodesian ridgebacks, Pembroke and Cardigan Welsh corgis, boxers and Chesapeake Bay retrievers, are most commonly affected, although cases have been documented in a number of other large-breed dogs and cross breeds.[39]

There is an insidious onset of progressive ataxia and paresis in the pelvic limbs. This is manifested as swaying, staggering, and scuffing of the rear limbs, often worse on one side. Spinal reflexes in the pelvic limbs are normal to exaggerated. An essential feature is that this disease does not cause spinal pain. Most patients become nonambulatory within 6 to 9 months, at which point many clients elect euthanasia. Otherwise the signs eventually progress to loss of reflexes in the pelvic limbs, paresis of the thoracic limbs, urinary and fecal incontinence, and brainstem involvement, such as dysphagia.[39]

Diagnosis is based on clinical features, imaging, and spinal fluid analysis to rule out other causes, and DNA

testing to detect the SOD1 mutation. Affected dogs have two copies of the abnormal gene, one inherited from the mother and one inherited from the father. Dogs that test positive for both copies of the abnormal gene are at risk of developing the disease and will pass the mutation to offspring. Dogs with one copy of the abnormal gene do not develop the disease but can pass the trait on to their offspring. Dogs with two copies of the normal gene do not develop the disease and cannot pass on the trait.[39]

Other spinal cord diseases can cause similar or identical signs, including intervertebral disk protrusion, spinal cord tumor, and inflammatory diseases. It is important to exclude these diseases on the basis of imaging tests such as MRI and analysis of spinal fluid.

There is no medication that has been shown to be effective for degenerative myelopathy in randomized controlled studies. Steroids and other antiinflammatory drugs have been used but are generally not helpful. A study evaluating combination therapy with aminocaproic acid, *N*-acetylcysteine with vitamins B, C, and E failed to detect a benefit.[40] Physical rehabilitation may improve the quality of life of affected dogs and improve survival time. A nonrandomized study found patients with extensive physical rehabilitation had significantly longer survival times (mean 255 days) than dogs that received moderate (mean 130 days) or no physical rehabilitation (mean 55 days).[41] This is likely due to dogs maintaining their mobility longer with physical rehabilitation as loss of mobility is a main factor in euthanasia. Common rehabilitation includes range of motion and therapeutic exercise, aquatic therapy, and maintaining function as long as possible.

Miscellaneous Conditions

Miscellaneous diseases creating neurologic dysfunction that are treatable and have a reasonable prognosis may benefit from physical rehabilitation. These include immune-mediated polyradiculoneuritis (Coon Hound paralysis), congenital vertebral anomalies and arachnoid cysts. Because of the uncommon clinical presentation of these conditions and the lack of experience in rehabilitating these conditions, the use and success of specific protocols are unknown. Adherence to therapeutic principles and selecting protocols used for similar diseases should provide a sound basis for rehabilitation of these patients.

Summary

The rehabilitation of dogs with neurologic disease involves a combination of nursing care, active and passive exercise, functional activities, and therapeutic modalities. The foundation for maximizing the patient's functional recovery is cooperation and participation of the patient, the owner, and the therapist.

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35 Physical Rehabilitation for Geriatric and Arthritic Patients

Denis J. Marcellin-Little, David Levine, and Darryl L. Millis

Geriatric and arthritic patients have specific medical and rehabilitation needs linked to the increased prevalence of chronic conditions and their progressive loss of mobility. In fact, geriatric patients can be a significant component of a rehabilitation practice. Aging triggers changes in a wide range of tissues. Also, the incidence and severity of chronic diseases increase over time. The life span of canine companions is increasing in part because of medical and surgical advances in veterinary medicine. As a consequence geriatric and arthritic patients are often candidates for rehabilitation, requiring a thorough assessment and potentially complex treatments. Geriatric and arthritic patients should also be reassessed on a regular basis to detect changes in their conditions and to design and deliver the most accurate and beneficial plan of care. This chapter discusses the effects of aging, medical considerations and common neurologic and musculoskeletal conditions in the aging dog, quality of life issues, pain management in geriatric and arthritic patients, nutritional considerations, and general management of the arthritic patient.

Aging in Dogs

The term *geriatric* relates to elderly people. The World Health Organization defines middle-age as being 45 to 59 years, elderly as being 60 to 74 years, and aged as being 75 years and older.[1] No such groupings have been proposed in dogs, although Table 35-1 describes the ages at which dogs are considered "geriatric" and are likely to develop diseases associated with aging.[2] Aging itself is not a disease. Multiple factors, including genetics, environment, and nutrition, play a role in the aging process. For example smaller dog breeds live longer than larger breeds, and mixed-breed dogs have a longer life expectancy than purebred dogs. Obese animals have a shorter life span than non-obese animals.[3] Dogs that are neutered live longer than those that are not neutered. Pets that live inside live longer than those that are housed outside.[4]

There are multiple changes that occur as a result of the aging process in dogs, as outlined in Box 35-1.

Most of these conditions are chronic (endocrinopathies, hepatic disease, renal disease, cardiac insufficiency) and require lifelong treatment and sustained owner compliance. It is important for the therapist to understand the effects these diseases have on function and activities of daily living. For example, hypothyroidism may cause lethargy and easily could be mistaken for arthritis and inactivity. It is important for the owner to make regular appointments with their veterinarian for patient monitoring and modification of the treatment plan, as necessary.[4] The changes associated with aging are progressive and are hastened by stress, the environment, genetic factors, malnutrition, and lack of activity.

Medical Considerations

The effects of aging have been documented in human beings,[5] and similar effects are seen in dogs. Quite often, owners of geriatric dogs attribute specific changes in behavior and lifestyle, such as a decreased appetite, changes in sleeping patterns, or decreased activity, to their pet becoming arthritic or being old. Although a component of these changes could be the aging process, the presence of concurrent neurologic, musculoskeletal, or metabolic diseases should also be considered. In geriatric dogs, the diagnosis and treatment of systemic diseases are necessary for the success of a rehabilitation program aimed at managing a disorder such as osteoarthritis (OA). The increased likelihood of multiple conditions being present in a geriatric dog warrants a thorough medical evaluation before a rehabilitation program is designed. A complete medical history, including previous medical diagnoses and treatments, may help determine the origin of the patient's primary problem and identify any previous or concurrent diseases that may affect the success of rehabilitation. In conjunction with the history, performing a thorough physical examination and obtaining hematologic information may confirm the presence of concurrent diseases.

Multiple disease processes in older animals, such as those listed in Table 35-2, can cause nonspecific signs that mimic or exacerbate the signs of OA or aging. These signs include weakness, lethargy, and exercise intolerance. Treatment of concomitant diseases before the implementation of a rehabilitation program allows the patient to be in the best condition to appropriately respond to rehabilitation

Table 35-1 Typical Ages at Which Various-Sized Dogs May Be Considered Geriatric

	Weight	Age (Mean ± Standard Deviation)
Small dogs	0-20 lb	11.48 ± 1.86
Medium dogs	21-50 lb	10.19 ± 1.56
Large dogs	51-90 lb	8.85 ± 1.38
Giant dogs	>90 lb	7.46 ± 1.27

Box 35-1 Effects of Aging in Dogs

Metabolic Effects

Decreased metabolic rate
Decreased immune competence
Decreased phagocytosis and chemotaxis (decreased ability to ward off infections)
Autoantibodies and autoimmune diseases may develop

Physical Effects

Percentage of body weight represented by fat increases
Skin becomes thickened, hyperpigmented, and less elastic
Footpads hyperkeratinize and claws become brittle
Muscle, bone, and cartilage mass are lost, with possible development of arthritis
Lungs lose elasticity, fibrosis occurs, and pulmonary secretions become more viscous
Pulmonary vital capacity is diminished
Cough reflex and expiratory capacity decrease
Urinary incontinence frequently develops
Cardiac output may decrease
Number of cells in the nervous system decreases

and allows the clinician to provide the most accurate prognosis. If the concurrent disease requires long-term treatment, frequent adjustments of the medical therapy and the rehabilitation program may be required to reach the goals of each treatment. For example some of the most common systemic diseases in older animals are endocrine disorders and neoplasia.[6] Those conditions that have associated neurologic and musculoskeletal involvement, such as hyperadrenocorticism, hypothyroidism, hyperparathyroidism, osteosarcoma, and insulinoma interfere with the implementation of a successful rehabilitation program and accurate assessment of the patient's progression during the program. Understanding the pathophysiologic mechanisms is important not only for the successful treatment of the concurrent disease but also for the development of a rehabilitation plan tailored to the needs and weaknesses of the individual patient. Communication between the veterinarian, therapist, and owner is important and facilitates the

Table 35-2 Common Medical Conditions Present in Geriatric Dogs, Organized by Organ Systems

Organ System	Condition
Cardiovascular	Cardiomyopathy
	Endocardiosis
	Arrhythmias
	Heartworm disease
	Pericardial disease
	Systemic hypertension
	Endocarditis
Upper respiratory tract	Laryngeal paralysis
	Brachycephalic respiratory syndrome
	Laryngeal and pharyngeal neoplasia
	Bronchomalacia
	Collapsing trachea
Lower respiratory tract	Pneumonia
	Neoplasia (primary or metastatic)
	Pleural effusion
	Pulmonary edema
	Bronchitis
	Pneumothorax
Gastrointestinal	Malabsorptive disease (inflammatory bowel disease)
	Protein-losing enteropathy
	Neoplasia (adenocarcinoma, lymphoma)
Hepatobiliary	Hepatic parenchymal disease (chronic hepatitis, cirrhosis, neoplasia, toxic hepatitis)
	Biliary tract abnormalities (pancreatitis, bile duct neoplasia, cholelithiasis, cholecystitis)
Endocrine	Hyperparathyroidism
	Hypothyroidism
	Hyperadrenocorticism
	Hypoadrenocorticism
	Thyroid neoplasia
	Insulinoma
	Diabetes mellitus
	Pheochromocytoma
	Thymoma
	Hypoparathyroidism
Urinary tract	Renal failure
	Urinary tract infection
	Urinary incontinence
Hematologic and immunologic	Anemia (regenerative or nonregenerative)
	Neoplasia (lymphoma, hemangiosarcoma, leukemia)
	Hemostatic disorders
	Immune-mediated polyarthritis
	Systemic lupus erythematosus
Neurologic	Intervertebral disk disease
	Cervical spondylomyelopthy
	Lumbosacral instability
	Degenerative Myelopathy
	Neoplasia
Musculoskeletal	Osteoarthritis
	Neoplasia

assessment of the response to the rehabilitation program and the management of concurrent diseases.

Common Musculoskeletal and Neurologic Considerations

The list of common geriatric diseases is outlined in Box 35-2. The most important systems related to rehabilitation care are the musculoskeletal and nervous systems. Muscle mass and bone mass decrease with aging. Muscle function is diminished as a result of muscle fiber atrophy, loss of elasticity, and reduced oxygen transport to muscles. Cartilage tends to deteriorate as a result of altered biomechanical integrity and loss of tensile strength. The most common problem of the musculoskeletal system in geriatric dogs is OA. Obesity compounds the effects of arthritis (Figure 35-1).[4] Decreased intestinal absorption of calcium and decreased activity level may result in decreased bone mineral content. Osteoporosis, or reduction in the quantity of bone, is rarely clinically significant in aging dogs. The primary importance of senile osteoporosis is its influence on fracture healing. Callus formation is slower in older animals, and fractures require additional healing time. This increases the likelihood of delayed or malunion fractures, muscle atrophy, and disuse osteoporosis. Neoplasia involving the musculoskeletal system, although less common in some breeds of dogs, also must be considered. Primary bone tumors are the most common and include osteosarcoma, chondrosarcoma, fibrosarcoma, and hemangiosarcoma (Figure 35-2).[4]

Several conditions affecting the nervous system also affect aging dogs. In addition to intervertebral disk disease, degenerative conditions such as degenerative myelopathy and spinal neoplasia may occur in older dogs (Figure 35-3). Cognitive dysfunction is a common issue in older dogs. These dogs may exhibit signs such as loss of house training, disturbances in sleep and wake cycles, inattention to food or their environment, and inability to recognize familiar people and places. Short-term memory is affected by changes in interneurons that cause a prolonged effect to a stimulus and increased response time to the external environment.[4] Cognitive dysfunction also may affect the patient's ability to participate in some activities of a rehabilitation program.

Maintaining Quality of Life

A geriatric screening program should be implemented as part of a general wellness program for animals 8 years and older. This allows the veterinarian to target geriatric-related health problems, detect early geriatric disease to implement effective medical care, and offer preventive health care measures. Client education plays a large role in this process. Owners should be instructed on the normal process of aging, nutrition, appropriate exercise, and bereavement counseling, when indicated. A questionnaire is helpful to provide a baseline for function and may be used to reassess the animal's quality of life over time (Figure 35-4).

The normal process of aging includes changes in muscle physiology, hair coat and skin, cognitive function, hearing, and vision. These changes should be assessed, recorded, and addressed as part of the overall treatment program. The initial amount of exercise in the aging dog should be easily tolerated, with increasing amounts added as the rehabilitation program progresses. Low-impact exercise, such as frequent short leash walks and swimming should occur regularly, possibly on a daily basis. Owners should be aware of changes in water or food consumption, rapid weight changes, abnormal urination or defecation, changes in activity level, vomiting, or diarrhea (Figure 35-5). When appropriate, clinicians should advise owners during the final weeks of the animal's life and after their death.[1]

Box 35-2 Common Geriatric Conditions in Dogs

Obesity
Cardiovascular disease
Degenerative joint disease
Cataract(s)
Cancer
Dental disease
Hypothyroidism
Urolithiasis
Hyperadrenocorticism
Diabetes mellitus
Anemia
Urinary incontinence
Hepatopathies
Chronic renal disease



Figure 35-1 The most common problem of the musculoskeletal system in aged dogs is osteoarthritis. Dogs may have difficulty arising, and obesity compounds the effects of arthritis.

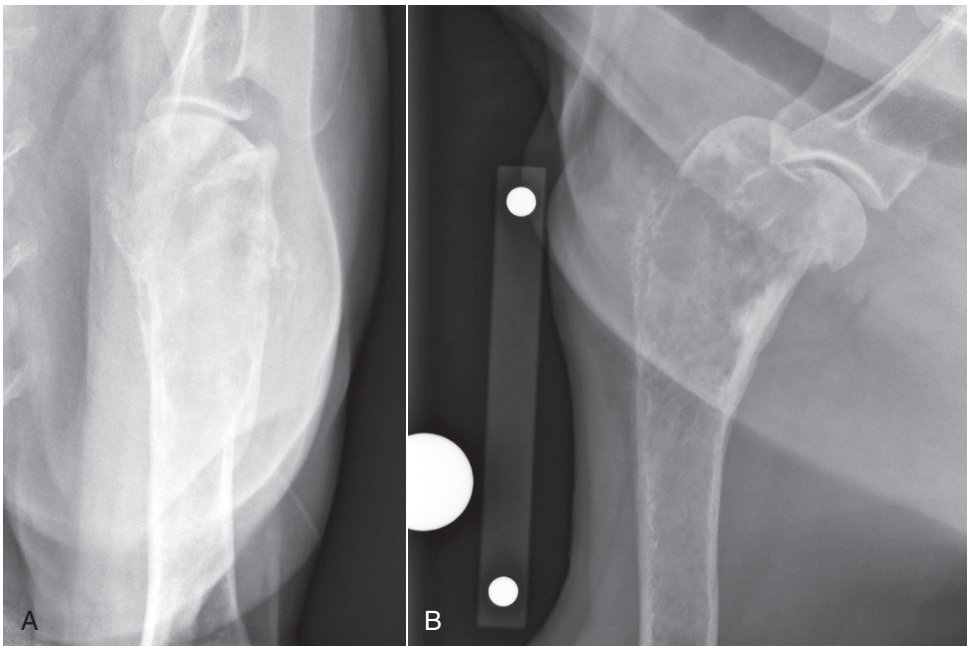


Figure 35-2 This 8-year-old spayed female rottweiler has an osteosarcoma in the proximal portion of the humerus visible on caudocranial **(A)** and mediolateral **(B)** radiographic projections. Classic clinical signs of osteosarcoma include lameness, lethargy, and decreased appetite.

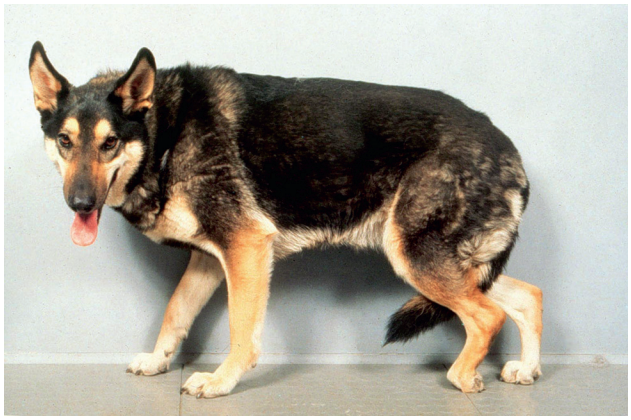


Figure 35-3 Degenerative myelopathy may affect geriatric dogs. The diagnosis is often confounded by the presence of osteoarthritis of multiple joints.

Pain Management in the Geriatric Dog

Because aging dogs may have a fragile physiologic balance, pain management of geriatric dogs should rely as much as possible on therapeutic options with minimal physiologic impact. For example, geriatric people are more sensitive to the gastrointestinal side effects of antiinflammatory medications than younger people.[7] The same most likely applies to dogs.[8] Nonsteroidal antiinflammatory drugs should therefore be used with caution in geriatric dogs. The pain management options with minimal physiologic risks include but are not limited to low-impact exercises, massage, and therapeutic heat and cold. Analgesic medications that may be helpful include amantadine, gabapentin, and tramadol. In patients with severe pain, such as may be present with neoplasia, opioid drugs may be prescribed, or transcutaneous fentanyl patches may be applied for sustained delivery of medication. Although these patches may be helpful in the short term, they are rarely used over extended periods of time because of the cost and other factors involved with prolonged treatment.

Nutritional Considerations

The weight management of geriatric patients is particularly important because obesity is one of the most common conditions in geriatric patients. Because of decreased muscle mass and the possible presence of OA in geriatric dogs, excess weight may predispose aging dogs to critical and potentially irreversible locomotion difficulties.[3] In one lifelong study involving 48 Labrador retrievers, restricted food intake increased median life span and delayed the onset of signs of chronic disease in dogs. In that study littermate dogs were paired, with one dog in each pair fed 25% less food than its littermate, which was fed the same diet ad libitum, from 8 weeks of age until death. Body composition was measured annually until 12 years of age. Compared with control dogs, food-restricted dogs weighed less and had lower body fat content and lower serum triglycerides, triiodothyronine, insulin, and glucose concentrations. Median life span was 18 months longer for dogs in which food was restricted. The onset of clinical signs of chronic disease, including OA, was delayed and generally less severe for food-restricted dogs.

FUNCTIONAL QUESTIONNAIRE

Please complete the questions on this form pertaining to your pet's functional abilities to help us monitor its progress.

1 = not able to perform this activity (needs assistance 100% of the time)
2 = moderate assistance to perform activity (needs assistance >50% of the time)
3 = minimal assistance to perform this activity (needs assistance <50% of the time)
4 = independent with activity (no assistance needed)
5 = N/A

Client:_____Patient:_____Date: _____
Medications:_____
How often is (are) medication(s) given? _____

1. Able to position itself to urinate?	1	2	3	4	5
2. Able to position itself to defecate?	1	2	3	4	5
3. Able to transfer from lying to sitting and vice versa?	1	2	3	4	5
4. Able to transfer from sitting to standing and vice versa?	1	2	3	4	5
5. Able to transfer from lying to standing and vice versa?	1	2	3	4	5
6. Able to roll over?	1	2	3	4	5
7. Able to scratch behind its ears?	1	2	3	4	5
8. Able to ascend stairs?	1	2	3	4	5
9. Able to descend stairs?	1	2	3	4	5
10. Able to walk up an incline/hill?	1	2	3	4	5
11. Able to get in and out of your car?	1	2	3	4	5
12. Able to get on/off a couch or bed?	1	2	3	4	5
13. Able to run?	1	2	3	4	5
14. Able to jump?	1	2	3	4	5

Figure 35-4 Functional questionnaire to assess and monitor the progress of a geriatric patient.

15. Experienced an increase or decrease in weight?	Increase	Decrease	Same
16. Experienced an increase or decrease in endurance?	Increase	Decrease	Same
17. Have you noticed a change in your pet's temperament/attitude? Y N Please elaborate:	Increase	Decrease	Same
18. What does your pet like to do for fun?			
Is he/she able to do that activity? Please elaborate:			
19. Has your pet been able to resume normal activities? Y N Please elaborate:			
20. Able to go on a walk? Y N How long? _____ minutes Could your pet walk longer? Y N Does anything prevent him/her from taking longer walks? Y N If so, what?			
21. Do you notice any problems (limping, stiffness) or are the problems worse after taking a walk? Y N If yes, how so?			
22. Does your pet tire quickly, have to make rest stops, or lag behind during walks? Y N Please elaborate:			

Figure 35-4, cont'd

Continued

23. Does your pet seem to be in pain? Y N

What makes you think this?

24. Is there anything your pet CAN do now that it was NOT able to do before? Y N
Please explain:

LAMENESS SCALE		
0 = Normal stance	0 = No lameness	0 = No lameness
1 = Slightly abnormal stance (partial weight bearing)	1 = Lameness barely perceptible	1 = Lameness barely perceptible
2 = Moderately abnormal stance (toe-touch weight bearing)	2 = Lameness obvious, but not severe	2 = Lameness obvious, but not severe
3 = Severely abnormal stance (holds limb off the floor)	3 = Severe lameness	3 = Severe lameness
4 = Unable to stand	4 = Partial or complete non–weight-bearing lameness	4 = Partial or complete non–weight-bearing lameness

25. Does your dog walk or trot with a limp? (Please select degree of lameness according to the chart below)

Degree of lameness (standing) Degree of lameness (walk) Degree of lameness (trot)

Score: _____ _____ _____

26. Are there any other problems that you have noticed that have not been covered in this form?

27. Additional comments:

Figure 35-4, cont'd



Figure 35-5 Owners should be aware of changes in water or food consumption, rapid weight loss, abnormal urination or defecation, changes in activity level, and vomiting or diarrhea, especially after a major illness or surgery. This dog developed sepsis after an amputation for osteosarcoma.



Figure 35-6 As osteoarthritis progresses, a vicious cycle of pain, reduced activity level, joint stiffness, and loss of strength occurs. The resulting weakness and pain may mimic a neurologic condition.

In addition to restricting calories, adequate protein, minerals, and vitamins also must be present in the diet of the aging patient. Most high-quality commercial diets have adequate nutritional composition to support most phases of life, and some concentrate specifically on aging dogs. Adequate protein is necessary to help maintain muscle mass, and it should be of adequate composition and quality to support muscle maintenance. Concurrent medical conditions, such as heart disease, renal disease, and liver disease, also should receive consideration because dietary management of these conditions is vital to successful management.

Management of the Geriatric Patient with Osteoarthritis

Joint disease is a common problem, affecting up to 20% of adult dogs. Osteoarthritis, often referred to as degenerative joint disease (DJD), is a progressive degenerative condition of joints. Many inflammatory mediators are present in arthritic joints, including interleukins, prostaglandins, and metalloproteinases. There is a progressive cascade of mechanical and biochemical events in patients with OA, which leads to cartilage destruction, subchondral bone sclerosis, synovial membrane inflammation, and periarticular osteophytes. Patients with OA have decreased quality of life, limited activity, reduced performance, muscle atrophy, pain and discomfort, and joint stiffness with decreased range of motion (ROM).[9-11] A vicious cycle of pain, reduced activity level, joint stiffness, and loss of strength occurs as OA progresses (Figure 35-6).

Veterinarians are approached frequently to treat arthritic patients. Traditional management of dogs with OA has included antiinflammatory and analgesic drugs, changes in lifestyle, and surgical management. More recent advances in the management of OA include weight loss, therapeutic exercise, and physical modalities to reduce the severity of clinical signs and reliance on medications to control pain and discomfort.[12,13] Management of the arthritic patient involves a number of modalities and must be tailored to each patient and owner. Nutritional management (including weight control), physical rehabilitation, and medications are the main components for OA management. Cooperation among the veterinarian, therapist, veterinary technician, and owner is vital to carry out an appropriate management program. Regular monitoring of the progress of therapy is essential to help with decision making for further treatment and maintaining enthusiasm for the program. Some of the benefits of a complete program include increasing muscle strength and endurance, increasing joint ROM, decreasing pain, and improving performance, speed, quality of movement, and function. This is particularly important for patients that have become exercise intolerant or have limited mobility.

Much of the pain associated with OA has been attributed to synovitis.[14] The goal of treatment is to provide adequate analgesia to reduce the severity of clinical signs, which allows the patient to be active and undergo therapeutic exercise and weight loss. These treatments are important to accomplish increased muscle strength and joint function, maintain an acceptable quality of life, control pain and discomfort, slow the progression of disease, and promote repair of damaged tissue when possible.

The surgical management of OA focuses on correcting joint disease to prevent further joint degeneration. Early intervention to remove an osteochondritis dissecans lesion, perform a juvenile pubic symphysiodesis or triple pelvic osteotomy, stabilize a cranial cruciate ligament rupture,



Figure 35-7 Early intervention and surgical stabilization of the stifle joint of dogs with a ruptured cranial cruciate ligament may reduce the progression of osteoarthritis.

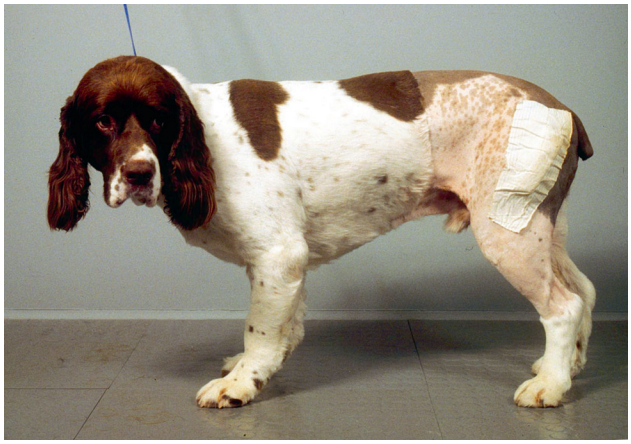


Figure 35-8 Although total hip replacement is a salvage procedure for end-stage osteoarthritis of the hip joint, dogs can have a high quality of life following surgery and rehabilitation.

repair an articular fracture, to reduce hip subluxation associated with hip dysplasia, and correct angular limb deformities to avoid abnormal stresses on joints are all surgical procedures that help slow the degenerative process of affected joints (Figure 35-7). Salvage treatments for severe OA include total joint replacement and arthrodesis of joints, such as the carpus and tarsus (Figure 35-8).

The medical treatment of OA is multifaceted and includes antiinflammatory medications, weight optimization, controlled exercise, physical modalities, alteration of the environment, and disease-modifying OA agents. Veterinarians must impress on owners that the management of chronic OA is a lifelong commitment and is hard work. It is critical to evaluate patients on a regular basis and provide feedback and encouragement to owners. Management of the arthritic patient should be approached in a logical, stepwise progression.

Antiinflammatory Agents

Osteoarthritis is classified as a noninflammatory joint disease because of the relatively low number of inflammatory cells in the synovial fluid. However, many inflammatory mediators (e.g., prostaglandins and leukotrienes), degradative enzymes (metalloproteinases) released as a result of cell membrane injury, and inflammatory cells are intricately involved in a cascade of events leading to the deterioration of articular cartilage. Inflammatory products are produced through enzymatic action (phospholipase A2) on cell membrane phospholipids to produce arachidonic acid. Cyclooxygenase (COX) and lipoxygenase (LOX) act on arachidonic acid to produce prostaglandins, leukotrienes, and related products known as eicosanoids. Nonsteroidal antiinflammatory drugs (NSAIDs) have been the foundation for medical management of OA, particularly in advanced cases. NSAIDs inhibit the COX enzyme, thereby decreasing inflammatory mediators and reducing pain associated with OA. Two isoforms of the COX enzyme have been identified, COX-1 and COX-2. Cyclooxygenase-1 is a constitutive enzyme and is normally produced in physiologic amounts in many tissues. It has protective functions, such as protection of the gastric mucosa, maintaining renal perfusion, and maintaining normal platelet function. The inhibition of the COX-1 enzyme by traditional NSAIDs is believed to be responsible for the adverse side effects of gastric ulceration, prolonged bleeding time, and decreased renal perfusion. The COX-2 enzyme is primarily inducible, and its production increases in response to inflammation. Traditionally NSAIDs have inhibited both COX-1 and COX-2 enzymes. The identification of the two COX isoforms has resulted in research to develop products that selectively inhibit the inducible COX-2 enzyme, while sparing the constitutive COX-1 enzyme. Selective inhibition of COX-2 with preservation of COX-1 could potentially reduce the adverse effects associated with the gastrointestinal tract and kidneys. NSAIDs that are frequently used in veterinary medicine include carprofen, deracoxib, firocoxib, etodolac, and meloxicam. Acetaminophen with or without codeine is occasionally used in dogs, but should never be used in cats. COXIB-class drugs are those that are diarylheterocyclic compounds that preferentially and irreversibly bind the COX-2 enzyme and are relatively COX-1 sparing. Deracoxib and firocoxib are COXIB-class drugs that are approved for use in veterinary medicine. Carprofen, etodolac, and meloxicam also have some preferential selectivity to inhibit mainly COX-2. No NSAID has been shown conclusively to clearly be more efficacious than the others. The effectiveness of various NSAIDs appears to vary between patients.[15] One NSAID could be more effective than another in a patient, and the reverse could be seen for another patient. This means that, for each patient, the optimal NSAID may be identified after several

different NSAIDs are used (successively but not concurrently) for 2-week trial periods. A washout period of a few days should be employed, as long as the patient is not in excessive pain during the washout period. It is important to have owners also evaluate the response to treatment because they evaluate the patient in the home environment in which the patient negotiates various obstacles and situations that are not present in a veterinary clinic. Caution must be used when using any NSAID because of their potential side effects. Before prescribing any medication, the patient's physiologic state, especially liver and kidney function, should be assessed. It is important to educate the owners regarding potential side effects, including gastrointestinal events (primarily vomiting and diarrhea), hepatic disease, and renal disease.

Certain forms of polyunsaturated fatty acids (PUFAs), especially omega-3 fatty acids, may reduce the production of certain eicosanoids, especially the more potent inflammatory 4-series leukotrienes, and help reduce inflammation. Some humans with rheumatoid arthritis respond to treatment with PUFAs. Polyunsaturated fatty acids, particularly eicosapentaenoic acid, appeared beneficial to dogs with OA in two studies.[16,17]

Oral or injectable corticosteroids are also prescribed for patients with OA. Steroids are effective antiinflammatory drugs and result in clinical improvement in patients. However, their use as a short-term solution in lieu of other modalities should be avoided because of their probable long-term adverse effects on cartilage and other body systems. For example, glycosaminoglycan (GAG) synthesis by chondrocytes is reduced 30% in patients receiving prednisone. Although veterinarians may feel pressured to provide quick results, client education is critical to emphasize that management of OA is a lifetime endeavor.

Slow-Acting Disease-Modifying Osteoarthritic Agents

Agents thought to alter the course of OA are termed slow-acting disease-modifying osteoarthritic agents because it is thought that they improve the health of the articular cartilage or synovial fluid. These agents may be more beneficial in early OA than in end-stage OA. Nutraceuticals are nutritional supplements believed to have a positive influence on cartilage health by providing precursors necessary for repair and maintenance, and they may also alter the pathophysiology of OA and its progression. Glucosamine and chondroitin sulfate (CS) are routinely combined as disease-modifying agents. Glucosamine is a precursor to the disaccharide units of GAGs, which comprise part of the proteoglycan (PG) articular cartilage matrix. Studies have shown that glucosamine helps to improve cartilage metabolism and upregulates PG synthesis.[18,19] Chondroitin sulfate is the predominant GAG found in articular cartilage. Extracellular and intracellular mechanisms are stimulated by CS to produce GAG and PG. Chondroitin sulfate also competitively inhibits degradative enzymes found in cartilage and synovium.

Polysulfated glycosaminoglycans (PSGAGs) is a drug and is administered as an intramuscular injection. Polysulfated glycosaminoglycans reduce the production of metalloproteinases and increase production of hyaluronic acid and cartilage GAGs. In addition they improve stifle ROM, clinical use of the limb, and the health of the synovium in dogs recovering from experimental cranial cruciate ligament transection and stifle stabilization surgery.[20] Hyaluronic acid (HA) is a nonsulfated GAG and a major component of synovial fluid and cartilage. Hyaluronic acid is available as an intraarticular injection and helps increase synovial fluid viscosity, reduces inflammation and prostaglandin production, and scavenges free radicals. Some patients could potentially benefit from periodic administration of HA.

Obesity

Obesity is strongly associated with the development of OA in people and likely contributes to the progression of OA in dogs.[21,22] For example heavy people are 3.5 times more likely to develop OA than light people, and loss of 5 kg decreases the odds of developing OA by greater than 50%.[23] Additionally, weight loss results in less joint pain and a decreased need for medication to treat OA.[24] In one clinical report weight reduction of 11% to 18% of the initial body weight of obese dogs resulted in improvement of hindlimb lameness associated with hip OA.[25] Another study of obese dogs with hip dysplasia indicated that there is a significant increase in weight bearing as measured with a force plate following weight loss.[26] In addition the owners felt that weight loss resulted in significant improvement in a number of areas in the home environment.

In addition to restricting intake of the normal diet and eliminating treats, prescription diets are available that can help achieve and maintain ideal body weight. In general, a goal is to reduce body fat composition to 20% to 25% of an animal's total body weight. Clinically the ribs should be easily palpable, and there should be a "waist" when the animal is viewed from above and the side.[27]

Physical Rehabilitation Modalities

Mild to moderate exercise and training in normal humans and dogs do not cause OA by themselves, but articular cartilage may undergo biochemical, histologic, and biomechanical changes.[28-31] Most studies of moderate running have indicated that there is no injury to articular cartilage with this form of activity, assuming that there are no abnormal biomechanical stresses acting on the joints. Heavy training programs, however, result in changes that may predispose to the development of OA.[32-34] Controlled therapeutic exercise is a valuable but underused management option for OA patients. Arthritic humans participating in controlled, low-impact exercises have improved function

and reduced pain and need for medication.[35,36] The goals of therapeutic exercise should be to reduce body weight, increase joint mobility, and reduce joint pain through the use of low-impact exercises designed to strengthen supporting muscles. Muscle disuse results in atrophy and weakness. Because muscles act as shock absorbers for joints, strengthening periarticular muscles may help protect joints. Mild weight-bearing exercise also helps stimulate cartilage metabolism and increases nutrient diffusion. Exercise may also increase endogenous opiate production; indirectly decreasing OA pain.[37] Exercise programs are tailored to the condition and personality of each patient and to their owners (Box 35-3). An improper program could hasten the progression of OA. Overloading joints should be minimized by performing low-impact activities, such as walking and swimming, until weight loss occurs. Unrealistic demands placed on the owner will likely decrease compliance, and the physical condition of the owner must be considered. Joint instability should be corrected before initiating an exercise program. Exercise programs must be tailored to account for the typical course of exacerbations and remissions of OA. The animal should not be forced to exercise during times of exacerbation of the arthritic condition because inflammation may increase. In preparation for exercising, warming and stretching affected muscle groups and joints during a warm-up period are recommended.[38,39] Tissue warming promotes blood flow to the area, promotes tissue and collagen extensibility, and decreases pain, muscle spasms, and joint stiffness. Heat is contraindicated if swelling or edema is present in a limb or joint. Heating agents such as moist or dry hot packs, circulating warm water blankets, and warm baths typically heat the skin and subcutaneous tissues to a depth of 1 to 2 cm. Another physical agent used for heating is therapeutic ultrasound. Ultrasound frequencies of 1 and 3 MHz in continuous mode produce thermal and nonthermal effects. The effects are related to the treatment time, intensity, frequency, and area being treated. Tissue heating may penetrate to 5 cm, much deeper than with superficial heating modalities. Nonthermal effects include increased cell membrane permeability, calcium transport across the cell membrane, removal of proteins and blood cells from the interstitial spaces, and nutrient exchange. Any stretching should be done during the latter part of warming or immediately after. Massage also has been used to increase blood flow to muscles to warm up the area before activity, and to decrease stiffness after activity. Electrical stimulation of muscle also appears to be of benefit when treating dogs with OA. Transcutaneous electrical stimulation (TENS) was applied to the stifle joint of dogs with experimentally induced OA. Peak vertical force was significantly increased after TENS application in these dogs.[40]

Controlled leash walking, walking on a treadmill, jogging, swimming, and going up and down stairs or ramp are excellent low-impact exercises (Figure 35-9). The length of the exercise should be titrated so there is no increased pain after activity. Also exercise sessions should be short in the early phases of training: three 10-minute sessions is preferred to one 30-minute session. Walks should be brisk and purposeful, minimizing stopping. Avoiding sudden bursts of activity will help avoid acute inflammation of arthritic joints. Swimming and walking in

Box 35-3 Guidelines for an Osteoarthritis Home Care Program

Osteoarthritis tends to follow a course of exacerbation and remission. During a period of aggravation, do not force exercise. Low-impact exercises, such as leash walking and swimming, are ideal.

One sign of osteoarthritis is stiffness, especially in the morning. Begin the day by taking time to warm up the joints. This can be done using warm water bottles, hot packs, heating wet towels in the microwave (comfortable enough for you to tolerate on your own skin), or using a bathtub/whirlpool (≈85°) for 15 to 20 minutes.

Following warm-up, apply passive range of motion to the affected joints. Slowly flex and extend the joints—DO NOT force beyond a comfortable range. Fifteen to twenty SLOW repetitions are sufficient. This helps to decrease stiffness and increase motion in the joints.

Continue throughout the day taking the dog on several short walks (at least three) with long rest periods in between. Let the patient set the time and distance. Begin walking 10 to 15 minutes at a time, gradually increasing the time and length. Multiple short walks are more beneficial than one or two long walks. Note any discomfort, increased fatigue or stiffness, or refusal to go any further. Should this occur, cut the previous level of activity in half and continue from there.

Differentiate between muscle soreness and joint pain:

- Muscle soreness—painful upon palpation of muscle groups
- Joint pain—painful when flexing or extending a joint

As tolerance and endurance improve, activities to further improve mobility, reduce pain, and increase muscle strength may be added. Some ideas may be to incorporate ramps, inclines, declines, or stairs into the walking program in incremental amounts. Once again, let the patient set the boundaries.

Following exercise, a period of cool-down is necessary. Massage and use a bag of frozen vegetables or an ice pack on the joints for 15 to 20 minutes to minimize any postexercise swelling that may occur.

No antiinflammatory medications should be given for the first day when beginning a rehabilitation program. The antiinflammatory effect may mask painful joints associated with exercise progression. After a day if no joint pain is noticed, medications may be given 1 hour before exercise. Withhold medication again as exercise is increased to be certain that the medication is not masking excessive pain.

Osteoarthritis treatment is a lifelong commitment. If rehabilitation is discontinued, any benefits gained will likely be lost.



Figure 35-9 Going up and down an inclined ramp is an excellent low-impact exercise. A handicap access ramp also makes it easier to get into buildings.



Figure 35-10 Training on an underwater treadmill may significantly increase weight bearing comparable to achievements obtained using medication in many patients.

water (Figure 35-10) are some of the best activities for dogs. The buoyancy of water is significant and limits the impact on the joints while promoting muscle strength and joint motion. If joint pain is perceived to be greater after exercising, the length of the activity can be decreased by half. When stepping up the amount of activity, the increase should be approximately 10% to 15%, once a week unless increased pain or decreased function occurs. Ideally, anti-inflammatory drug use should be minimal and consideration should be given to avoid administration immediately before stepping up activity because it is important to determine if the level of exercise is too great and causes pain. The exercise periods should be evenly spaced throughout each day and over the entire week. Training and conditioning help maintain an ideal body weight, improve ROM, and increase muscle strength and tone, which help to stabilize joints. Following exercise a 5 to 10-minute cool-down period is recommended. A slower paced walk may be initiated for 3 to 5 minutes, followed by ROM and stretching exercises. A cool-down massage may help decrease pain, swelling, and muscle spasms. Finally, cryotherapy (cold packs or ice wrapped in a towel) may be applied to painful areas for 15 to 20 minutes to control postexercise inflammation. Application of cold decreases blood flow, inflammation, hemorrhage, and metabolic rate.

Environmental Modifications

Altering the environment may be helpful for dogs with moderate to severe arthritis. The principles of environmental modifications for dogs are similar to those for arthritic humans.[41] Whenever possible animals should be moved from a cold, damp environment (i.e., outdoor) to a warm, dry environment (indoor). A soft, padded bed should be provided. A circulating warm-water blanket under the blankets provides heat that may reduce morning stiffness. Provide good footing to avoid slipping and falling. Minimize stair climbing through the use of handicapped ramps and keeping pets on ground floors. Steps are negotiated more easily if they are wider, have a gradual rise, and are spaced farther apart. Portable ramps are available to assist patients getting in and out of vehicles. Avoid overdoing activities on the weekends, and prevent excessive play with other pets because arthritic animals may attempt to keep up, and in the process, become more lame and painful. In some instances, however, play with other animals stimulates activity and provides a welcome break in the exercise routine.

Rehabilitation for the Patient with Neoplasia

Geriatric patients with neoplasia may benefit from specific rehabilitation programs. For example fibrosis of the skin and subcutaneous tissue may result from radiation therapy. When such fibrosis involves limb tissues, the ROM of a joint may be limited and limb use may be negatively impacted. A gentle massage of the affected region, stretching, and ROM exercises may help limit the impact of fibrosis. It is particularly critical to develop a comprehensive pain management program in cancer patients because the chronic pain associated with some neoplasms negatively affects the quality of life of patients and may lead to anorexia and decreased ambulation. In addition to all the conventional strategies used for pain management, fentanyl patches may be used to provide pain relief in the short term. Palliative radiation therapy may also be used to provide pain relief in a limb with osteosarcoma. Although

the majority of patients respond positively to three or four sessions of palliative radiation therapy generally spaced a week apart, the likelihood of fracture of the affected bone increases after the therapy. The rehabilitation programs of geriatric cancer patients are designed to be gentle, low-intensity exercises and manipulation to minimize the stress to the patient.

Summary

The optimal rehabilitation of geriatric patients involves a thorough initial patient assessment, focusing on the identification of underlying musculoskeletal and metabolic disorders, weight management, OA management (if necessary), environmental modifications that promote low-stress activities, and patient assessment by the veterinarian at regular time intervals to monitor patient progress.

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36 Physical Rehabilitation for Wound Care

June Hanks and Sallye Gregg

Patients undergoing rehabilitation frequently have wounds, including surgical wounds and wounds resulting from nonsurgical trauma. Therefore, an understanding of wound healing and the care of wounds is necessary to speed the rehabilitation process and identify abnormal wound healing. Normal restoration of injured tissue follows a series of predictable overlapping events often grouped as the inflammatory, proliferation, and maturation/remodeling phases of wound healing (Figure 36-1). Biochemical substances modulate cellular activities occurring during the phases of tissue repair. Acute wounds tend to follow a predictable progression through the wound healing phases, whereas chronic wounds may have a prolonged period of inflammation, proliferation, or excessive wound contraction.

This chapter reviews the wound healing phases and discusses factors that may delay the tissue restoration process. Modalities, dressings, and alternative treatments to speed the healing process are discussed.

Wound Healing Physiology

Inflammatory Phase

The first response to injury is inflammation, a process that includes vascular and cellular events to control blood loss, combat bacterial invasion, and set the stage for further healing by signaling the cells necessary for repair and regeneration to come to the site of injury. For a few seconds to several minutes following injury, blood vessels in the area reflexively constrict to reduce blood loss. Platelets aggregate at the injury site and are activated by contact between damaged endothelial cells lining the blood vessel walls and exposed collagen. The activated platelets adhere to one another and the injured endothelium and interact with fibrin to help form a plug to wall off the injured area. The activated platelets also release chemical mediators such as cytokines (signaling proteins), growth factors (hormone-like substances that control cell metabolism), and chemotactic agents (substances that attract cells important for repair to come to the site of injury).[1,2]

Following vasoconstriction blood vessels dilate, allowing more fluid, inflammatory cells, and growth factors into the interstitial space. The walls of injured blood vessels allow fluid to leak into the interstitial space, causing localized edema that serves to increase pressure in the interstitial space and decrease pressure in the blood vessels. As the wound area accumulates more proteins, white blood cells, and cell fragments, the fluid in the interstitial space becomes more viscous and changes from a transudate to an exudate. Mast cells in the area release inflammatory mediators and secrete enzymes. Vasodilation is induced by histamine released by mast cells and prostaglandins released by injured cells.[3] Swelling and chemical release in the area continue to increase, causing pain. The entire series of events leads to the hallmark signs of inflammation: edema, heat, redness, pain, and decreased function.

Cellular events that accompany the vascular events include margination of neutrophils to the sides of the blood vessel wall and eventual migration through the wall. Guidance to the injured area occurs as neutrophils follow the chemical gradient formed by bacteria and dead or dying cells through a process called *chemotaxis*. Circulating monocytes migrate into the tissue spaces and are then called *macrophages*. Neutrophils and macrophages release enzymes, engulf bacteria, produce tissue proteases that further degrade debris, and release chemotactic agents and chemical mediators that attract more inflammatory cells to the area. Although less effective than neutrophils in the phagocytic process, macrophages produce numerous growth factors and are critical in directing the tissue repair process.[4,5]

Proliferative Phase

As the cells necessary for tissue repair respond to signals to enter the injured area, the proliferative phase of wound repair begins. Endothelial cells adjacent to the site of injury respond to ischemia, growth factors, and chemical mediators and begin to bud into the injured area through the process of angiogenesis. As buds connect through capillary loops, nutrients are carried to the area and waste products are removed. The blood vessel buds appear as tiny red dots in the tissue space. Previously damaged lymphatic vessels begin to function again and work with the blood vessels to reduce local tissue edema. Proteases produced by various cells work alongside phagocytic cells to degrade wound debris.

Fibroblasts from the local tissue and general circulation begin to fill the spaces between the capillary loops. As the fibroblasts proliferate among the capillary loops, an

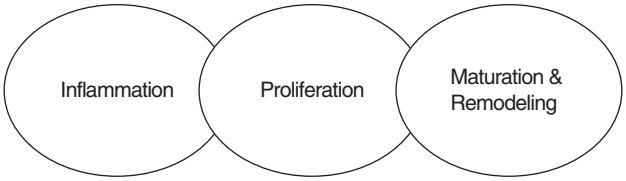


Figure 36-1 Overlapping phases of wound healing.



Figure 36-2 Granulation tissue covering a large wound.

extracellular matrix composed of water, proteoglycans, and other cells is formed. The matrix fills the space between collagen and elastin fibers in the wounded area, allowing more fibroblasts to migrate across the wound bed. The newly formed vascularized connective tissue is called granulation tissue (Figure 36-2). Chemotactic agents and oxygen levels guide fibroblasts to migrate and proliferate, thus filling in the wound bed. Integrins, surface cell receptors, serve as chemotactic agents and adhesion molecules to assist the migration of fibroblasts within the extracellular matrix.[1] As oxygen levels increase in the wounded area, less proliferative activity is stimulated. Some of the fibroblasts transform into myofibroblasts which have contractile properties and begin to pull the wound edges inward. The wound contraction decreases the size of the defect. In larger and deeper defects, more granulation tissue is needed to fill the void calling for a greater degree of wound contraction. The shape of the wound affects the speed of contraction. Linear wounds contract faster than rectangular wounds. The slowest to contract are circular wounds.

Epithelial cells (primarily keratinocytes) migrate across the granulation tissue bed from the wound margins and epidermal appendages to cover the wound defect. Growth factors stimulate epithelial cell proliferation while oxygen levels and chemotaxis guide migration across the wound bed. Ease of migration is facilitated in a moist wound environment.[6] After the wound has been completely resurfaced with epithelial cells, the proliferative phase is considered complete.

Maturation/Remodeling Phase

Although the wound bed is resurfaced through the cellular and vascular events in the inflammatory and proliferative phases, the wound is not yet completely healed. The newly formed tissue must be strengthened and reorganized through collagen synthesis and lysis in the newly formed scar tissue. Through chemical events, internal stress, and external tension, the collagen fibers transform from immature type III collagen to mature type I collagen.[7] Interventions of range of motion exercise and scar tissue mobilization aid the process of scar tissue remodeling eventually achieving a balance between collagen formation and degradation to produce a strong healthy scar. Most of the remodeling of the scar occurs in the first 6 to 12 months after wound closure, but can continue for up to 2 years. Even when fully remodeled, the scar tissue attains only 75% to 80% of the strength and elasticity of the original tissue.[8]

Types of Wound Closures

Wounds that heal following approximation of the wound edges with little or no granulation tissue formation (as in a surgical wound) are said to heal by primary closure or primary intention. An uncontaminated, clean wound bed with wound margins that can be brought together with little tension on the surrounding tissues is required for healing by primary closure. Healing by secondary closure, or secondary intention, occurs when the wound edges cannot be brought together. The process of granulation tissue formation, wound contraction, and epithelialization is required to fill the wound defect. Healing by secondary closure is more complicated, results in more scar tissue formation, and requires more time and energy to achieve closure than does healing by primary intention. In some cases, a traumatic or surgical wound cannot be immediately closed because of contamination or risk of infection. The wound may be cleaned over a period of a few days. When the wound is free of debris, it may be cleaned, surgically debrided and closed. Such a wound is said to heal through delayed primary closure.

Abnormal Wound Healing

A variety of factors may result in failure of an acute wound to move through the inflammatory, proliferative, and maturation phases of healing in a predictable fashion. Wounds may become chronic because of a larger number of proteases and lack of protease inhibitors, leading to a degradation of the extracellular matrix and a larger wound bed. Chronic wounds have higher amounts of inflammatory immunomodulating agents and are less responsive to growth factors. Patients who are immunocompromised or who have arterial insufficiency may be unable to mount the inflammatory response necessary to clean the wound of debris and produce chemotactic cells that signal other cells necessary for the production of granulation tissue in the wounded area. Such wounds may be void of the classic signs of inflammation. In contrast, some wounds may persist in the inflammatory phase, exhibiting debris, rolled,

Table 36-1 Factors Affecting Wound Healing

Wound Characteristics	Local Factors	Systemic Factors	Improper Management
Time since onset	Circulation	Age	Allow wound to become too wet or too dry
Location	Sensation	Inadequate nutrition	Failure to fill wound cavity with dressing
Dimensions	Mechanical stress	Co-morbidities	Failure to thoroughly cleanse wound
Temperature		Medication	Overuse of cytotoxic agents
Hydration			
Necrotic tissue			
Infection			

Myers BA: *Wound management: principles and practice,* ed 2. Englewood Cliffs, NJ, 2008, Pearson Prentice Hall.

thickened wound edges, and a lack of adherence of the wound edge to the wound bed. Common causes of chronic inflammation include the presence of a foreign body in the wound bed, repetitive trauma (e.g., excessive licking of the wound bed), and the overuse of agents that are cytotoxic to the cells involved in the healing process. Currently there are insufficient data to assess safety, efficacy, and recommendations for use of some topical agents because of inconsistency or lack of verification of equivalent comparisons between in vitro and in vivo studies.[9] Agents in question include povidone-iodine, hydrogen peroxide, sodium hypochlorite bleach, and chlorhexidine.[10-12]

Some wounds may granulate too little (hypogranulate) because of poor support for a granular matrix. A "pothole" wound is formed as epithelial cells pile up at the wound edge and fail to migrate across the wound bed. The wound edges of such pothole wounds should be gently wiped with gauze and the wound cavity should be lightly filled with a dressing to promote the formation of a granular matrix and prevent premature migration of epithelial cells. In contrast, hypergranulation tissue may be formed when a wound defect has been filled, yet the granulation process continues, resulting in granulation tissue that extends well above the wound surface. Hypergranulation tissue, or *proud flesh*, can be resolved by the application of pressure to the tissue through certain dressing types, such as hydrocolloid dressing, the use of silver nitrate to cauterize the tissue and level the wound bed, hypertonic saline dressings, or surgical excision of excess tissue.

Factors Affecting Wound Healing

Deterrents to expedient wound healing include wound characteristics, local factors, and systemic factors (Table 36-1). Acute wounds and surgically induced wounds tend to heal more quickly than severe traumatic wounds or wounds of insidious onset. Wounds located over bony prominences, in areas with few epidermal appendages, across joints, and in areas of thick skin have slower healing times. Because more cellular activity and rebuilding of tissue are required, larger and full-thickness wounds require more healing time than smaller or more superficial wounds. The close proximity of wound edges results in shorter healing times for linear wounds than for rectangular or circularly shaped wounds. Maintenance of appropriate wound moisture within the wound bed encourages granulation tissue formation and epithelialization. Thus, wound beds and periwound areas that are too dry (desiccated) or too wet (macerated) have prolonged healing times. Devitalized tissue within the wound bed and dry exudate or scabs can slow the migration of epithelial cells across the wound bed, prolong the inflammatory response, and create an environment conducive to infection.[13,14]

Important to consider is the fact that all wounds are contaminated. All body surfaces, including skin, and the digestive tract have a normal microflora of bacteria and fungi that include *Staphylococcus*, *Micrococcus*, *Peptococcus*, *Streptococcus*, *Acinetobacter*, and yeast. The resident microflora and pH of the skin create a slightly acidic environment, serving a protective function against disease-causing microorganisms.[15] In an open wound the microflora adhere to the surface and replicate, resulting in colonies that do not necessarily adversely affect the host as the normal immune response is activated. However, if the bacteria or fungi continue to replicate, a point of critical colonization is reached in which the microorganisms become a bioburden, adversely affecting the host. When microorganisms multiply to the point of invading viable body tissues, the wound is considered infected. Because of the presence of individual factors in each host, there is no specific threshold for infection, although in general, wounds with a bacterial quantity of greater than 10^5 microbes per gram of tissue are considered infected.[16] However, the type of microorganism must also be considered because some are more virulent and produce exotoxins that may inactivate or modify host responses, leading to host cell dysfunction or death. Bacterial endotoxins, molecules within the walls of gram-negative bacteria, may induce a strong inflammatory response leading to fever, bleeding, and clotting. In addition interactions among bacteria can lead to a microorganism community called a biofilm that attaches to the wound surface and is encased in a self-developed glycocalyx

(which protects the bacteria). The microorganisms encased in the biofilm exhibit increased virulence and resistance, making them less responsive to biological and pharmaceutical intervention.[17] Immunocompromise, malnutrition, and impaired local circulation of the host can also affect resistance to invading organisms.

In general, infection should be suspected when the signs of inflammation are disproportionate to the size and extent of the wound (e.g., drainage is copious, has uncharacteristic color, wound is very painful, periwound area is extremely red or hot, etc.). Wound cultures such as tissue biopsies, swab cultures, or fluid aspirations may be used to identify an offending microorganism. Although tissue biopsy is the gold standard for culturing, properly performed swab cultures are a reliable method to identify and quantify bacteria in a wound.[18] To properly perform a swab culture, the clinician must first debride and thoroughly rinse the wound bed. The swab should then be moved slowly over viable tissue in the wound bed in a crisscross pattern back and forth, moving from top to bottom while rolling the swab and applying enough pressure to express some of the wound bed fluid. Wounds with a small surface area of little granulation tissue should be cultured by swabbing only the small area for 5 seconds while rotating the applicator.[19] Tissue biopsies are preferred for evaluating pressure/decubital ulcers, burns, or wounds with a suspected neoplastic process.

Because endogenous or exogenous microbes can lead to infection, meticulous adherence to infection control procedures must be exhibited. Procedures include hand washing before and after glove use, the use of gloves, which should be changed between procedures on the same patient and promptly after touching contaminated materials, and isolation of patients with antibiotic-resistant infections.

Emergency Care

In caring for a patient with a wound, the safety of the patient and staff is of upmost concern. When in pain, pets can become suddenly aggressive. Staff should have muzzles and blankets readily available for use as necessary. A muzzle can be used during the administration of parenteral pain medications and placement of an intravenous (IV) line if patient respirations are normal. For patients having problems breathing, a thick blanket can be placed over the animal to control the head during the evaluation and administration of pain medications. It is necessary to exercise universal precautions including the use of gloves to protect the provider from human blood that may be present on the coat of the patient and to protect the wounded area of the patient from bacteria that may be spread by health care workers.

Medications to relieve pain should not be withheld from a patient, because analgesic agents do not prevent a full patient evaluation from being completed. Pain medications do not mask the physiologic indicators, because the heart rate will still be elevated in response to hypovolemia, hypoxia, hypotension, or hypercarbia, even when high doses of opioid medications have been delivered to the patient. In the emergency patient opioids are unlikely to cause respiratory depression or the deterioration of hemodynamic parameters.[20] The use of nonsteroidal antiinflammatory drugs (NSAIDs) and corticosteroids are contraindicated for use in a patient in shock until renal perfusion, blood pressure, and dehydration issues have been addressed. In addition, endogenous endorphins released in a stressful situation, such as an emergency room, can mask the seriousness of the patient's condition; therefore chemical sedation for fear and aggression is a humane way to examine the emergency patient. An oxygen mask or oxygen induction chamber allows the patient to relax while medications are administered. [21]

Severity of the injury determines the proper medications for pain control. Severe to excruciating pain present after trauma with fractures and muscle damage require high-dose opioids and additional pain medications for adequate relief. Fractures should be immobilized as soon as possible to reduce pain. Muscle hemorrhage, hematomas, or bruising may cause significant discomfort. These injuries may benefit from local application of ice during the first 72 hours or until the acute inflammatory phase has ended, and heat application thereafter. For injuries to the perineum, pelvis, and rear limbs, epidural anesthesia may be necessary if opioids are inadequate to control pain. After the patient has been stabilized, adjunctive medications such as NSAIDs, medications for nerve pain, and low-dose sedatives may be administered.[1]

Nursing care is extremely important in controlling pain and anxiety. The patient should be kept clean, warm, and dry on thick padded bedding. The extra attention and care shown to the patient may decrease the anxiety level and lessen the length of time the patient must be hospitalized.

Initial Assessment

Although external bleeding wounds often capture the attention of the owners, the health care worker must be aware that more life-threatening injuries may be present. The initial stabilization includes an examination of the airway with administration of oxygen, as appropriate. In the case of severe head wounds, facial trauma or tracheal damage, rapid induction may be necessary to accomplish intubation and obtain an open airway (Figures 36-3 and 36-4). Puncture wounds or trauma to the chest wall should increase suspicion of a pneumothorax, for which a thoracocentesis or chest tube may be necessary. All vital signs should be obtained to evaluate the need for IV fluids and/or blood products to treat hypovolemic shock.

Bleeding from a wound may be controlled following or simultaneously with establishing an adequate airway and stabilizing hemodynamic parameters. Capillary bleeding and venous hemorrhage normally can be controlled with gauze sponges, direct pressure, and a bandage. With



Figure 36-3 Puncture wounds over the neck of a dog that sustained bite wounds.

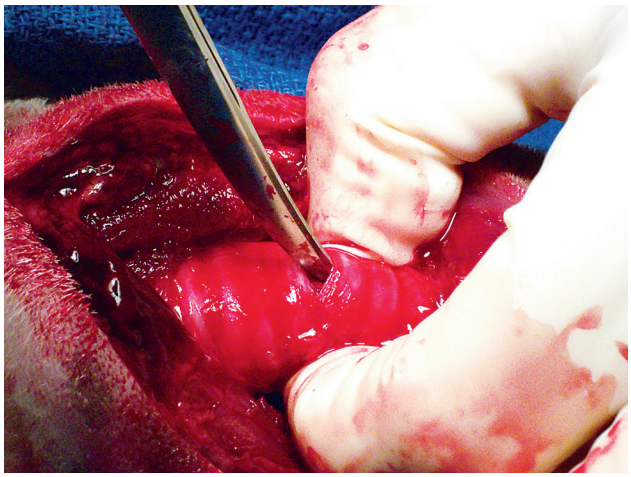


Figure 36-4 Exploration of the wound in Figure 36-3 resulted in finding a laceration of the trachea.

a gloved hand, a sterile gel should be placed in the wound and the wound should be covered with a layer of gauze sponges. Compression over the gauze sponges may be maintained with a layer of elastic bandage or tape until the wound can be definitively addressed. More aggressive therapy is needed for arterial bleeding. For appendicular wounds, a 5- to 10-cm wide blood pressure cuff can be applied proximal to the wound. The cuff is inflated to 20 to 30 mm Hg higher than the systemic arterial pressure to control the bleeding while sedation is given. The inflated cuff can serve as a temporary tourniquet for 30 to 45 minutes while the patient is sedated and prepared for surgical exposure to the artery. A sterile hemostat is applied to the bleeding vessel and ligation with a small diameter antimicrobial suture is performed. Even though the blood pressure cuff can be left in place for an extended period without permanent damage to the limb, the pressure should be released every 5 to 10 minutes to allow blood flow to and from the region. Commercial tourniquets made from narrow bands of elastic material or Penrose drains should not be used because they can place excessive pressure on neurovascular structures and cause permanent damage.

After life-threatening injuries have been addressed, a thorough examination of the wounded area should be conducted. Traumatic wounds are classified according to type and contamination. Wound types include lacerations, avulsions, shearing injuries, punctures or bites, penetrating wounds, and pressure necrosis (Table 36-2). Wounds also may be classified based on degree of contamination (Table 36-3).

The initial lavage, debridement, and aftercare are to encourage healthy tissue that is free of infection and necrosis, and has a good blood supply. The golden period of wound care refers to the first 6 hours after the traumatic event occurred. It is during this time that the wound may be cleaned and closed without the development of infection. While stabilization is occurring, the wound should be kept as moist as possible by placing sterile water-soluble gel into the wound and covering with a saline-soaked sponge. Wounds that are kept moist heal up to 50% faster than wounds that are allowed to dry out. Wide clipping of the wound enables the clinician to examine the injury and often exposes wounds that were not apparent upon initial inspection. In the case of a bite wound, the opposite side of the affected limb or body part should be carefully examined for a potential matching injury from the rest of the teeth involved in the bite.

The wounded area must be cleansed of all debris and blood. In a grossly contaminated wound, tap water is practical to use for the first cleaning. Obvious debris can be washed away with warm water using a gentle spray attachment. A surgical scrub solution should then be applied to the intact skin and the area around the wound. Detergent-containing scrubs are cytotoxic and may delay healing, especially if used for extended periods of time. An exception is the topical microbicide chloroxylenol (brand name Techni-Care®), which, in a retrospective clinical trial, appeared to decrease infection rates and allow the wound to progress toward healing[22] (Table 36-4).

Following the removal of gross contamination and cleansing of the surrounding skin, the wound must be lavaged with large amounts of fluid (at least 500 mL). The statement, "The secret to treating pollution is dilution" truly applies to initial wound management. Fluid can be sprayed on the wound using an 18-gauge catheter attached to a 35-mL syringe via a three-way stopcock. A warm l-L bag of fluid is also attached, enabling the operator to rapidly fill the syringe and flush the wound. This mechanism provides a pressure of 7 to 9 pounds per square inch (PSI), which is sufficient for decreasing the bacterial population, but not so high as to drive the bacteria deeper into the tissues. Room temperature or cold lavage solutions are uncomfortable and possibly painful to the patient. The least toxic, most inert lavage solution is ideal. The topical application of a mixture of sugar or honey, sugar and povidone-iodine (PI), or honey and PI may be used to promote granulation tissue formation and reduce bacteria in the wound. Used alone or in combination with PI, sugar acts to inhibit bacterial growth and modulates keratinocytes and fibroblasts.[23,24]

Table 36-2 Types of Traumatic Wounds

Laceration	The wound exhibits minimal necrosis or compression and has sharp, linear, or jagged edges. Normally this is a relatively clean injury that requires minimal debridement, but inspection of the deeper tissues is important.
Avulsion	The injury results from tensile forces pulling the tissue free of its natural attachments. Deep exploration with delayed closure is beneficial for this type of wound.
Shearing or degloving	The high friction of the pavement on the skin during a motor vehicle accident will often result in tearing of the skin and its attachments as well as damage to the deep vascular, nerve, and bony structures. These wounds have minimal to moderate contamination and benefit from delayed closure. Skin grafts and orthopedic repairs are common with this injury.
Punctures	The small skin wound is only the tip of the iceberg in this injury. The compressive forces and trauma to the underlying tissues from a bite injury involves significant disruption of the deep tissue planes as well as deep seeding of bacteria into the wound. Foreign bodies that range from hair to pieces of sticks and gravel are often found in these wounds. The wound should be opened and explored to clean, debride, and alleviate dead space. Delayed closure and antibiotics are beneficial in these patients.
Penetrating injuries from projectiles	These wounds are considered to be contaminated and require surgical exploration if the wound is over the abdominal cavity or the bullet is located within a joint.
Pressure necrosis	Devitalized necrotic tissue occurring from sustained pressure causing ischemia such as from a tightly wrapped bandage, lying for extended time periods on a bony prominence, or from crushing and vascular injury during a trauma.

Halling K: Wounds and open fractures. In Mathews KA, editor: *Veterinary emergency and critical care manual,* ed 2. New York, 2007, Lifelearn; Swaim SF, Henderson RA: *Small animal wound management.* Philadelphia, 1990, Lea & Febiger.

Table 36-3 Wound Classification/ Contamination

Clean	Injury created by a grossly clean object and is less than 6 hours old
Contaminated	Created by an object with bacterial contamination and/or is greater than 6 hours old
Dirty/infected	Old traumatic wound with purulent material or necrotic tissue present

Dernell WS: Initial wound management. *Vet Clin North Am Small Anim Pract* 36(4):714-773, 2006; Garzotto CK: Wound management. In Silverstein DC, Hopper K, editors: *Small animal critical care medicine.* St. Louis, 2008, Saunders.

Box 36-1 Wound Management Techniques to Create and Maintain a Moist, Clean Wound Environment

Dirty wound?	→	Clean it
Cavity/hole?	→	Fill it
Wet wound?	→	Use more absorptive dressing/change more frequently
Dry wound?	→	Use less absorptive dressing
Infected wound?	→	Address underlying cause, clean wound, use antimicrobial dressing

Principles of Wound Management

Basic principles of wound management include cleaning the wound, preventing infection, providing a moist wound environment, and attending to systemic needs. Provision of a moist, but not wet, wound environment leads to increased angiogenesis, decreased dehydration and cell death, enhanced autolytic debridement, increased epithelialization, and the provision of a bacterial barrier to decrease infection rates. The appropriate choice, technique of application, and frequency of dressing changes to maintain a moist wound bed facilitates granulation tissue formation and migration of epithelial cells across the wound bed (Box 36-1).

Wound Dressings

The ideal wound dressing provides an optimal moist wound healing environment, thermal insulation, gaseous exchange, pain relief, edema control, dead space elimination, protection from contaminants, and assistance with removal of dead tissue without disruption of the wound bed. The primary dressing or contact layer may be adherent or nonadherent, although in general nonadherent contact layers are recommended to prevent disruption of

Table 36-4 Properties of Surgical Scrubs

	Chlorhexidine	Povidone-Iodine	Alcohol	Techni-Care®
Length of cidal Capacity	>6 hr	3 hr	None	>6 hr
Ocular irritation	Yes	Yes	Yes	Minimal
Scrub time	8 min	3.5 min	11 min	0.5 min
Amount needed	10 mL	10 mL	N/A	2.6 mL
Tissue contraindication	Yes	Yes	Yes	None
Toxicity	Skin, eyes, ears, genitals	Skin	Skin, eyes	Nontoxic
Denatured by organic material	Minimal	Yes; inactivated by blood	No data	Minimal
Transdermal penetration	No	Yes	No	Yes
pH	5.84	4.0	6.8	7.2

Grubbs BC, Statz CL, et al.: Salvage therapy of open infected surgical wounds: a retrospective review using Techni-Care®. *Surg Infect* 1(2):109, 2000; Statz CL: Fighting wound infections. *Plastic Surg Prod* 11:34-40, 2005.



Figure 36-5 Various dressings used over multiple wounds.

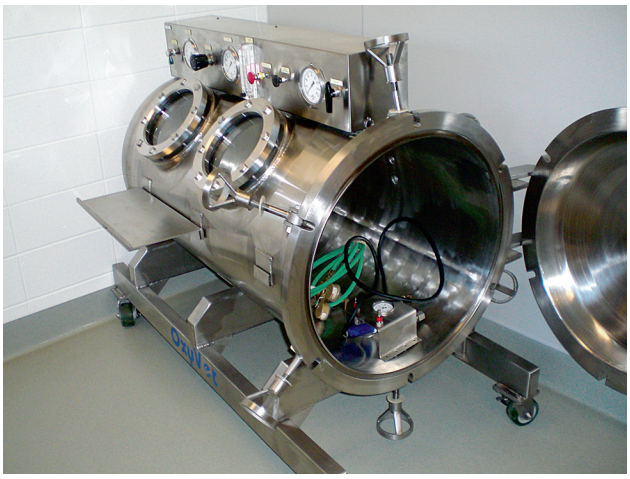


Figure 36-6 Small animal hyperbaric oxygen chamber.

granulating tissue when dressings are removed. Permeability and occlusive properties vary among dressings. Semiocclusive dressings are most commonly used to create a moist wound environment, allowing air to pass through the dressing and exudate to be absorbed into the dressing from the wound bed. The secondary dressing is placed over the primary dressing to increase protection, cushioning, absorption, or occlusion and may be used to hold the primary dressing in place[25] (Figure 36-5). Major dressing types include gauze, alginate, films, foam, hydrogels, and hydrocolloids (Table 36-5).

Other Wound Healing Therapies

Hyperbaric Oxygen Therapy

Hyperbaric oxygen therapy (HBOT), traditionally used to treat decompression illness (the bends), has been used for some time in wound care. Only minimal increases in plasma oxygen levels occur with oxygen delivery by mask or other indirect routes, whereas large increases of dissolved oxygen in the plasma occur with oxygen delivery under increased atmospheric pressure. Compared with normobaric conditions, the increase in partial pressure of dissolved oxygen in plasma and the increase in delivery of oxygen to the tissues provides an environment conducive for healing of ischemic or otherwise hypoxic tissues. Angiogenesis is stimulated, immune system function is improved, and proliferation is enhanced.[26]

Hyperbaric oxygen therapy chambers come in various sizes and can accommodate more than one small animal per treatment session (Figure 36-6). Each HBOT session is managed by trained personnel. Contraindications include deep vein thrombosis and congestive heart failure. Relative contraindications include severe arterial deficiency, chronic obstructive pulmonary disease, and pregnancy.

Table 36-5 Wound Dressings

Type of Dressing	Absorption Capacity	Benefits	Limitations	Common Uses
Gauze	Low (moderate if multiple layers are used)	Inexpensive	May adhere to wound bed; require more frequent dressing changes	Infected wounds; wound requiring packing; wounds requiring frequent dressing changes
Hydrogels	Low to moderate	Moisture retentive; encourage autolytic debridement	Cannot be used on highly exudative wounds; should not be used on infected wounds; generally require a secondary dressing	Soften eschar and provide moisture for dry wounds
Semipermeable foam	Moderate	Moisture retentive; may be left in place for several days; provides cushioning and insulation	Should not be used on infected wounds unless changed frequently; may roll in areas of friction	Minor burns; skin grafts; pressure ulcers
Hydrocolloid	Moderate	Impermeable to urine, stool, bacteria; provides thermal insulation	May traumatize periwound area on removal; may cause hypergranulation; should not be used on infected wounds	Pressure ulcers; burns
Alginate	High	Can be used on infected or uninfected wounds; are nonadherent and are biocompatible	Require secondary dressing; should not be used on dry wounds	Heavily exudating wounds
Silver-impregnated dressing	Varies, dependent on base dressing (e.g., hydrocolloid, alginate, foam, gel, etc.)	Broad spectrum antimicrobial properties	Distribution of silver within dressing and release of silver into wound greatly affects antimicrobial activity	To reduce bioburden in wound, reduce risk of infection

Electrical Stimulation

The effectiveness of electrical stimulation as an adjunctive therapy to wound healing is supported in the literature.[27-31] Electrical stimulation is thought to facilitate wound healing by providing electrical current to the injury, causing galvanotaxis, stimulating cells, enhancing blood flow, combating infection, reducing edema, and enhancing autolysis. Indications include chronic or recalcitrant wounds. Contraindications include untreated osteomyelitis and concurrent use with dressings containing heavy metal ions. Common methods of treatment include high volt direct current with direct or periwound application at a frequency of 80 to 125 Hz, submotor (no contraction seen) intensity of 75 to 200 volts, with 45 to 60 minutes of treatment 3 to 7 days a week, or 30 minutes twice a day. With the direct treatment method of placing an electrode directly in the wound bed and the ground electrode placed in a location to maximize current flow through the wound. Polarity recommendations vary but most recommend placement of the cathode in the wound bed for an infected wound, and placement of the anode in the wound bed when promotion of granulation tissue and epithelialization are desired. The polarity should be changed when a plateau of healing is observed.[32]

Negative Pressure Wound Therapy

Use of negative pressure wound therapy (NPWT), also known as vacuum-assisted closure, may enhance wound healing through topical subatmospheric pressure applied to the wound bed. An open-cell foam cut precisely to the size of the wound is placed within the wound bed and an evacuation tube with side ports is embedded within the foam with the free end of the tubing exiting at one end. The foam contact layer may be lightly moistened with saline to improve comfort. A skin sealant may be applied to the periwound area and a hydrocolloid or sheet hydrogel dressing may be placed where the evacuation tube exits the foam to protect the underlying tissue. The film drape is then applied over the foam and wound with enough overlap in the periwound area to ensure an airtight seal.[33] The evacuation tube with a clamp is attached to an adjustable negative pressure pump and canister. The amount of negative pressure typically ranges from 50 to 175 mm Hg and may be set for continuous or intermittent vacuum.[34] Although various pressures are recommended based on wound etiology, the most common pressure is approximately 125 mm Hg because this pressure has been associated with maximum increase in blood flow. Lower pressures may be used for chronic wounds or over skin grafts and higher pressures may be used for acute wounds or wounds with large cavities.[35]

The beneficial effects of NPWT appear to result from increased blood and lymphatic flow; enhanced tissue perfusion, exudate management, release of growth factors, cell proliferation; and reduced edema. Contraindications include use on dry wounds, necrotic wounds, in body cavities, over malignancies, or in the presence of exposed blood vessels. Use is indicated for acute, chronic, and traumatic wounds; partial thickness burns; pressure ulcers; and neuropathic wounds.[36]

Holistic Treatment

Throughout this chapter, the primary emphasis has been on local wound care. However, the clinician must recognize and attend to global factors that may affect wound healing, such as nutrition of the patient, adherence of all staff to exercising excellent wound management procedures at all times, and the capabilities of the at-home caregivers. It is critical that the patient receive sufficient amounts of water, protein, fats, and carbohydrates to support wound healing. Maintenance of an appropriate fluid balance is essential for all cellular and systemic functions. Protein deficiency predisposes the patient to altered osmotic pressures, resulting in interstitial edema and restriction of blood flow, which may decrease the body's oxygen diffusion into the affected area. In the presence of inadequate protein and caloric intake, the nitrogen balance of the body is disturbed, resulting in the use of stored proteins for energy, leaving little for wound repair processes. Carbohydrates, particularly glucose, have a protein-sparing effect, allowing the body to use fats and carbohydrates for energy. Fats carry fat-soluble vitamins, help with thermoregulation, and, in the form of free fatty acids, compose important components of cell membranes vital to the synthesis of new cells.

Provision of a proper wound healing environment at the local and systemic level leads to effective and faster wound healing. Through proper wound cleansing, debridement, dressing management, and nutritional support, the body should have the elements necessary to provoke an appropriate inflammatory, proliferative, and maturation response that results in healed wounds within the typical time frames.

Conclusion

Rehabilitation patients frequently have wounds, including surgical wounds and wounds resulting from nonsurgical trauma. A good understanding of wound healing and the care of wounds is necessary to speed the rehabilitation process and identify abnormal wound healing. This chapter summarizes the wound healing phases and discusses factors that may delay the tissue restoration process. Modalities, dressings, and alternative treatments to speed the healing process are discussed. Further reading in this specialty can be found in these resources.[37-41]

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37 Physical Rehabilitation for the Critically Injured Veterinary Patient

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The critically injured veterinary patient can present a daunting challenge to the clinician trying to provide physical rehabilitation. Types of injuries sustained may range from single or multiple orthopedic injuries; soft tissue trauma to the chest, abdomen, or head; or a combination of these injuries. Burn injury or smoke inhalation can cause severe metabolic and cardiorespiratory dysfunction. The common thread in all of these patients is a debilitating injury that renders them partially or completely immobile for prolonged periods. Although bedrest is important to the recovery from critical injuries, prolonged inactivity can have deleterious effects on both injured and healthy body systems. The goals of providing physical rehabilitation to critically injured patients are to prevent complications stemming from injuries, prevent complications secondary to prolonged inactivity, maintain body condition, improve patient comfort, and hasten recovery. The purpose of this chapter is to discuss the effects of prolonged inactivity on traumatized and healthy body systems and to describe the application of basic physical rehabilitation techniques for specific critical injuries.

The Musculoskeletal System

Rarely do traumatized animals escape injury to the musculoskeletal system. Injuries can include single or multiple fractures of the appendicular or axial skeleton, hemorrhage into skin, muscle, and joint spaces, or injury to nerves supplying these structures. Patients with head, chest, or abdominal trauma may be unable to move because of decreased consciousness or pain. Disuse or injury to the musculoskeletal system may result in muscle weakness and muscle atrophy; skin, muscle, and joint contractures; and eventually may limit an animal's ability to move, groom, eat, and ambulate. With these global implications maintenance of this body system is paramount.

The balance between collagen synthesis and degradation is abnormal in immobile patients. Trauma with bleeding into soft tissue and muscle and subsequent inflammation and degeneration can trigger increased collagen synthesis. Increased collagen synthesis in an inactive patient leads to tighter packing of collagen fibers and contributes to the development of contractures.

Muscle contracture is shortening of the muscle owing to either intrinsic or extrinsic causes. Intrinsic changes are structural and can result from inflammatory or traumatic processes. Trauma, inflammation, ischemia, and hemorrhage in injured patients can restructure muscle tissue components and result in fibrosis and the development of adhesions. Following hemorrhage into a muscle, fibrin deposition occurs. Within 2 to 3 days fibrin is replaced with reticular fibers that form a loose connective tissue network. If the affected muscle is kept immobile, the network becomes more dense and resistant to stretch.[1]

Extrinsic muscle contracture is secondary to neurologic abnormalities or mechanical factors and is commonly associated with prolonged immobilization. Causes of extrinsic muscle contracture include paralytic or spastic muscle conditions. Paralyzed muscles cannot provide adequate resistance to opposing muscles across a joint. Eventually the antagonistic muscle will shorten. Carpal contracture in brachial plexus injuries illustrates this type of extrinsic contracture. To prevent contracture of the carpus in this situation, stretch must be applied to the normal muscle. Spastic muscle, which may exist in the pelvic limb musculature following trauma to the spinal cord between T3-L3, results from imbalance of muscle control. Increased muscle tone reduces the resting length of the spastic muscle and results in abnormal joint positioning. Treatment is aimed at stretching the abnormal muscle. Joint contracture is defined as an inability to move a joint through its full range of motion (ROM). Conditions that contribute to joint contracture include absence of mobility at the joint, immobilization of a joint in an inappropriate position, joint pain, paralysis, and muscle damage.

The position in which limbs are immobilized also contributes to the development of contractures. Muscle fibers lose up to 40% of the length of sarcomeres when immobilized in a shortened position. Therefore whenever possible joints should be kept in a neutral position to keep muscle fibers at equal length and tension to minimize contracture.[2] Additional factors that affect the rate of contracture development include the precipitating cause, duration and degree of immobilization, and preexisting joint restrictions. Edema, ischemia, and bleeding accelerate the development of muscle and joint contracture. Immobile, injured patients

Table 37-1 Treatment of Musculoskeletal Injuries

Technique	Benefits	Frequency
Cryotherapy	Reduces inflammation, swelling, and pain	Every 4-6 hr (after initial 24-72 hr)
Superficial or deep heating	Improves circulation, reduces muscle spasm, provides pain relief	Every 4-6 hr (after acute inflam. has subsided)
Massage	Increases circulation to paralyzed musculature, reduces/breaks down adhesions, provides pain relief, reduces edema	4-6 times/day
	Range-of-Motion (ROM) Exercises	
Passive	Maintains existing mobility of joints and soft tissue, minimizes contracture formation, prevents loss of ROM, maintains muscle elasticity, improves circulation, reduces edema	4-6 times/day
Active	Same as passive ROM exercise plus maintains physiologic muscle elasticity, maintains bone integrity, and may increase muscle strength	4-6 times/day
Splinting and casting	Corrects severe muscle and joint contracture by providing prolonged stretch to muscle and joint structures	

therefore require efforts directed at prevention and treatment of contracture development following soft tissue musculoskeletal injuries (Table 37-1).

Respiratory System

The respiratory complications of prolonged immobility and those resulting from primary injuries are numerous and potentially catastrophic in critically injured patients. Injuries to the head, chest, and abdomen often result in reduced respiratory function because of pain, inability to move, altered consciousness, or damage to thoracic structures. Impaired consciousness from head injury may lead to altered breathing patterns, diminished ability to cough, and inability to change body position. Rib fractures, pneumothorax, pulmonary contusions, and painful soft tissue injuries to the chest and abdomen may result in a restrictive breathing pattern characterized by shallow, rapid ventilation. This type of breathing pattern causes a reduction in tidal volume, functional residual capacity, and lung compliance. Altered respiratory patterns in conjunction with immobility may lead to atelectasis, accumulation of respiratory secretions, and pneumonia. It is important for the clinician to note any injuries that may compromise respiratory function and devise a physical rehabilitation plan to address those concerns. Chest physical rehabilitation is a term that describes the use of techniques aimed at improving lung volumes or facilitating the removal of airway secretions.[3] The goals of chest physical rehabilitation are to maintain bronchial hygiene, eliminate secretions from the airways, re-expand atelectatic lung segments, improve oxygenation, and reduce the incidence of pneumonia

Table 37-2 Goals of Chest Physical Rehabilitation

Technique	Goals
Positioning	Reduce risk of atelectasis, improve ventilation/perfusion, improve gas exchange, reduce risk of pressure sores, minimize muscle and joint stiffness
Cough	Remove secretions from trachea to fourth-generation bronchi
Postural drainage	Increase mobilization and elimination of secretions from upper airways
Percussion	Dislodge bronchial secretions
Vibration	Move secretions into larger airways

(Table 37-2). These goals can be achieved through techniques such as stimulating the animal to cough, frequent repositioning, postural drainage, percussion, vibration, and exercise. Each of these techniques is reviewed in detail.

Cough

Coughing is the most important defense mechanism to eliminate retained secretions. A cough can eliminate secretions from the trachea to the level of the fourth-generation segmental bronchi.[4] An effective cough is initiated by a deep inspiration, followed by closure of the glottis and contraction of the chest wall and abdominal musculature

to generate high intrathoracic pressure. After high intrathoracic pressure is attained, the glottis opens and is followed by rapid expulsion of air during exhalation. Critically injured patients may not be able to initiate a cough because of impaired consciousness, pain, weakness, or injuries to the chest or abdominal wall. These animals can be assisted to cough by applying gentle pressure to the trachea at the level of the third tracheal ring. Placing an animal in sternal recumbency may also improve an animal's ability to cough. Animals should be assisted to cough after postural drainage, percussion, and vibration and before changes are made in the patient's position.

Positioning

Injured animals are often unable to move or change position within their cage. Alternating right, sternal, and left lateral recumbency every 4 hours is recommended for immobile patients. Frequent repositioning helps prevent atelectasis and pooling of secretions in dependent lung segments. Because position influences the distribution of blood flow to the lungs, changing body positions alters ventilation/perfusion ($\dot{V}/\dot{Q}$) relationships within the lungs. When a patient is positioned with the compromised lung segment in a dependent position, that lung receives increased blood flow, resulting in increased $\dot{V}/\dot{Q}$ mismatch and impaired gas exchange.[5] Therefore if pulmonary contusions or pneumonia are present, positioning the patient with the good lung down improves oxygenation by limiting $\dot{V}/\dot{Q}$ mismatch and improving gas exchange. Improved oxygenation and a reduction in the number of patients with postoperative fever have been documented in humans when side-to-side turning is used.[6]

Frequent position changes in conjunction with comfortable bedding also help reduce the risk of pressure sores, edematous limbs, and muscle and joint stiffness. Repositioning is a simple physical rehabilitation technique that should be employed in all immobile patients.

Postural Drainage

Postural drainage is the use of changes in body position to allow gravity to aid in the removal of tracheobronchial secretions from different lung segments. This method of thoracic physical rehabilitation prevents pooling of respiratory secretions in the lungs, enhances drainage of the periphery of the lung, accelerates clearance of mucus, and increases the functional residual capacity of the lungs.[7] The patient must be positioned so that segmental bronchi are vertical to the affected lung to allow movement of secretions into the larger airways. After secretions reach the mainstem bronchi and trachea, a cough will aid in their removal. Postural drainage is most effective when used in conjunction with percussion and vibration to loosen thick airway secretions.

In human patients 11 postural drainage positions are used to drain 14 lung segments.[8] Figure 37-1 illustrates seven postural drainage positions that can be used to drain specific areas in the lungs of the canine patient.[9] The duration of postural drainage therapy depends on the number of lung segments requiring drainage, the amount of secretions produced, and the patient's tolerance for the procedure. In general 5 to 10 minutes per position is necessary for this therapy to be effective. Because exercise is superior to postural drainage for mobilization of respiratory secretions, ambulatory patients should be encouraged to walk for 5 to 10 minutes, three to four times a day.[7] Postural drainage is indicated in injured patients with pulmonary contusions, atelectasis, or altered consciousness, and those patients that are immobile secondary to injuries.

Percussion

Chest percussion (coupage) is performed by rhythmically striking the patient's chest wall with cupped hands over the affected lung segments (Figure 37-2). With this technique a mechanical shockwave is applied to the chest wall and transmitted to the lungs to dislodge secretions from bronchial walls. The goal of percussion in combination with vibration is to move secretions from small to larger airways where they can be cleared by a cough or postural drainage techniques. Percussion is performed throughout both inspiration and expiration. The amount of force used is determined by patient tolerance to the procedure and the patient's medical problem. Correct hand form and positioning over the affected lung segment are more important than the amount of force used. Only affected lung segments should be percussed because normal lung does not benefit from percussion. The rate of percussion should be steady and the therapist should proceed in a circular motion over the affected area.[10] Erratic percussion, that is, frequent stops and starts, should be avoided. Generally percussion should be sustained for 3 to 4 minutes followed by vibration. Three to four periods of percussion are generally adequate for each postural drainage position. Percussion is not recommended for those patients that produce little or no sputum with a cough[11] as well as patients with exacerbation of chronic obstructive pulmonary disease (COPD).[12]

Vibration

After each period of percussion, vibration (shaking of the chest wall) is performed to move the dislodged secretions toward larger airways (Figure 37-3). Unlike percussion, which is performed during both inspiration and exhalation, vibration is performed only during exhalation. The technique is performed with both hands placed on the same side of the chest over the affected lung segment. With arms straight and locked the therapist simultaneously contracts all of his or her muscles from the hands to the shoulder and vigorously shakes the chest wall. A mechanical vibrator also may be used. Vibration is performed on four to six consecutive breaths following each episode of percussion.

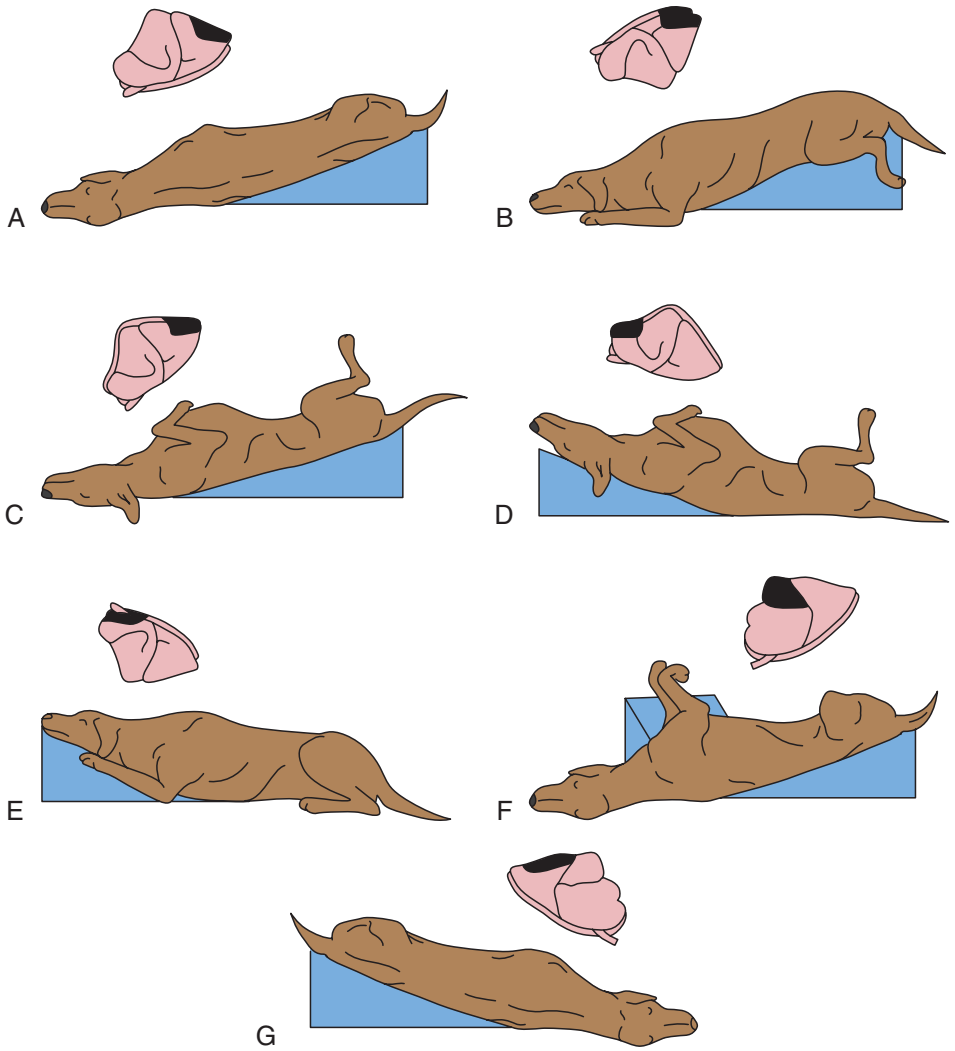


Figure 37-1 Positions for draining various portions of the lungs. **A,** Lateral segment of the left caudal lung lobe. The patient is in left lateral recumbency with the hind end elevated 40 degrees. **B,** Left and right caudal dorsal lung fields. The patient is in sternal recumbency with hind end elevated 40 degrees. **C,** Left and right caudal ventral lung fields. The patient is a dorsal recumbency with the hind end elevated 40 degrees. **D,** Left and right cranial ventral lung fields. The patient is in dorsal recumbency with the front end elevated 40 degrees. **E,** Left and right cranial dorsal lung fields. The patient is in sternal recumbency with the front end elevated 40 degrees. **F,** Right middle lung lobe. The patient is in dorsal recumbency. A pillow has been placed under the left side of the thorax so that the right side is higher than the left side. The hind end is elevated 40 degrees, and the front end is rotated one-quarter turn to the right. **G,** Lateral segment of the right caudal lung lobe. The patient is in left lateral recumbency with the hind end elevated 40 degrees. (Modified from Manning A, Ellis D, Rush J: Physical therapy for critically ill veterinary patients. I. Chest physical therapy. *Compend Contin Educ Pract Vet* 19:680, 1997.)

Any indication for postural drainage is an indication for percussion and vibration.

Contraindications for Postural Drainage, Percussion, and Vibration

Although most patients tolerate chest physical rehabilitation, critically injured patients may be at risk for complications. In human patients postural drainage has been associated with transient decreases in oxygen saturation of arterial blood (SaO_2), and percussion can potentiate cardiac arrhythmias.[11,13] Animals with pulmonary contusions often have some degree of hypoxia and should be supplemented with oxygen before and during these techniques. If oxygen desaturation occurs SaO_2 values generally return to pretreatment values within minutes of position changes and the conclusion of percussion. Animals with chest trauma are at risk for cardiac arrhythmias secondary to traumatic myocarditis. Chest percussion in these animals may precipitate an arrhythmia or cause an existing arrhythmia to worsen. Continuous electrocardiographic monitoring is recommended before and during chest physical rehabilitation.



Figure 37-2 Chest percussion is performed by striking the chest wall with cupped hands over the affected lung segments. The goal is to help break up secretions to larger airways so that they may be expelled by coughing or other means.



Figure 37-3 After percussion, vibration (shaking of the chest wall) is performed to move the dislodged secretions toward larger airways. Vibration is performed only during exhalation. Both hands are placed on the same side of the chest over the affected lung segment. With arms straight and locked, the therapist vigorously shakes the chest wall. Vibration is performed on four to six consecutive breaths following each episode of percussion.

Other injuries in which chest physical rehabilitation is contraindicated are listed in Box 37-1.

Exercise

Exercise is superior to chest physical rehabilitation in reducing atelectasis, mobilizing secretions from the lung periphery, and preventing pooling of secretions in the lungs. A systematic review on human patients suffering from COPD revealed moderate evidence of positive influences of a walking program and chest therapies (except percussion) on patients with COPD exacerbation.[14] Exercise enables deep breathing, which improves ventilation and expiratory flow. Exercise has also been shown to improve sympathetic activity, which increases ciliary beat frequency and decreases mucus viscosity.[15,16] In addition to the respiratory benefits, early mobilization reduces many of the musculoskeletal complications associated with prolonged immobility.

Box 37-1 Contraindications for Chest Physical Rehabilitation in Critically Injured Patients

- Flail chest or rib fractures
- Pneumothorax
- Platelet count <30,000 platelets/μL
- Areas with open wounds
- Subcutaneous emphysema of the neck or thorax
- Pain
- Unstable cardiovascular condition. Use caution with postural drainage techniques in patients with cranial trauma.

Ambulatory patients should be encouraged to stand and walk for short periods of time (5-10 minutes) every 4 to 6 hours. Weak but ambulatory animals can be supported with slings or harnesses. Nonambulatory patients should be assisted to stand for a few minutes every 4 to 6 hours in conjunction with changes in body position. Refer to chapter 29 for examples of exercises that may be used early postoperatively or for patients with extreme weakness and injuries.

Clinical Applications of Physical Rehabilitation for the Critically Injured Patient

Soft Tissue Trauma and Orthopedic Injuries

Trauma to soft tissue structures such as skin and muscle may be confined to a limited area or may be extensive. Extensive bruising, swelling, and pain may result from injury. Initial treatment should consist of immobilization, cryotherapy for 10 to 20 minutes every 4 to 6 hours, and comfortable bedding. If open wounds are evident, protective bandaging should be applied. Animals should be repositioned at least every 4 to 6 hours to prevent dependent edema from forming in the limbs and to prevent lung atelectasis. If the extremities are injured the involved limbs should be elevated above the level of the heart to promote drainage and reduce edema. Bandaging the limb can prevent or reduce edema by compressing lymphatics and blood vessels to limit fluid accumulation in the interstitium. Bandages of cast padding and reusable elastic bandage material should be applied beginning with the distalmost aspect of the extremity.[9] Bandages may need to be changed several times daily to allow inspection of the area and for physical rehabilitation.

After acute inflammation has resolved, warm compresses or warm water baths may be used. If the animal is capable, restricted exercise is encouraged beginning 48 hours after the initial insult. If spinal cord or orthopedic injuries prevent the patient from ambulation but permit movement of the limb, gentle passive ROM exercises

should be initiated. The limb and all joints should be cycled through their full ROM 10 times, four to six times daily. Range of motion exercises may be performed in conjunction with massage therapy. If fractures are present, ROM exercises should be withheld until appropriate fixation of the fracture. After surgical stabilization of fractures, cryotherapy should be applied to reduce inflammation, pain, and muscle spasms. Following resolution of the acute inflammatory period, application of warm compresses may be beneficial to help relieve pain and muscle spasms. Passive range of motion of the joints above and below the fracture site and massage therapy of the soft tissues are usually initiated 1 to 4 days after surgery if the patient permits.[17] Physical rehabilitation techniques to prevent contracture formation include encouraging ambulation, active and passive range of motion exercises, and stretching affected joints and muscles. Massage may be used to break down adhesions, reduce muscle spasm, and limit edema. If severe contractures have already formed, ROM exercises can be combined with serial casting or splinting. The use of casts or splints allows joints and muscles to be stretched for prolonged periods of time. Moderate contractures may be treated by splinting the area for 20 to 30 minutes, four times daily, in conjunction with ROM exercises and massage. Severe contractures may require placement of a cast or splint for 2 to 3 days. At the end of this period, the area is re-examined, and another cast or splint is placed as necessary to achieve further gains in motion. Splinting and bracing are described in detail in chapter 17.

Burn and Smoke Inhalation Injuries

Severe burn injury and smoke inhalation injury may occur as a result of thermal, radiation, chemical, or electrical injury. Burn injury in animals can lead to severe metabolic, cardiovascular, and pulmonary disorders. The burn classification system classifies burns by the amount of total body surface area (TBSA) involved and by increasing depth: superficial, superficial partial-thickness, deep partial-thickness, and full-thickness.[18,19] Although burn injuries have not been well described in the veterinary literature, rapid cooling of burns within 30 minutes of thermal injury seems to be beneficial in preventing tissue damage, reducing edema formation, and increasing the speed of re-epithelialization.[20] Current recommended treatment includes cooling the wounds with cold tap water (15° C [59° F]) for 20 minutes.[21] Ice-cold water must be avoided because of the risk of hypothermia.

Smoke inhalation injuries are not common in veterinary practice, but can occur. Smoke contains toxic substances that can cause, apart from respiratory disorders, neurologic signs in animals after inhalation. One of the most important considerations is carbon monoxide (CO), a product of combustion. The ability to bind to hemoglobin multiple times greater than oxygen, results in cellular hypoxia.[22] The use of hyperbaric oxygen therapy has been reported in some articles as controversial, because although hyperbaric oxygen reduces the half-life of carboxyhemoglobin in humans, there are conflicting reports of its efficiency of preventing late sequelae.[23]

Heat Stroke

Heat exhaustion and heat stroke are the most common heat-related diseases involving dysfunction or failure of temperature control in the body. They usually occur when the body is subjected to elevated ambient temperature and during strenuous exercise. When body hyperthermia lasts for a prolonged period of time, neurologic abnormalities such as hallucinations, seizures, and coma can occur, with the further effects of organ failure, disseminated intravascular coagulation, permanent brain damage, and death.[24]

The extent of damage to the body depends on the duration and level of hyperthermia. The primary treatment of an animal suffering from heat stroke is to reduce the body temperature immediately. Cooling methods include external and internal approaches. External methods use the convection and conduction abilities of cold, whereas internal methods are more invasive, including irrigation lavage or intravascular catheters with cool intravenous fluid administration. Some of the described external methods can be practical, such as immersion in a cold water bath or surface application of ice packs. White et al.[25] describe the use of evaporative cooling by spraying the dogs with tap water (15° C) (59°F) and compared the results with a peritoneal lavage group. The evaporative technique was as rapid as the lavage group.

Head Trauma

Patients that have sustained head trauma may have impaired consciousness and usually benefit from both chest and musculoskeletal physical rehabilitation. Depressed consciousness may cause abnormal breathing patterns such as hyperventilation, hypoventilation, irregular respiration, or a Cheyne-Stokes breathing pattern (apnea alternating with hyperpnea). Abnormal breathing patterns may lead to reduced tidal volume, functional residual capacity, and lung compliance, allowing respiratory secretions to accumulate. Comatose or stuporous animals are unable to mobilize secretions because of depressed cough and uncoordinated swallowing mechanisms. Atelectasis caused by immobility contributes to respiratory complications. Respiratory physical rehabilitation should be initiated in these patients as soon as they are stable. The therapist should assist the patient to cough and use postural drainage, percussion, and vibration techniques within 48 hours of presentation or as the animal's condition permits. Care should be taken when placing these patients in a head-down postural drainage position. If increased intracranial pressure or ongoing hemorrhage is suspected, head-down positions should be avoided.

Comatose or stuporous patients often exhibit abnormal trunk and limb posture. The limbs may be held in rigid

extension or flexion or may be limp. As soon as the patient's condition allows, musculoskeletal physical rehabilitation should be initiated to prevent muscle and joint contracture. A combination of frequent position changes, massage, and passive ROM exercises should be employed.

Pulmonary Contusions

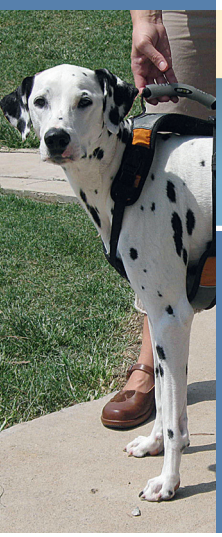
Chest physical rehabilitation is frequently used in human patients with pulmonary contusions. In patients with traumatic chest injuries, chest physical rehabilitation is believed to exert its greatest effect on the smaller airways rather than the larger airways.[26,27] Before initiating chest physical rehabilitation, chest radiographs should be taken and evaluated for the presence of pneumothorax, flail chest, or rib fractures that would preclude percussion and vibration. Chest radiographs should also be used to identify contused lung segments so that the therapist may choose the proper postural drainage positions and apply percussion and vibration over the appropriate area.

Conclusion

Rehabilitation should begin as soon as the critically ill patient is stable, and before the onset of complications associated with prolonged hospitalization and immobilization. A proactive approach to rehabilitation requires less time and effort and reaps greater rewards than one that is in response to developing problems. Most patients experience benefits from even simple rehabilitation therapies such as massage, thermotherapy, PROM, and controlled exercise regimens that involve a minimum amount of time on the owner's or therapist's part.

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SECTION VII

Business Considerations in Veterinary Rehabilitation

38 Development of a Rehabilitation Facility for Small Animals and Business Considerations

Jim Minick and Debbie Gross Saunders

Why Consider a Rehabilitation Enterprise?

The success of any business lies in its ability to take advantage of current trends and technologies to support its growth. Veterinarians today are looking for new ways to expand their practices, specialties, and income. Historically this has been accomplished through expansion of specialty areas (e.g., oncology, dentistry, ophthalmology). Most of these disciplines require specialized training and, in some cases, very expensive equipment. Rehabilitation for animals is similar to that currently being performed on humans. A person undergoing knee surgery would expect a regimented rehabilitation program for increased mobility and pain management. Of the more than 60,000 veterinarians currently practicing in the United States, only a small, but growing, percentage offer any form of rehabilitation for small animals.

Rehabilitation can be prescribed for:

- Presurgical and postsurgical procedures
- Recumbent patients, such as trauma or neurologic cases
- Increased mobility and strength in geriatric animals
- Pain management of injured and geriatric animals
- Conditioning canine athletes
- Improving quality of life
- Sports specific injuries
- Wound care and management
- Arthritis
- Weight management and control

Two scenarios exist for establishing a rehabilitation practice; a stand-alone facility or a facility associated with a veterinary practice. Both have advantages and disadvantages. A stand-alone rehabilitation facility offers the advantage of focusing on only rehabilitation with a team of veterinarians, physical therapists, technicians, and other support staff. A rehabilitation facility within a veterinary practice may be able to optimize benefits to patients by providing total patient care. Integration with a regular veterinary practice allows one to take advantage of economies of scale, especially when considering the purchase and use of expensive diagnostic equipment such as radiographic, ultrasound, and other imaging equipment. The ability to take a radiograph at a rehabilitation facility can be more efficient for the client if additional information is required for the therapist. In a stand-alone rehabilitation facility all pertinent information must be forwarded to the practitioner before the initial evaluation. Of course professionalism and sensitivity to professional relationships must be considered in a rehabilitation facility housed in a veterinary clinic.

Competence in the area of rehabilitation medicine is a prerequisite to provide legitimate service. Training is imperative for a successful rehabilitation practice. For individuals who do not choose to pursue training or when trained staffing is an issue, partnerships with other medical practices and staff may be used for the rehabilitation component.

Patient evaluation is the most critical component of the process because therapeutic protocols are established based on problems identified during the evaluation. As with humans, rehabilitation of animals involves numerous types and degrees of care. When the evaluation process is thorough, logical, and based on sound clinical fundamentals, positive results can be expected. By following a structured program the results may be further improved. The evaluation will determine the primary, secondary, and possibly tertiary problems with which the dog presents. After the problems are identified, the goals are established and the respective treatments are outlined. Specific considerations for each animal should be considered as well. For example aquatic therapy (Figures 38-1 and 38-2) may not be applicable to every dog for a variety of reasons.

Creating a Sound Business Strategy

When starting a business there are basic strategies that must be considered. No single individual can possess all the answers; therefore a well-planned business strategy must be developed with a common sense approach to success. Common sense is the knack of seeing things as they are, and doing things as they ought to be done.

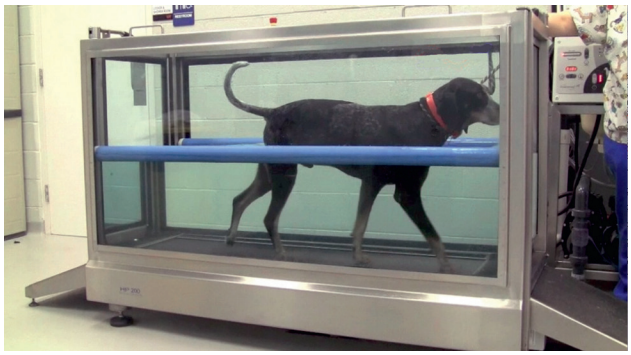


Figure 38-1 Underwater treadmill.



Figure 38-2 Rehabilitation pool.

The road map to a successful rehabilitation practice will result from sound business principles discussed throughout this chapter.

The basic rules for a successful practice may be summarized as follows:

- Know what you want.
- Find out what it takes to get it.
- Act on it and persevere.

As the process progresses, it cannot be stressed enough that the key to success in any worthwhile endeavor is to align yourself with the correct team. It is not difficult to pick the best and brightest individuals who can help you succeed. Canine rehabilitation is a relatively new field and it is important to examine clinics established in different areas of the country. In addition most cities have Small Business Administration (SBA) offices that can be wonderful resources for each of the points discussed in this chapter.

One of the initial steps in establishing a practice is determining whether a physical rehabilitation practice is viable in your community and your location. Some questions to consider include: (1) What is your referral source? (2) Is the location convenient for clients? (3) Are there other canine rehabilitation clinics within a 50-mile radius, and what services do they offer? (4) What are the economic demographics for your location? Even if a rehabilitation clinic is within an existing clinic, some portion of referrals will undoubtedly be from outside sources. Existing canine rehabilitation clinics within a 50-mile radius is not a negative, but it is advantageous to determine what services they offer and determine how you may compete.

Business Plan

After determining that a rehabilitation practice is something that can enhance a new or existing practice, a written plan must be drafted. Anyone who has considered a new business is always encouraged to have a well-defined written business plan. Until the plan is in writing you are not fully committed to the business entity.

If you already have your own practice you should be familiar with the need for and structure of a good business plan. If not there are many resources that may be consulted to help in this endeavor. Organizations such as the SBA can help to create the plan or, if preferred, numerous books and computer software are available to guide the process. Many existing canine rehabilitation centers have business plans and they may be able to offer advice and guidance.

Legal Entity

A logical plan should start with the choice of a legal entity such as a Corporation, Subchapter S Corporation, or Limited Liability Company (LLC). These entities can provide protection for you and your assets. The state in which you do business, the size of your company, and the potential tax benefits/burdens all influence the entity chosen. Discussions with a lawyer and accountant will help to determine the best entity for each situation. Doing Business As (DBA) may be set up initially under an LLC for a rehabilitation clinic in an existing facility. Advice from legal counsel should be pursued regarding this matter.

Accounting and Legal Support

Two of the most important people to have on any team are a competent business attorney and an accountant with tax experience that specializes in small to medium-sized service businesses. Accounting and legal expertise can save the average business owner thousands of dollars over a 5- to 10-year period. An accountant makes sure bills are paid in a timely fashion, supplies banks with information that is required, such as profits and losses, accounts receivable, and cash flows, and makes sure that a sound accounting system minimizes tax liability. A good attorney can reduce or minimize legal issues related to employees, employment agreements, partnerships, contracts and, to some extent, construction and equipment contracts. If there is an ongoing working relationship with specific individuals, these business associates will help you and your business. These services will be invaluable in making sound business recommendations. The most successful small businesses incorporate tax and legal advice from their inception, making them members of the team and keys to success.

Financing

Financing the venture may be accomplished in a variety of ways. Bank loans are the most obvious avenue to raise funds. Community banks are becoming more creative in their loan practices and may be flexible if there is a good credit history. Alternative sources of funding such as investment partners who provide equity and, in some cases additional business acumen, may present valuable insights. In addition the SBA provides a number of financial assistance programs for small businesses. Credit unions and insurance companies may also provide financing to diversify their portfolios.

Consider segmenting the money needed into startup costs, working capital, equipment financing, and so on. For example an accountant can help determine if a lease is best for acquiring various pieces of equipment needed for the practice. At all costs the temptation to use credit cards for financing, as many small business owners have done, should be avoided. If venture capital funding partners are used, be certain to have an attorney(s) review any repayment agreements. Examine the various costs of rehabilitation clinics and determine the needed equipment. Constructing a needs list and a wants list may be helpful in determining the best financial strategies.

Marketing and Advertising

As the practice and team expand, consider adding someone who can market the practice (whether it is yourself or someone experienced in dealing with customers). One of the most important and sometimes underused focuses for any successful business is marketing. Practically speaking if nobody in the practice has a marketing background, a professional should be sought to assist in marketing efforts. The day-to-day responsibilities of the practice, coupled with the responsibilities as a business owner or manager, leave little time to dabble in marketing. Moreover, with online marketing tools, such as blogs, Web pages, and social media like Twitter, Pinterest, and Facebook, a great deal of time may be required to simply update the electronic marketing presence of the business. In today's society, Facebook and Twitter are both social media sites that are a must for any small business to market their product.

Marketing does not have to be expensive to be done well. Creativity in industries related to animals is welcomed and appreciated. Your marketing strategy should include most, if not all, of the following elements:

- Brochures
- Web site
- Target marketing
- Print advertising
- Cable TV advertising
- Self-promotion through participation in
  - Seminars
  - Local conferences and pet expos
  - Local referral groups
  - Professional associations

Attributes of each of the elements should include:

- Brochures should be colorful, informative, and uncomplicated.
- Web sites should be innovative, easy to navigate, informative, customer friendly, and dynamic. A static (not often changed) Web page can be more harmful than helpful.
- Web blogs can draw countless people to your Web site. Social media like Twitter, Facebook, and Pinterest allow you to contact numerous people with short, logical updates. Any blog visited should provide the opportunity to comment, which should be used to drive traffic to the practice blog or Web site.
- Target marketing to the consumer helps to define the specialty area and narrows the focus of marketing to particular groups such as clubs that participate in agility, tracking, lure coursing, and so on.
- Print advertising can take many forms, including articles written about the facility and the services performed. This media is probably considered more "promotion" than advertising, but the fact that the facility appears in print is the important point for branding and self-promotion.
- Cable TV is effective and relatively low cost in most areas. Cable companies do a terrific job of targeting the message to the area you specify. The key is to create a commercial that is eye catching and memorable. A professional presentation that focuses on the practice's strengths draws attention to the services provided. Promotion of the practice will be assisted by creating a commercial that includes a tag line that is memorable. Repeatability is one of the keys to successful television advertising. Crafting your spot to make it adjustable offers much flexibility and gives the most efficient use of money. Placement of the commercial on specific stations is paramount and is based on demographic information for your area. This information should be thoroughly researched before signing a contract.
- Promotion through continuing education is easy and can cost relatively little. Hosting or participating in educational seminars, hosting an open house, volunteering to speak at dog club events about the benefits of rehabilitation, or participating in local pet expos is a great way to get the word out about your practice and promote the amazing things that rehabilitation can accomplish for a client's animals.
- Advertise your success stories on your Web site, social media, and clinic with many photographs, quotes from owners, and progressions. People love to hear success stories about animals, especially heartwarming stories.
- Word of mouth in the canine world should never be underestimated. Happy clients refer more clients. Each time an existing client refers a client, a personalized letter or card of gratitude should be sent.
- Marketing to referral sources will encompass all of the preceding points. In addition, meetings with referring

veterinarians and regular in-service training programs to the staff should be part of the marketing plan. Packets of brochures, referral pads, and other marketing materials such as magnets and pens should be distributed to potential referral sources. Computer software programs may be used to track referral sources and the number of referrals from each clinic. Communication with the referring veterinarians and clinics through evaluation letters, progress notes, and discharge summaries will enhance the relationship.

- Be certain to obtain written permission from the owner before posting any pictures of their animal or information about the patient or owner.

Suppliers

Competitive bidding from specific suppliers and manufacturers and/or distributors can be invaluable in reducing purchasing costs. The 80/20 rule can be applied when it comes to suppliers. This philosophy gives a favorite vendor the majority of product sales, but keeps him or her competitive because competitors are always trying to gain more market share. Common products sold in rehabilitation clinics include harnesses, slings, fitness equipment, supplements, and educational DVDs. There is also a market for prescription foods, especially those related to rehabilitation and weight loss.

Overhead and Burden

When evaluating the financial implementation of a rehabilitation center, overhead and burden must be considered. Overhead is the cost of doing business and is generally composed of rent/mortgage for the space, utilities, equipment, and staff costs. Overhead costs often overlooked in a rehabilitation setting include increased electricity, dehumidification, water costs for aquatic therapy, cleaning costs for the center, insurance coverage for the clinic inclusive of the aquatic therapy, liability coverage, and workers' compensation for staff members. Burden, specifically labor burden, refers to the benefits that are paid to employees. It is critical to have the correct personnel in place who are well trained and capable of providing outstanding customer service. Most clients of rehabilitation services are repeat customers because of the nature of ongoing care. In the case of geriatric animals, there is a long-term relationship, and clients appreciate the continued interface with the therapist. Trained personnel who have completed an educational rehabilitation program, such as veterinary technicians or physical therapist assistants, can command salaries that range from $35,000 to $50,000 annually, depending upon experience and skill sets. Physical therapists trained in canine rehabilitation may command salaries between $50,000 and $100,000 annually.

Billing rates for services provided to the customer can range from a low of $50 per hour to $75 or more per hour. The rates vary depending on the facility and location. Additional fees can be recovered through:

- Expertise of the DVM
- Evaluation for illness or other rehabilitation services
- Pool/treadmill/manual manipulation
- Specialized equipment, such as therapeutic laser
- Specialized treatments, such as acupuncture or chiropractic manipulation

Information Technology Systems

Information technology (IT) has become increasingly important in running an efficient business. It allows for the integration of many functions, eliminating the need to do or write things more than once. In all likelihood the investment in IT will represent a larger percentage of the budget than might originally be anticipated. Fortunately this new technology can ultimately help save time and money. New technologies are developed regularly for client billing, communication, documentation, correspondence, medical records, video conferencing, and so on. A myriad of practice management software programs can seamlessly integrate accounting and operational functions. For example it is possible to attach radiographs and other image files directly to client files where they can be viewed from any designated computer. Consultations with specialists can be efficiently and conveniently managed by sending files electronically to anyone in the world. The investment in equipment, which is expandable and secure, offers immeasurable returns by offering the business owner tools that can help operate the business and communicate with clientele. In fact, many programs allow client interaction via the Internet if desired.

Practice management software allows reminders to be sent to clients to increase compliance in a rehabilitation practice, appointment scheduling, patient information updates online, and client surveys based on specific services.

Roles and Integration of Staff Members in a Rehabilitation Facility

Most state practice acts stipulate that a licensed veterinarian must oversee and regulate the rehabilitation facility. Because rehabilitation naturally encompasses modalities related to physical therapy, it is not only important to understand the veterinary practice acts in your state, but additionally, an understanding of the physical therapy practice acts is critical to the successful administration of a rehabilitation practice.

The unique nature of animal rehabilitation has made it possible for physical therapists, initially trained to work with humans, to now work with animals. Unheard of only 20 years ago, it is now practical for both a veterinarian and physical therapist to work together to create a comprehensive program that addresses both the biologic and physiologic well-being of animals. Ideally the rehabilitation team should be led by a veterinarian who is responsible for the overall health of the animals, and a physical therapist who develops the therapeutic programs and administers treatment to the animals.

To be practical, the success of any business depends on the volume of service it is able to offer. To that end, veterinary technicians should be employed to help provide hands-on care for the animals in addition to regular client interaction and education. This interaction enables the client to be more proactive in the animal's home environment through education and training, and assists the animal's rehabilitation (e.g., at-home exercises), which provides continued therapy designed to further the results of in-house rehabilitation.

After a treatment regime has been determined the veterinary technician can perform many of the day-to-day treatments of the animal. Having a veterinary technician perform many of the treatments allows the veterinarian and physical therapist to perform other professional duties, including consulting on additional cases. Of course it is vital to have frequent communication among the veterinary technician, veterinarian, and physical therapist.

Furthermore veterinary assistants can be invaluable in helping veterinary technicians with some tasks, such as supervising routine treadmill sessions or drying patients after aquatic treatments. They can also assist with notations in the medical record during patient evaluations with the veterinarian or physical therapist. This promotes an efficient workflow, and perhaps more accurate recording of notes. Technology also currently exists that allows dictation of notes directly to the computer.

For a business owner it is natural to provide training to team members according to the needs of the practice. In most businesses this is practical and, indeed, necessary for processes to be performed correctly. Continuing education is also critical for employees to obtain new information and to incorporate new methods and practices. It is also beneficial from the standpoint of running a business to encourage and support personnel in their desire for advancement in the field. Therefore it is imperative for the owner of a successful rehabilitation facility to maintain a provision for continuing education of all employees on the profit & loss statement. Any technician who works directly with patients should receive training in veterinary rehabilitation. Specialized training not only provides personnel with needed information, but it will also be evidence to current and potential clients that the staff are properly trained and enthusiastic about providing the utmost quality of care.

Facility

The size of a rehabilitation facility is generally dictated by the services offered as well as the anticipated patient flow. Like an animal hospital the design of a rehabilitation facility must take into account the flow of patients (how they will move from room to room), the amount of time they are to spend at each station/room, and the number of patients receiving services at any given time. The floor plan for new office space should include an entrance and an exit so dogs have minimal interaction with one another. Half walls offer views of the facility but do not allow visual dog interactions. Successful rehabilitation practices ideally see multiple patients at the same time, and the space should allow this through half walls, private rooms, aquatic rooms, and exercise rooms. Modalities and manual therapies are best provided in a quiet section, and aquatics and therapeutic exercises are best provided in their individual sections. The flow of the patients must be considered for both check in and check out.

When evaluating the potential case load consider rehabilitation clients as well as weight loss and conditioning programs. Programs for conditioning and weight loss may consist of aquatic therapy and therapeutic exercise only. These programs may be very profitable; offering continued wellness programs after weight loss goals are accomplished and skilled rehabilitation is completed is another source of revenue. For example a dog that has undergone rehabilitation for a repaired cranial cruciate ligament may be discharged to a conditioning program to maintain the progress.

Facility design is a priority item in the business plan. Design, plan, and functionality determine costs associated with the relocation or construction of a new facility. After the building's structure is completed and the interior walls are set, it becomes extremely costly to make any changes or modifications. Therefore in-depth planning is critical for the success of the project. Before committing to construction plans visit or view photos of other rehabilitation practices to determine what works best for your practice.

Assessment of the Concept and Design

The first priority is to engage a licensed architect who has experience in the design and construction of animal hospitals. Always interview potential business vendors and suppliers and obtain references. Your investigation should include site visits when ever possible to view previously completed projects and discussions regarding the process, cost overruns, punctuality, and responsiveness of the architect and his or her subcontractors. Your questions should cover documentation, scheduling, change orders, materials, and systems used. When evaluating design criteria with the architect, be sure basic needs are captured in the design. Be proactive and clear in your demands for the features required and desired in the facility. It is important to keep in mind that many architects have not created a canine rehabilitation facility and will need to be educated on the dynamics of the facility regarding flooring, noise control, aquatics, office flow, humidification and maintenance.

The approximate space requirement for a rehabilitation practice is 500 to 2500 square feet, depending on the type and amount of equipment and potential client base. This does not include examination/assessment rooms (Figure 38-3) and kennel/run areas (Figure 38-4), which can easily add an additional 2000 square feet. If adding a rehabilitation facility to an existing medical facility, certain in-house areas, such as the examination and housing areas,



Figure 38-3 Patient assessment areas.



Figure 38-4 Kennels/runs.

are usually used in conjunction with the rehabilitation facility.

Proper flooring in the aquatic and exercise areas is paramount, as is noise control if kennel areas are planned. The walls surrounding the aquatic area must be considered with respect to water. The walls will get wet whether there is an underwater treadmill, a pool, or both. The walls should be comprised of a surface that will endure the constant splashing of water. Tiles, faux tiles, epoxy coating, or specialized paints are options. The surrounding flooring must withstand the water and be safe for the dogs, staff, and owners. HVAC requirements and humidification should also be a prime consideration in the design.

Kennel areas should be as comfortable as possible, and heating and cooling considerations are necessary. If the clinic will be performing long-term boarding for dogs, radiant heat flooring provides a good option for both heating and practicality. Sound-proofing the walls provides a more peaceful environment for the rehabilitation clinic and there are many options available. Ideally the kennel area should be easily accessible to the rehabilitation area, but also far enough away to offer a quiet area.

Architectural Design

Architectural design should encompass the following:

- A clean and simple flow for movement of patients and staff throughout the building. The flow of movement including check in area, treatments, and check out should be considered. Handicapped access and access for dogs in carts must be considered.
- A plan for maintenance and repair of equipment. Be sure that access points to essential components, such as heating, ventilation, and air conditioning (HVAC), electrical, and plumbing are not hidden behind walls or hard-to-move equipment. While in the design process, consider if you want the equipment to be seen by only staff members or the owners. Many designs allow the majority of the equipment to be placed out of view of the public, but allow easy access. For example, the holding tanks and the equipment associated with aquatic therapy are often loud and may need to be in an area away from patient care. A proper design allows easy access to the equipment, but is aesthetically pleasing to the clients and staff.
- Always specify materials that are both cost effective and readily available. Also incorporate materials that can easily be sanitized. Avoid expensive, one-of-a-kind materials that can be hard to replace.
- During the design phase it may be appropriate to engage an HVAC/air purification expert who can address air flow and potential odor issues throughout the facility. Dehumidification is essential in a clinic with aquatic therapy and should be addressed before the construction process.
- Most architects will make recommendations regarding specific manufacturers and products with which they have had experience. Usually this applies to plumbing, electrical, and HVAC components. It can save both time and money in the end if you do your own research with various manufacturers. Technology changes quickly



Figure 38-5 Reception/waiting area.

and animal hospitals built as little as 2 years ago have systems that may already be either outdated or not environmentally friendly.

- Flooring is a major component of the building process and may be a major expense. There are many flooring options available. Floor surrounding the aquatic area must be able to absorb or drain water, provide a nonslippery surface, and be easy to clean. Ergonomically it should possess a friendly surface for staff to stand on for prolonged periods of time. This type of flooring is often more expensive than other flooring. The reception area (Figure 38-5), private offices, staff areas, and other areas may not require expensive flooring. The flooring designs should be implemented before construction.
- New technology should not be avoided if the principles are sound and the equipment has some track record. An example is tankless hot water heaters. These new systems, although more expensive to purchase and install, have a more efficient operating cost along with better longevity and fewer space constraints. They also provide hot water on demand.
- Operating systems necessary for the rehabilitation center include a heating and cooling system.

City and Municipality Permits

Before planning an addition or building a new rehabilitation center, it is essential to begin the process with the legal entity controlling permits. Local regulations may or may not allow the addition of the center, especially with aquatics. A swimming pool is often the issue and may require specialized locks and gates for the center. Stand-alone facilities may face difficulties with individual town regulations because of the nature of the business. Canine rehabilitation does not fall into a specific category and many towns have a difficult time categorizing the clinic. Overnight boarding, noise control, aquatic management, waste, and physical waste management are often issues.

Box 38-1 Considerations for Evaluating Location

- Area needs for services to be provided
- Potential competitors in the area, specifically within a 5-mile radius of your new facility
- Type of facility/services you would provide. For example, a rehabilitation center with 24-hour boarding facility or one with more traditional hours of operation
- Staff needed to support the planned facility.
- Demographics of potential clientele in trade area
- Business growth potential in the area
- Ease of entering and exiting the facility. Consider traffic patterns, ability to make left-hand turns, future highway construction projects that may limit access, and so on.
- Space outside of the building for animal elimination purposes and areas clients may walk dogs, and space for gait analysis and potential for performance equipment such as jumps, obstacles, and space for running. Fencing requirements must be considered in accordance with the municipal ordinances.

Location of a New Facility

When building a new facility, location should be one of the highest priorities in the evaluation process (Box 38-1).

After the area has been established for the site, due diligence is required. This is usually accomplished in one of two ways.

1. Engaging a reputable and experienced real estate broker as your agent to procure a specific site that meets your needs
2. Searching the trade area looking for signs or checking in the local newspaper for available properties

After site selection and preparing a contract to purchase with the sellers, contingency clauses should always be inserted into the contract relative to financing and zoning approval.

Zoning and Contracting

Considered a minor issue by some, improper zoning of the location and the time it will take to make the necessary changes can add 2 to 6 months to the acquisition and building processes, and in some cases, changes in zoning regulations may be denied. The zoning process itself is usually a one-time process and can take two to three face-to-face meetings with city council members and sometimes local residents, who usually reside within 100 feet of the proposed site and want to voice their opinion. The process can cost $3000 to $10,000 for attorney fees and services to make changes.

After the purchase contract and zoning issues have been resolved the contractors and builders who will build the facility should be engaged. Individual budgets must be considered in this process. For smaller facilities general

Box 38-2 Design/Build Concept versus Traditional Concept

Design/Build

This concept uses a specific architect and a general contractor working together as a team, or an architectural firm that has a construction arm or affiliation.

Pros

- One source responsibility
- Changes to scope and design are done quickly. Very little interface with construction arm as it deals with change orders.
- Fewer subcontractors are generally involved in the bidding process
- Minimal documentation as it relates to bank draws
- Fewer people are involved in the whole process

Cons

- Can be more expensive than the traditional method because fewer subcontractors compete in the bidding process
- Relationships can become strained if problems develop during the construction cycle.
- Communication with all parties can be challenging and time consuming.
- Problems may arise if subcontractors need to be replaced for nonperformance issues.

Traditional Method (Use of a General Contractor)

This concept requires that an independent general contractor be engaged directly. After the architectural plans are completed and approved, a general contractor is engaged. A contractor who has experience in building animal hospitals is highly recommended. Be cautious in engaging a general contractor who has no experience building health care facilities and may not be familiar with critical codes and regulations.

Most general contractors solicit bids from at least two or more subcontractors for the many different components of the process, which include:

- Site preparation
- Foundation
- Structure (masonry, frame, etc.)
- Plumbing
- Electrical
- HVAC
- Roofing
- Interior framing/dry wall and finishing
- Other

Pros

- This can be more cost effective.
- The general contractor can solicit bids from numerous contractors to get the most competitive price.
- A diversified subcontractor base can lead to better design with cost-effective changes.
- There is good control of the project.
- If subcontractors must be replaced, changes generally can be made quickly.
- Architectural interface is usually minimal unless design issues arise.
- Day-to-day issues are acted on in a timelier manner.
- There is consistent oversight of subcontractors and the project.
- Changes can be made quickly.
- Disputes can be resolved quickly.
- Timelines are more often met.
- Bank draws are certified before submission to the title company.
- Documentation is addressed quickly.
- The scope of changes can be justified or denied quickly.
- The potential time and cost savings can be significant.

Cons

- There is a high reliability on subcontractors instead of the architect.
- There are multiple layers of communication.
- Conflicts may arise if there are problems.
- More extensive paperwork and lien waivers are required for draw payouts to subcontractors.
- Staging and sequencing can become a problem.
- The time element can be increased because of the complexity of construction.

contractors may be able to help with the design of the facility and may eliminate the need for an architect. There are two basic concepts that must be addressed to determine the best situation for your purposes (Box 38-2).

Regardless of which method is chosen management of the entire process can be handled effectively with the use of a third-party owner's representative that has an architectural or construction background. The role of this individual is to oversee the entire project on behalf of the owner. The third-party provider usually does daily on-site visits, reviews change-orders with the owner, and reviews all bank draws for accuracy. Issues that may arise are cleared through this third-party representative. The cost of this service can vary based on the amount of interface required, and the fee is traditionally approximately 2% to 5% of the construction price. The benefit is usually a 10% to 20% overall saving on the project, including the time factor of making on-site decisions. Many smaller facilities adding on to their existing facilities may find that general contractors familiar with veterinary clinics are often the most beneficial to making additions to the clinic, and usually have architectural support services available to them.

In short the construction process can be complicated and confusing. The ability to rely on construction professionals and the opportunity to have an individual or group who can act on your behalf helps to make the process easier. Finding those individuals can be tricky, but a little research on your part, concentrating on the experience, references, and review of past projects of the candidates, is the key in choosing those resources.

Rehabilitation Equipment

There are two basic ways to acquire rehabilitation equipment, which can be costly (e.g., whirlpools, underwater treadmills, therapeutic laser).

1. Purchase: Generally there are financing options through a bank or other source.
2. Lease/Lease with Option to Buy: This is common practice and a very good option for both tax purposes and acquisition of updatable technologies. This method allows the practice to take full advantage of tax credits and deductions along with the opportunity to exchange older technology with new, state-of-the-art equipment without having to sell or trade the old units.

No matter how well manufactured, all equipment needs maintenance and occasionally breaks down. When selecting vendors be certain to form an ongoing working relationship with companies that provide service of the equipment. Price is not always the most important factor in making the decisions. There is some truth in the saying, "You get what you pay for," but that does not mean that the highest-priced item should always be purchased either. The key is the relationship with vendors and the service they can provide should equipment need repair. A nonworking unit can cost a great deal in lost revenues when it cannot be used to provide rehabilitation services. When a call is made for service, will assistance be provided in a timely manner? Determine who will service your equipment, especially the aquatic units and where they are located. Some companies only offer one service provider for an entire region of the United States.



Figure 38-6 Treatment mat.

Box 38-3 List of Supplies and Equipment for a Model Rehabilitation Facility

General Patient Management

- Bandage material
- Booties, different sizes
- Harnesses, different sizes
- Slings, different sizes
- Clippers
- Leashes, with rehab facility logo
- Muzzles, different sizes
- Padded table
- Rolling lift system with supporting sling
- Rolling low gurney to transport weak/paralyzed patients
- Mats
- Goniometers
- Gulick II tape measures
- Hi Lo table
- Scale
- Force plate analysis—stance analyzer

Exercise Equipment

- Wobble board
- Quadruped balance board
- Cavaletti poles
- Vertical weave poles
- Land treadmill
- Limb weights
- Stairs/ramp
- Therapy balls/physio rolls, different sizes
- Thera-bands
- Mats
- Mirrors

Modalities

- Cryotherapy—cold packs, ice compression systems
- Hot packs
- Combo neuromuscular electrical stimulation/ultrasound unit
- Electrodes and ultrasound gel
- Extracorporeal shockwave
- Therapeutic laser

Aquatic Therapy

- Underwater treadmill—1 or 2
- Whirlpool (ideal but not necessary)
- Swimming pool (ideal but not necessary)
- Life jackets, different sizes
- Cage dryers
- Towels (consider towels with company logos)
- Harnesses for aquatic therapy
- Cleaning supplies specific to the aquatic systems
- Water treatment—chlorine, bromine, pH, etc.

Practice/Office Equipment

- Freezer/refrigerator
- Microwave
- Camera with video capability
- Computers/color printer
- Potential mobile computer devices for staff documentation, such as iPads
- Office furniture—desk, phone, chairs, file cabinets
- Telephone/facsimile system
- Desk and chairs
- Client seating/office furniture

Office Management

- Washer/dryer
- Wet vacuum
- Dry vacuum
- Cleaning supplies
- Mop bucket
- Tool kit
- First aid kit
- Trash cans

The total cost of equipment for a rehabilitation facility can range from $10,000 to $150,000 depending on what is required to provide the desired services. To choose the necessary items, consult with practices or organizations that currently offer rehabilitation services or distribution networks that supply components to similar businesses. A basic list of suggested items such as treatment mats (Figure 38-6) is provided in Box 38-3.

Strategic Alliances

Strategic alliances with manufacturers of equipment and suppliers can allow the practice to run more efficiently and offer an opportunity to try new medications, equipment, and so on. It is generally easier for manufacturers or suppliers to get their products into the market if they have a proven track record, so many are willing to allow a trial of new products (sometimes at a reduced cost) if a testimonial is offered. Most appreciate honest opinions regarding their products so that they can make improvements.

Customer Service

Who are the most important people in your organization? Veterinarians? Veterinary technicians? Physical therapists? The answer is yes, but without committed and caring customer service representatives there will be no long-term business. Customer service begins with the receptionist, who usually answers the phone, and the person who takes the client and his or her pet into the exam room. People do business with people they like! If a customer has a bad experience, how does he or she react? Nine out of ten simply do not return. How is a difficult situation handled? Most people do one of three things: (1) Nothing, (2) hope for the best, or (3) confront the situation head-on. The best response of course is the third choice. Most people appreciate honesty and the opportunity to discuss and hopefully rectify a situation. It may require a phone call or personal interface from the veterinarian or therapist. Personal contact is usually the best way to resolve an issue.

Canine physical rehabilitation allows a bond to be established between clients and practitioners that is not always witnessed or allowed from a time management point of view in other areas of traditional veterinary medicine.

How do we understand what customers want? Ask them. Customer surveys are a great way to receive feedback. Surveys should be concise, short, have no ambiguous questions, and should allow targeted and meaningful feedback to the practice. People are generally willing to answer a few questions, especially if they do not have to write a dissertation. Immediacy is also important. A call to the customer to inquire about the experience is an invaluable tool. Web surveys are also an inexpensive, data-rich tool that can be used to improve service. In fact, some software can be programmed to send the Web survey automatically after the patient's appointment. Most customers who have a good experience are happy to refer your services if they are asked. But customers who have a bad experience may take every opportunity to tell everyone they know about that experience, whether asked or not. Bad news generally spreads quickly while good news spreads slowly.

How is the staff trained?

- Take a course on the basics of customer service
- Evaluate and improve how things are done on a daily basis
- The do's and don'ts of customer interface
- Decision on attitude (a bad attitude in an organization can spread like wildfire.)

An often overlooked but important detail is appearance. As an example consider that you are about to board an aircraft and you look in the cockpit and see that the pilot is wearing a t-shirt with holes in it, and has uncombed hair and a 3-day beard. Most people would probably get off the aircraft.

Image! Today's world is very image conscious. Make sure that personnel are well-groomed and have a neat appearance. Shirts, polo shirts, tops, and sweaters with the company's logo are beneficial and provide a great image to clients.

Summary

In summary, four basic components must be considered when opening a rehabilitation practice:

- A good business plan
- The right team members
- Marketing and customer service
- Personnel who are properly trained and educated in the science and art of rehabilitation

As a veterinary practitioner one of the last things you may want to think about is the business of running your business. Most people would rather spend their time and efforts concentrating on veterinary medicine and rehabilitation rather than managing an office. With proper planning and a top notch staff you will be able to set up your practice and work with your team to make it successful.



APPENDIX 1

Protocol Development and Protocols

Andrea Henderson, David Levine, Darryl Millis, and Marti Drum

The sample protocols in this appendix are designed to address common conditions in patients not experiencing complications. Individual patient characteristics must be considered when designing an individual patient protocol. These are working protocols that should be modified according to clinical necessity, efficacy, and each patient's progress within the rehabilitation program. These protocols are not to be rigidly adhered to, but serve as guides to allow an effective rehabilitation plan of care. Furthermore, diagnosis and treatment should be provided by trained personnel.

During rehabilitation, particular attention should be paid to any deterioration in the patient's progress. Specifically, attention should be focused on lameness, stiffness, or pain following activity. If the patient appears to have stiffness or increased pain or lameness at any time after a therapy session, the level of activity should be decreased and a slower rate of progression should be instituted. It is very important that dogs be as comfortable and pain-free as possible during the rehabilitation period.

It is important to realize that the dog may not be totally normal even with intensive physical rehabilitation. The patient's condition may preclude return to normal function. However, rehabilitation is key in obtaining and maintaining as much function as possible. If the dog does not appear to be making progress, has any complications, or if there are any questions, the patient should be discussed with the clinician to determine an appropriate course of action.

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CCL Rupture—Extracapsular Repair

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Preoperative Goals: Prepare for surgery	From diagnosis to time of surgery	Therapeutic Exercises • Passive Range of Motion, 10-20 repetitions, SID-BID • Downhill Treadmill Walking—5-10% decline walking at a slow-to-medium walking speed, 5 min SID-TID • Brief training to aquatic therapy to ensure patient comfort and compliance Modalities • Transcutaneous electrical stimulation for pain relief (15-20 min SID) • NMES for muscle strengthening (15 min SID PRN) • Cryotherapy (15-20 min BID) Medications • Injectable polysulfated glycosaminoglycan agents (*discontinue within 24 hours of surgery*) • NSAIDs (*discontinue non-selective NSAIDs within 48 hours of surgery*)	Therapeutic Exercises • Rest in a confined space • Slow, controlled leash-walking only to go outside, no more than five minutes at a time Modalities • Cryotherapy (15-20 min BID) Medications • NSAIDs • Tramadol, Tylenol 3 • NMDA antagonists • Oral chondroprotective agents	• Baseline bilateral stifle ROM (and other joints if applicable)—goniometry • Baseline bilateral thigh circumference • Gait evaluation • Weight	Surgery should be performed as soon as possible after diagnosis to reduce the progression and severity of osteoarthritis as a sequela of motion on an unstable stifle.
Step 1 Postoperative to Early Toe-Touching at a Walk Goals: Pain control Swelling and edema control Improve stifle ROM Limit muscle atrophy	Immediate to 48-72 hours postoperatively	Therapeutic Exercises • PROM for stifle flexion/extension of operated limb (10-20 reps BID) • Slow leash walking only to go outside, no more than five minutes at a time Modalities • Gentle massage • Transcutaneous electrical stimulation for pain relief (15-20 min SID-TID) • NMES—quadriceps and biceps femoris muscle groups (15 min SID) • Cryotherapy (15-20 min BID-TID) • Therapeutic Laser SID Medications • Injectable polysulfated glycosaminoglycan agents • NSAIDs/other analgesics • Oral chondroprotective agents	Inpatient status preferred during this phase If home: Therapeutic Exercises • PROM for stifle flexion/extension of operated limb (10-20 reps BID) • Slow leash walking only to go outside, no more than five minutes at a time Modalities • Gentle massage • Cryotherapy (15-20 min BID-TID) Medications • Injectable polysulfated glycosaminoglycan agents • NSAIDs/other analgesics • Oral chondroprotective agents	• Postoperative bilateral stifle ROM (other joints if applicable)—goniometry • Postoperative bilateral thigh circumference • Response to activity and subjective pain level • Gait evaluation at a walk • Weight	• Consistent toe-touching on operated limb • Adequate resting analgesia • Decreased perioperative swelling and no incisional drainage • ROM 60-80 degrees flexion, 120-135 degrees extension

Step 2 Early Weight-Bearing Goals: Continue pain control Reduce swelling Work to return ROM to a more normal state Increase toe-touching weight-bearing	48-72 hours to two weeks postoperatively	Therapeutic Exercises • PROM and flexion/extension stifle stretches of operated limb (10-20 reps) • Slow leash walking only to go outside, no more than five minutes at a time • Slow ground treadmill walking • Gentle weight shifting at a stance and walk • Balance exercises on a soft foam pad or bi-directional balance board to encourage weight-bearing and joint position awareness Modalities • Heat therapy (10-15 min BID-TID, prior to exercises, NOT within 72 hours after surgery or if acute inflammation is present) • Therapeutic laser PRN • Cryotherapy (15-20 min BID) following exercises Medications • Injectable polysulfated glycosaminoglycan agents • NSAIDs/other analgesics PRN • Oral chondroprotective agents	Therapeutic Exercises • PROM and flexion/extension stifle stretches of operated limb (10-20 reps) • Slow leash walking only to go outside, no more than five minutes at a time BID-TID Modalities • Heat therapy (10-15 min BID-TID, prior to exercises, NOT within 72 hours after surgery or if acute inflammation is still present) • Cryotherapy (15-20 min BID) following exercises Medications • Injectable polysulfated glycosaminoglycans • NSAIDs/other analgesics PRN • Oral chondroprotective agents	• Response to activity and subjective pain level • Gait evaluation at walk and trot • Goniometry—Stifle ROM and other joints if applicable • Thigh circumference • Weight	• Consistent partial weight-bearing with each stride on operated limb • Minimal pain with light activities • Incisional healing without complications • ROM 60-80 degrees flexion, 120-135 degrees extension
Step 3 Consistent Weight-Bearing at a Walk Goals: Achieve normal ROM Improve weight-bearing at a walk and trot	Two to six weeks postoperatively	Therapeutic Exercises • PROM and flexion/extension stifle stretches of operated limb (10-20 reps BID-TID) if normal ROM not yet achieved • Slow consistent leash walking—add 5 min every week if progressing well BID-QID • 5 minutes of uphill inclines—*introduce at 3-4 weeks postoperatively* • Begin slow jogging 2-5 minutes daily (*begin at weeks 3-4 postoperatively*) • Aquatic therapy—Underwater Treadmill (UWTM) walking, begin at 5-10 min up to 15-30 min. (Ensure *incision is sealed before beginning aquatic therapy*) • Balance exercises on a 360-degree wobble board or moderately unstable surface 5-10 min	Weight loss program can be initiated at this phase if warranted (minimizes loss of lean tissue mass). Therapeutic Exercises • PROM and flexion/extension stifle stretches of operated limb (10-20 reps BID-TID) if normal ROM not yet achieved • Slow consistent leash walking—add 5 min every week if progressing well BID-QID • 5 minutes of uphill inclines *introduce at 3-4 weeks postoperatively*	• Response to activity and subjective pain level • Gait scale at walk and trot • Goniometry—Stifle ROM and other joints if applicable • Reassess thigh muscle girth at 2, 4, and 6 weeks postoperatively • Weight	• Consistent weight-bearing on operated limb at a trot • No evidence of pain with activities • ROM 40-60 degrees flexion, >150 degrees extension

Continued

CCL Rupture—Extracapsular Repair—cont'd

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
		• Sit-to-stand exercises—5-10 reps working up to 10-15 reps BID—*begin at 4 weeks postoperatively* • Stair climbing—1-2 reps up and down working up to 5-10 reps BID—*begin at 4-6 weeks postoperatively* • Aquatic therapy—Underwater Treadmill (UWTM) walking, begin at 5-10 min up to 15-30 min • Swimming 2-5 minutes two-three times per week—*begin at 4-6 weeks postoperatively* • Cavaletti Rails 5 reps × 6 rails to begin *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program* Modalities • Ultrasound therapy if loss of tissue extensibility—perform in conjunction with PROM and stretching if needed • Therapeutic laser PRN • Cryotherapy (15-20 min BID) following exercises if needed	• Begin slow jogging 2-5 minutes daily (*begin at weeks 3-4 postoperatively*) • Sit-to-stand exercises—5 reps worked up to 20 reps BID—*begin at 4 weeks postoperatively* • Stair climbing—1-2 reps up and down working up to 5—*begin at 4-6 weeks postoperatively* Medications • NSAIDs—PRN • Injectable polysulfated glycosaminoglycans • Chondroprotective agents		
Step 4 Consistent Weight-Bearing at a Trot Goals: Improve ability to use the limb with some speed	Six to twelve weeks postoperatively	Therapeutic Exercises • Trotting 5-10 min BID, increasing to 15-25 min TID, as long as pain and lameness do not worsen • Sit-to-stand exercises—15-20 reps BID • Aquatic therapy—brisk UWTM walking • Swimming several times per week • Cavaletti rails 15-20 reps BID, gradually raising rails	Continue weight loss program if needed Therapeutic Exercises • Trotting program, beginning at 5-10 min BID working up to 15-25 min TID • Uphill inclines jogging, beginning at 1-2 minutes and working up to 5-10 minutes per session	• Response to activity and subjective pain level • Gait evaluation at walk and trot	• Minimal to no lameness at a trot of normal speed • No evidence of pain with activities

Improve muscle mass and strength Improve stamina and endurance		• Ground treadmill walking with leg weights and/or resistance bands *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program* Modalities • Heat therapy (10-15 min BID-TID, if needed for stretching • Cryotherapy (15-20 min BID) following exercises if needed	• Controlled ball play at fast trot or slow lope (alternate days with trotting program) • Sit-to-stand exercises, 15-20 reps BID • Stair climbing—1-2 flights 2-5 reps as tolerated *Caution: Choose only 2-4 exercises per session. Doing all exercises may result in lameness and pain early in the program* Medications • NSAIDs—PRN • Injectable polysulfated glycosaminoglycans • Chondroprotective agents	• Reassess thigh muscle girth at 10-12 weeks postoperative • Weight	• Stifle range of motion within 5-10 degrees of normal limits (depending on pre-operative values)
<u>Step 5</u> Trotting at Speed with Minimal to No Lameness <u>Goals:</u> Continue improving speed at a lope and slow gallop Continue improving muscle mass, strength and power Continue improving stamina and endurance Return to as normal activity as possible	Twelve weeks postoperatively and beyond	Therapeutic Exercises • Trotting/running 10-20 min BID, as long as pain and lameness do not worsen. Some running up hills • Trotting up stairs 1-2 flights, 2 repetitions BID • Ground treadmill jogging with leg weights and/or resistance bands • Aquatic therapy—UWTM for conditioning, consider progressively increasing speed and use of jets when available • Swimming 10-15 minutes 3-4 times per week *Caution: Choose only 2-3 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program* Modalities • Heat therapy (10-15 min BID-TID, NOT within 72 hours after surgery) • Therapeutic laser PRN • Cryotherapy (15-20 min BID) following exercises if needed	Therapeutic Exercises • Running up and down hills • Ball playing at a gallop, 3-5 min working up to 15-20 minutes daily • Jogging for prolonged distances regularly throughout the week • Trotting up stairs—1-2 flights 2-3 reps as tolerated *Caution: Choose only 1-2 exercises per session. Doing all exercises may result in lameness and pain early in the program* Medications • Polysulfated glycosaminoglycans • NSAIDs—PRN • Chondroprotective agents	• Response to activity and subjective pain level • Gait scale at walk and trot • Weight	Progressions may be continued as long as lameness or pain with activity is not observed. If this occurs, decrease activity by 50% for 1 week and increase again by 10-20% weekly.

This protocol is for use only by licensed personnel who have been trained in canine rehabilitation.

*The therapist must keep in mind that these timelines are intended as guidelines and may be influenced by many factors affecting individualized patient care.

CCL Rupture—TPLO/TTA

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Preoperative	From diagnosis to time of surgery	Aquatic Therapy Exercises • Brief training to aquatic therapy to ensure patient comfort and compliance Medications • Injectable polysulfated glycosaminoglycan agents (*discontinue within 24 hours of surgery*) Modalities • Transcutaneous electrical stimulation for pain relief (15-20 min SID) • NMES for muscle strengthening (15 min SID PRN) • Cryotherapy (15-20 min BID)	Therapeutic Exercises • Rest in a confined space • Slow, controlled leash-walking only to go outside, no more than five minutes at a time Medications • NSAIDs (*discontinue within 48 hours of surgery*) • Oral chondroprotective agents Modalities • Cryotherapy (15-20 min BID)	• Baseline bilateral stifle ROM—goniometry; other joints if applicable • Baseline bilateral thigh circumference • Weight	Surgery should be performed as soon as possible after diagnosis to reduce the onset and severity of osteoarthritis as a sequela of motion on an unstable stifle.
Step 1 Non-Weight-bearing to Toe-Touching	Immediate to 48-72 hours postoperative	Therapeutic Exercises • PROM for stifle flexion/extension of operated limb (10 reps BID-TID or more if PROM is poor)** • Slow leash walking with sling support available, only to go outside (up to 5 minutes, 2-3 times per day) Medications • NSAIDs (*administration 30-60 minutes prior to first therapy session recommended*) • Opioids PRN Modalities • Mild pressure bandage (*first 24 hours postoperative*) • Gentle massage and thigh/stifle soft tissue mobilization • Transcutaneous electrical stimulation for pain relief (15-20 min SID-TID) • Cryotherapy (15-20 min BID)—*first session immediately postoperative* • Therapeutic laser therapy SID	Inpatient status during this phase	• Postoperative bilateral stifle ROM—goniometry; other joints if applicable • Postoperative bilateral thigh circumference • Response to activity and subjective pain level • Lameness score at a stance and walk • Weight	• Early toe-touching • Adequate resting analgesia • Decreased perioperative swelling and lack of incisional drainage • ROM 60-80 degrees flexion, 120-135 degrees extension

Step 2 Early Weight-Bearing	72 hours to three weeks postoperative	Therapeutic Exercises • PROM and flexion/extension stifle stretches of operated limb (10-15 reps BID-TID) until near normal stifle ROM achieved • Slow, controlled leash walking, 5 minutes working slowly up to 20 minutes at a time TID • Balance exercises on a soft foam pad or bi-directional balance board for weight-bearing • Aquatic therapy—Underwater Treadmill (UWTM) walking 5-10 min SID-BID (*do not begin until incision is sealed, approximately 1-2 weeks postoperative*) Modalities • Heat therapy before activity (10-15 min BID-TID, NOT within 72 hours after surgery or if CS of acute inflammation are still present) • Therapeutic laser therapy PRN • Cryotherapy (15-20 min BID) following exercises	Therapeutic Exercises • PROM and flexion/extension stifle stretches of operated limb (10-15 reps) • Slow, controlled leash walking, 5 minutes working slowly up to 20 minutes at a time TID Medications • NSAIDs—PRN • Wean off Tramadol, NMDAs • Oral chondroprotective agents Modalities • Heat therapy (10-15 min BID-TID, prior to exercises, NOT within 72 hours after surgery or if CS of acute inflammation are still present) • Cryotherapy (15-20 min BID) following exercises	• Goniometry—Stifle ROM and other joints if applicable • Reassess thigh muscle girth at 3 weeks postoperative Response to activity and subjective pain level • Lameness score at a stance and walk • Weight	• Consistent partial weight-bearing on operated limb on all strides at a walk • Minimal pain with light activities • Incisional healing without complications • ROM 50-60 degrees flexion, >150 degrees extension
Step 3 Consistent Weight-Bearing	Three to six weeks postoperative	Therapeutic Exercises • PROM and flexion/extension stifle stretches of operated limb (10-20 reps BID-TID) if normal ROM not yet achieved • Balance exercises on a 360-degree wobble board or moderately unstable surface 5-10 min • Sit-to-stand exercises—5 working up to 10-15 reps BID-TID—operated side against a wall • Aquatic therapy—Underwater Treadmill (UWTM) walking working from 5-10 min up to 15-30 min SID-BID • Low Cavaletti Rails 5 reps × 6 rails to begin	Weight loss program can be initiated at this phase if warranted (minimizes loss of lean tissue mass). Therapeutic Exercises • Slow consistent leash walking—working up to 20-30 minutes BID-TID if progressing well • 5-10 minutes of uphill inclines introduced at 4-5 weeks postoperative • Sit-to-stand exercises—5 working up to 10-15 reps BID-TID—operated side against a wall	• Response to activity and subjective pain level • Gait scale at walk • Goniometry—Stifle ROM and other joints if applicable • Weight	• Consistent improvement in weight-bearing to no more than subtle lameness on operated limb at a walk • No evidence of pain with activities • ROM 40-60 degrees flexion, >150 degrees extension

Continued

CCL Rupture—TPLO/TTA—cont'd

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
		Modalities • Ultrasound therapy if loss of tissue extensibility—perform in conjunction with PROM and stretching if needed • Therapeutic laser therapy PRN • Cryotherapy (15-20 min BID) following exercises if needed	• Walking in circles to left and right or Figure 8s to improve proprioception (*beginning with wider and working towards narrower turns*) Medications • NSAIDs—PRN • Chondroprotective agents		
Step 4 Improved Weight-Bearing at a Trot	Six to eight weeks postoperative	Therapeutic Exercises • *Radiographs to demonstrate healing of osteotomy site are required to proceed.* • Sit-to-stand exercises—20-30 reps BID • Aquatic therapy—UWTM walking • Swimming several times per week, 5 up to 20 min per session (*gentle water entry/exit required*) • Cavaletti rails 10-20 reps BID, gradually reducing distance between rails • Stair climbing: 5-10 stairs up and down, BID-TID • Controlled tug-of-war, 5 min per session to tolerance SID Modalities • Heat therapy (10-15 min BID-TID) prior to exercises if needed • Cryotherapy (15-20 min BID) following exercises if needed	Therapeutic Exercises • *Radiographs to demonstrate healing of osteotomy site are required to proceed.* • Prolonged leash walks, 30-40 min BID-TID, longer leash allowed • Controlled on-leash trotting, beginning at 5 working up to 10 min BID, in a straight line • Trotting in Figure 8s, no sharp turns • Sit-to-stand exercises, 20-30 reps BID • Stair climbing—5-10 stairs up and down, BID-TID • Dancing on rear limbs 30 sec-1 min per rep, BID-TID • Controlled tug-of-war, 5 min per session to tolerance SID Medications • NSAIDs—PRN • Chondroprotective agents	• Response to activity and subjective pain level • Gait scale at walk and trot • Reassess thigh muscle girth at 7-8 weeks postoperative • Weight	• Consistent weight-bearing at a trot • No evidence of pain with activities

Step 5 Consistent Weight-Bearing at a Trot	Nine weeks post-operative and beyond	Therapeutic Exercises • Aquatic therapy—UWTM for conditioning, consider progressively increasing speed and use of jets when available • Swimming 15-20 minutes 3-4 times per week • Weights on affected limb (begin at 0.5-1 lb depending on dog size) or therabands for preferential strengthening • Playing ball • Jogging up hills and stairs • Additional therapeutic exercises as indicated for strength and speed Modalities • Cryotherapy (15-20 min BID) following exercises if needed	Therapeutic Exercises • Program may continue as before with the below additions • Running in a straight line • Trotting in Figure 8s and zigzag patterns • Weights on affected limb (begin at 0.5-1 lb depending on dog size) for preferential strengthening • Stair climbing—1-2 flights 1-2 reps as tolerated Medications • NSAIDs—PRN • Chondroprotective agents	• Response to activity and subjective pain level • Gait scale at walk and trot, eventually at faster gaits • Reassessment of thigh girth and joint goniometry as applicable • Weight	Progressions may be continued as long as lameness or pain with activity is not observed. If this occurs, decrease activity by 50% for 1 week and increase again by 10-20% weekly. Goals are based on multifactorial considerations, including age, presence/degree of osteoarthritis, and musculoskeletal status of other limbs.

*This protocol is for use by licensed personnel who have been trained in canine rehabilitation. The therapist must keep in mind that these timelines are intended as guidelines and may be influenced by many factors affecting individualized patient care.

**Loss of extension range of motion is more common than flexion after a tibial plateau leveling osteotomy.

Cervical Spondylomyelopathy (postoperative)

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Rest Goals: Pain control Maintenance of joint ROM Maintain incisional health Weight-bearing in standing with moderate support Minimize limb muscle atrophy Independent urination and defecation	Immediate to five to seven days postoperatively	Therapeutic Exercises • Assisted standing with physioball abdominal support on a stable, high-traction surface (*beginning 48-72 hours postoperatively*) • PROM with bicycling movements all joints of all four limbs (10-15 reps BID-TID). • Flexor reflex stimulation exercises (8-10 reps BID-TID for each limb) Modalities • TENS, premodulated or interferential electrical stimulation across incision site, 15-20 min SID • NMES to scapular and/or thigh muscles 15 min SID • Cryotherapy at cervical incision site, 20 min BID-TID (*first 48-72 hours after surgery*) • Heat therapy at cervical incision site, 10 min BID-TID (*after 72 hours postoperatively*) • Gentle massage of shoulder and limb musculature +/– tapotement BID as tolerated • Therapeutic laser therapy of peri-surgical cervical and shoulder musculature SID PRN	Therapeutic Exercises • Assisted standing with thoracic and abdominal support as needed on a stable, high-traction surface (*beginning 48-72 hours postoperatively*) • PROM with bicycling movements all joints of all four limbs (10-15 reps BID-TID) Modalities • Cryotherapy at cervical incision site, 20 min BID-TID (*first 48-72 hours after surgery*) • Heat therapy at cervical incision site, 10 min BID-TID (*after 72 hours postoperatively*) • Effleurage BID-TID Medications • NSAIDs (if corticosteroids have not been used)/opioids • NMDA receptor antagonists • Muscle relaxants BID-TID PRN Other • Bladder expression TID-QID PRN • Use of soft bedding with rotation every 4-6 hours to prevent decubital ulcers	• Subjective pain level • Limb muscle tone • Degree of support required in standing • Limb muscle circumference (baseline) • Subjective scapular muscle mass	• Postoperative comfort • Incisional integrity maintained • Standing in weight-bearing with moderate support

		Other • Bladder expression TID-QID PRN • Use of soft bedding with rotation every 4-6 hours to prevent decubital ulcers	*Note: A home exercise program may be impractical for many owners early in the postoperative period, depending on patient size and function*		
Partial Weight Support to Initial Motor Function <u>Goals:</u> Continued pain control Weight-bearing in standing with minor support Minimize muscle atrophy Voluntary movement of all four limbs, with supported early walking Self-righting into a sternal position	One to two weeks postoperatively	Therapeutic Exercises • Assisted standing and weight-shifting/rhythmic stabilization, gradually decreasing support (eg physioball beneath abdomen, standing in water) • PROM with bicycling movements all joints of all limbs (10-15 reps BID-TID)—during supported standing if feasible • Early walking with sling support (patient capability will vary) short distances, eg 1-2 yoga mat lengths up to 2-3 minutes Modalities • Gentle massage of limb musculature BID-TID • TENS, premodulated or interferential electrical stimulation across incision site, 15-20 min PRN • NMES to hindlimb and forelimb musculature as needed for contraction 15 min EOD-SID • Heat therapy at cervical incision site, 10 min BID-TID (*after 72 hours postoperatively*) Other • Bladder expression TID-QID PRN • Use of soft bedding with rotation every 4-6 hours to prevent decubital ulcers	Therapeutic Exercises • Assisted standing and weight-shifting/rhythmic stabilization, gradually decreasing support (eg physioball beneath abdomen) • PROM with bicycling movements all joints of all limbs (10-15 reps BID-TID) • Flexor reflex stimulation (8-10 reps BID-TID for each limb) Modalities • Gentle massage of hindlimb musculature BID-TID • Heat therapy at cervical incision site, 10 min BID-TID Medications • NSAIDs (if corticosteroids have not been used)/opioids • NMDA receptor antagonists Other • Bladder expression TID-QID PRN • Use of soft bedding with rotation every 4-6 hours to prevent decubital ulcers	• Response to activity and subjective pain level • Degree of support required in standing • Supported walking endurance (seconds to minutes) • Degree of appropriate gait patterning in motion • Limb muscle circumference/subjective scapular muscle mass • Degree of comfortable active cervical ROM	• Voluntary motor activity in all four limbs • Early stepping activity all four limbs (supported walking) for short distances • Incisional healing • Minimal pain with light activities • Standing with minor support

Continued

Cervical Spondylomyelopathy (postoperative)—cont'd

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Initial Motor Function to Good Motor Function with Proprioceptive Deficits Goals: Improve proprioception during walking activity Maintain normal limb ROM Increase limb muscle mass Maintenance of balance during turns or on a moderately unstable surface when standing	Two to four weeks postoperatively	Therapeutic Exercises • Supported standing and weight-shifting on a bi-directional balance board • Standing PROM with bicycling movements all joints of all limbs (10-15 reps PRN based on ROM) • Aquatic therapy: Swimming 5-10 minutes with supported standing during frequent breaks SID. *Cervical patients should wear lifejacket and be accompanied by assisting personnel in the pool at early stages of use* • Aquatic therapy: Underwater Treadmill (UWTM) standing (up to 10 minutes) to slow walking (up to 5 minutes with breaks) SID. *Cervical patients should wear lifejacket and be accompanied by assisting personnel in the UWTM at early stages of use* • Turning exercises during standing with positive reinforcement held at various locations (BID-TID, concentrate efforts on the weaker side)—caution with cervical ROM • Sit-to-stand exercises while sling-supported with hindquarters sitting on an elevated surface *Caution: Choose only 2-3 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program* Modalities • NMES to hindlimb musculature for contraction 15 min PRN • Cryotherapy at cervical incision site, 20 min PRN after exercise	Therapeutic Exercises • Supported standing and weight-shifting on a moderately unstable surface • Standing PROM with bicycling movements all joints of all limbs (10-15 reps PRN based on ROM) • Slow leash walking with sling and forelimb harness support 5-10 minutes BID-TID • Turning exercises during standing with positive reinforcement held at various locations (concentrate efforts on the weaker side) Modalities • Cryotherapy at cervical incision site, 15-20 min PRN after exercise Medications • NSAIDs PRN • NMDA receptor antagonists	• Response to activity and subjective pain level • Length of time with minimally supported/ unsupported standing • Length of time walking in aquatic therapy and on dry land • Frequency of steps in which appropriate proprioception is used • Limb muscle circumference/ subjective scapular muscle mass • Degree of comfortable active cervical ROM	• Consistent gait patterning of all limbs at a slow walk • Minimal pain with moderate activities • Walking endurance of 5-10 minutes with breaks • Early appropriate proprioceptive limb placement

Good motor function with proprioceptive deficits to near normal gait Goals: Consistent proprioception with slow-moderate activity Limb muscle mass approaching normal Comfortable cervical AROM approaching normal Maintenance of balance during turns and negotiating obstacles while walking	Four to eight weeks postoperatively	Therapeutic Exercises • Assisted standing and weight-shifting on a 360-degree balance board • Aquatic therapy: Swimming 10-15 minutes with supported standing during frequent breaks • Aquatic therapy: Underwater Treadmill (UWTM) walking, 5-10 minutes SID with lifejacket or harness • Gait patterning on a ground treadmill, 5-10 minutes with resistance band use PRN to encourage proprioceptive placing • Turning exercises when walking: weave poles gradually reduced in distance 5-10 reps SID-BID • Low cavaletti rails 5-10 reps SID-BID • Sit-to-stand exercises, rising from an elevated surface 10-15 reps SID-BID *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program*	Therapeutic Exercises • Assisted standing and weight-shifting on an unstable surface • Walking in circles and figure-eights 3-5 minutes BID-TID • Sit-to-stand exercises, rising from an elevated surface 5-10 reps SID-BID • Sling-supported leash walking 10-15 min BID-TID with 3-5 minutes gentle inclines/declines • Turning/stretching exercises during standing with positive reinforcement held at various locations (concentrate efforts on the weaker side) Medications • NSAIDs PRN • NMDA receptor antagonists	• Response to activity and subjective pain level • Length of time with minimally supported/unsupported standing • Walking endurance • Frequency of steps in which appropriate proprioception is used • Limb muscle circumference/subjective scapular muscle mass • Height of Cavaletti rails navigated • Tightness of turns successfully executed • Cervical AROM	• Consistent pelvic limb proprioception at a walk • Minimal to no pain with moderate controlled activities • Normal gait patterning at a walk • Near-normal active cervical ROM

Continued

Cervical Spondylomyelopathy (postoperative)—cont'd

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Near Normal Gait to Normal Gait at a Walk Goals: Elimination of proprioceptive deficits during controlled walking and trotting Normal limb muscle mass Normal cervical AROM/PROM Maintenance of balance during turns and negotiating obstacles while trotting	Eight or more weeks postoperatively	Therapeutic Exercises • Standing and weight-shifting on a 360-degree balance board • Aquatic therapy: Swimming 15-20 minutes SID • Aquatic therapy: Underwater Treadmill (UWTM) walking, 15-20 minutes working up to variable speeds • Gait patterning on a ground treadmill, 5-10 minutes walking working up to a trot • Turning exercises with weave poles 10-20 reps SID-BID at a walk working up to a trot • Higher Cavaletti rails 10-20 reps SID-BID • Stair Climbing beginning with 1-3 flight repetitions SID-BID • Sit-to-stand exercises 10-20 reps SID-BID *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Gradual increase to normal activity can be initiated as early as 8 weeks postoperatively, but many patients may take significantly longer to reach the goals listed at this stage.*	Therapeutic Exercises • Standing and weight-shifting on an unstable surface • Turning exercises with weave poles 10-20 reps SID-BID at a walk working up to a trot • Stair Climbing 1-3 flight repetitions SID-BID • Sit-to-stand exercises 10-20 reps SID-BID • Controlled ball play in a confined space • Leash walking 15-20 minutes BID with 5-8 minutes mild to moderate inclines and 3-5 minutes controlled trotting (working up to longer periods)	• Response to activity and subjective pain level • Walking and trotting endurance • Tightness of turns in which balance is maintained at a walk and trot • Height and distance of Cavaletti rails navigated • Limb muscle circumference/ muscle mass • Proprioceptive placing at a controlled trot	• Minimal to no pain with moderate-to-extensive controlled activities • Normal gait and proprioception at a controlled trot • Normal comfortable cervical AROM/ PROM

This protocol is for use by licensed personnel who have been trained in canine rehabilitation.

*The therapist must keep in mind that these timelines are intended as guidelines. Rate of recovery will vary widely among patients for reasons that include status of nociception prior to surgery.

Degenerative Myelopathy**

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Early Stage: Pelvic limb paraparesis and ataxia Goals: Minimize loss of muscle strength/ endurance Maintain quality of life Prolong survival time	Variable	Therapeutic Exercises • Assisted standing and weight-shifting on a 360-degree balance board 5-10 min BID • Aquatic therapy: Underwater Treadmill (UWTM) walking, 5-10 working up to 20-30 min at variable speeds SID-BID • Swimming 10 working up to 30 minutes with breaks • Sit-to-stand exercises 10-15 reps BID • Cavaletti Rails (walking or trotting as able) 10-15 reps BID • Weave Poles (walking or trotting as able) 10-15 reps BID • Walking up and down stairs 2-4 flights BID-TID • Land treadmill walking/jogging as capable 5-10 min SID-BID. Add incline to challenge pelvic limbs or decline to facilitate appropriate movement of pelvic limbs *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in over-exertion and pain early in the program* Modalities • Gentle massage of limb and paraspinal musculature SID-BID prior to or following exercise • NMES Quadriceps and Hamstrings 15 min SID-EOD Other • Consider use of paw protective boots as proprioceptive deficits develop	Therapeutic Exercises • Hemi-standing or diagonal leg two-legged standing with weight-shifting 5-10 min divided into 1-2 min reps BID-TID • Leash walks at routinely performed pace/distance, incorporating inclines and 5 minutes of trotting as able BID-TID • Controlled ball play • Sit-to-stand exercises 10-15 reps BID • Walking/trotting as capable across an unstable surface such as a foam mattress 10-15 laps SID-BID • Walking up and down stairs 4-6 flights BID-TID • Walking/trotting as capable through sand or tall grass 5 min SID-BID *Caution: Choose only 2-4 exercises per session. Doing all exercises may result in over-exertion and pain early in the program* Modalities • Effleurage BID-TID Other • Consider use of paw protective boots as proprioceptive deficits develop	• Response to activity and subjective pain/ exertion level • Walking and trotting endurance on dry land • Walking endurance in underwater treadmill • Frequency of steps in which appropriate proprioception is used (if deficits are present) • Height of Cavaletti rails navigated • Thigh circumference/ muscle mass	• Inconsistent pelvic limb proprioception at a walk • Moderate paraparesis/ ataxia at a walk • Severe paraparesis/ ataxia at a trot • Moderate-severe paraparesis/ ataxia when turning

Continued

Degenerative Myelopathy—cont'd**

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Early-Mid-Stage: Moderate to severe pelvic limb paraparesis and ataxia Goals: Delay loss of muscle strength/ endurance Provide continued proprioceptive feedback Maintain quality of life Prolong survival time	Variable	Therapeutic Exercises • PROM with bicycling movements all joints of pelvic limbs (10-15 reps BID-TID) • Flexor reflex exercises for pelvic limbs, 10 reps BID • Assisted standing and weight-shifting on a foam pad or bi-directional balance board with assisted pelvic limb support as needed • Aquatic therapy: UWTM walking, 5-15 min at a steady pace SID-BID • Swimming 10-15 minutes with breaks 3-4 times per week • Sit-to-stand exercises with sling assistance if needed 10-15 reps BID • Low Cavaletti Rails 5-10 laps as able BID • Weave Poles (walking as able) 10-15 reps BID • Land treadmill slow walking as capable 5-10 min SID-BID. Add decline to reduce weight-bearing forces on pelvic limbs, +/− resistance band to help encourage proprioceptive placing *Caution: Choose only 2-3 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in over-exertion and pain early in the program*	*Fitting for a four-wheel cart if desired can take place at this time* Therapeutic Exercises • PROM with bicycling movements all joints of pelvic limbs (10-15 reps BID-TID) • Flexor reflex exercises for pelvic limbs, 10 reps BID • Balance and weight-shifting on a moderately unstable surface with pelvic limb support as needed; 5-10 min divided into 1-2 min reps BID-TID • Walks (with four-wheel cart when prescribed) at routinely performed pace/distance on level ground, 10-15 min BID-TID • Walks in front and rear assistive harness with resistance bands at the tarsus/metatarsal bones pulling craniodorsally • Sit-to-stand exercises with sling assistance if needed 10-15 reps BID • Assisted walking through sand or tall grass 5 min SID-BID *Caution: Choose only 2-3 exercises per session. Doing all exercises may result in over-exertion and pain early in the program* Modalities • Effleurage BID-TID	• Response to activity and subjective pain/exertion level • Walking endurance and frequency of proprioceptive placing in underwater treadmill • Presence or absence of proprioception at a walk • Ability to support weight in standing • Height of obstacles navigated • Pelvic limb voluntary movement • Thigh circumference/ muscle mass	• Poor pelvic limb proprioception at a walk • Severe paraparesis or paraplegia at a walk • Extensive assistance required for pelvic limbs in weight-bearing

		Modalities • Gentle massage of limb and paraspinal musculature SID-BID prior to or following exercise • NMES Quadriceps and Hamstrings 15 min SID-EOD Other Use paw protective boots as proprioceptive deficits increase	Other Use paw protective boots as proprioceptive deficits increase		
Late-Stage: Pelvic limb paraplegia <u>Goals:</u> Reduce loss of muscle strength/ endurance Provide continued proprioceptive feedback Maintain quality of life Prolong survival time	Variable; 6-9 months post-diagnosis	Therapeutic Exercises • PROM with bicycling movements all joints of all limbs (10-15 reps BID-TID) • Flexor reflex exercises for pelvic and thoracic limbs, 10 reps BID • Aquatic therapy: Swimming 10-15 minutes with supported standing during breaks • Aquatic therapy: UWTM walking, 10-15 minutes SID. Use flotation devices/resistance bands to assist with proprioceptive placing if needed. Modalities • NMES to hindlimb musculature for contraction 15 min EOD-SID PRN	Therapeutic Exercises • PROM with bicycling movements all joints of all limbs (10-15 reps BID-TID) • Flexor reflex exercises for pelvic and thoracic limbs, 10 reps BID • Walks (with four-wheel cart) at routinely performed pace/ distance on level ground, 10-15 min BID-TID. *Use protective boots on pelvic limbs in cart unless limbs are completely suspended*	• Proprioception and postural reactions of thoracic limbs • Pelvic/thoracic limb reflexes • Subjective thoracic limb muscle mass • Patient attitude	• Complete loss of voluntary motion of pelvic limbs • Early, mild thoracic limb paraparesis
Late Stage: Pelvic Limb Paraplegia with Thoracic Limb Paraparesis <u>Goals:</u> Maintain patient comfort	Variable; up to 19-20 months post-diagnosis	Therapeutic Exercises • PROM with bicycling movements all joints of all limbs (10-15 reps BID-TID) • Flexor reflex exercises for pelvic and thoracic limbs, 10 reps BID • Aquatic therapy: Swimming 10-15 minutes with supported standing during breaks. *Personnel assistance should be provided in the pool to achieve limb movement and for safety* Other • Bladder expression TID-QID PRN • Use of soft bedding with rotation every 4-6 hours to prevent decubital ulcers	Therapeutic Exercises • PROM with bicycling movements all joints of all limbs (10-15 reps BID-TID) • Flexor reflex exercises for pelvic and thoracic limbs, 10 reps BID Other • Bladder expression TID-QID PRN Use of soft bedding with rotation every 4-6 hours to prevent decubital ulcers	• Pelvic/thoracic limb reflexes • Patient attitude	*End-stage of the disease with variable urinary and/or fecal incontinence and probability of eventual dysphagia or respiratory arrest*

This protocol is for use by licensed personnel who have been trained in canine rehabilitation.

*The therapist must keep in mind that these timelines are intended as guidelines. Rate of progression will vary widely among patients for reasons that include initial status of nociception and degree of neurologic deficits at the time of injury.

**Exercise regimen proposed to prolong survival time in dogs affected by degenerative myelopathy is based on Kathmann, I. et al. Daily Controlled Physiotherapy Increases Survival Time in Dogs with Suspected Degenerative Myelopathy. *J Vet Intern Med* 2006; 20: 927-932.

Distal Femoral Physeal Fractures

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Immobilization Goals: Pain control Swelling and edema control Encourage/improve ROM and hindlimb extension	Immediate to 48-96 hours postoperatively	Therapeutic Exercises • PROM and gentle stifle flexion/extension (10-15 reps TID-QID). *Specific attention should be paid to stifle flexion* • Elicit flexor reflex with interdigital manipulation 10 reps BID-TID • Balance and weight-shifting exercises BID-TID to encourage weight-bearing and active stifle flexion/extension on the operated limb Modalities • Cryotherapy (15-20 min (*first session immediately postoperative*) TID • TENS across fracture site SID-BID. Consider edema electrical stimulation protocol Medications • NSAIDs—daily PRN • Opioids—BID-TID PRN	Therapeutic Exercises • PROM and gentle stifle flexion/extension (10-15 reps TID-QID). *Specific attention should be paid to stifle flexion* • Balance/weight-shifting exercises to encourage toe-touching on a stable surface Immobilization between sessions for 2-4 days in a 90-degree/90-degree stifle/tarsal flexion sling** Modalities • Cryotherapy (15-20 min BID) following exercises	• Weight • Goniometry—stifle ROM • Swelling/edema • Response to PROM and subjective pain level	• Postoperative comfort • Incisional integrity maintained • Minimal pain with PROM • Early toe-touching to partial weight-bearing
Early Weight-Bearing Goals: Continued pain control Near-normal ROM and hindlimb extension Minimize muscle atrophy of operated limb Achieve consistent partial weight-bearing on operated limb	48-96 hours to 10 days postoperatively	Therapeutic Exercises • PROM bicycling movements with mild-moderate resistance and stifle flexion/extension stretching (10-15 reps BID-TID). *Specific attention should be paid to stifle flexion* • Slow, controlled leash or land treadmill walking, 5-10 min BID-TID to encourage active range of motion in the stifle • Balance and weight-shifting exercises BID-TID to encourage weight-bearing and active stifle flexion/extension on the operated limb	Therapeutic Exercises • PROM and flexion/extension hip stretches of operated limb (10-15 reps BID-TID) • Slow, controlled leash walking, 5-10 min TID-QID to encourage active range of motion in the stifle • Balance exercises on a soft foam pad for weight-bearing BID-TID Medications • NSAIDs—daily PRN • Opioids—BID-TID PRN	• Goniometry—stifle ROM and other joints if applicable • Thigh muscle girth/symmetry • Response to activity and subjective pain level • Lameness score at a stance and walk • Weight	• Consistent partial weight-bearing on operated limb on all strides at a walk • Minimal pain with light activities • Incisional healing without complications

		• Use of a syringe cap or other non-harmful irritant on contralateral foot during exercise to improve operated limb weight-bearing • Continued sling use to discourage stifle hyperextension; if present, should be considered with removal only for rehabilitation sessions Modalities • Heat therapy to quadriceps muscles 10 min prior to PROM/ exercise BID-TID • Massage of quadriceps muscles before active exercise • TENS across fracture repair site for analgesia SID-BID • Therapeutic laser PRN • Cryotherapy (15-20 min PRN) following exercises	Modalities • Cryotherapy (15-20 min BID) following exercises		
Consistent Weight-Bearing at a Walk <u>Goals:</u> Minimize pain during low- to moderate-intensity activity Maintain normal ROM and hindlimb flexion and extension Reduce and prevent further muscle atrophy Achieve minimal lameness at a walk	Ten days to three weeks postoperatively	Therapeutic Exercises • PROM and gentle stifle flexion/ extension (10-15 reps BID-TID), continued through 3-4 weeks postoperative • Underwater treadmill walking, 10-15 min SID-BID to encourage active range of motion in the stifle • Swimming 8-10 minutes SID-BID if hindlimbs are used • Balance and weight-shifting exercises BID-TID on increasingly unstable surfaces to improve muscular adjustments and active stifle flexion/extension on the operated limb	Therapeutic Exercises • PROM and flexion/ extension hip stretches of operated limb (10-15 reps BID-TID) • Slow, controlled leash walking, 10-15 min TID-QID to encourage active range of motion in the stifle • Include inclined surface into slow leash walks (3-5 minutes with each walking session) for quadriceps and gluteal muscle strengthening • Balance exercises on an unstable surface for weight-bearing BID-TID	• Goniometry—stifle ROM and other joints if applicable • Response to activity and subjective pain level	• Consistent substantial weight-bearing on operated limb on all strides at a walk • Minimal pain with moderate activities • Near-normal stifle passive ROM • Minimal to no stifle effusion

Continued

Distal Femoral Physeal Fractures—cont'd

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
		Modalities • Heat therapy to affected quadriceps BID-TID, 10 minutes • Massage of quadriceps muscles before active exercise PRN • Cryotherapy (15-20 min PRN) following exercises	Medications • NSAIDs—PRN • Opioids—SID-BID PRN Modalities • Heat therapy PRN before activity • Cryotherapy (15-20 min BID) following exercises	• Lameness score at a stance and walk • Weight	• Radiographic evidence of bone healing 3-4 weeks postoperatively
Partial Weight-Bearing at a Trot <u>Goals:</u> Pain-free moderate- to high-intensity activity Normal muscle mass of operated hindlimb Achieve soundness at a trot	Three to eight weeks postoperatively	Therapeutic Exercises • Aquatic therapy: UWTM walking 15-20 minutes SID-BID • Swimming 20-30 minutes SID-BID • Balance exercises on an inflatable disk or 360 degree wobble board for weight-bearing 10 min BID-TID • Land treadmill walking with caudal pull from resistance band to encourage quadriceps strengthening: 10-15 min SID-BID, wait until 4-6 weeks postoperative *Note: Ensure radiographic evidence of fracture healing and implant integrity present before proceeding to these exercises:* • Sit-to-stand exercises 10-20 reps BID • Cavaletti rail walking 5-10 reps over 5 rails • Stair Climbing 1 working up to 5 flights BID-TID Modalities • Cryotherapy PRN after exercises	Therapeutic Exercises • Leash walks 20-30 min including up to 10-15 min of inclines • Incorporate challenging surfaces to walks, eg snow, sand, tall grass—to increase stifle flexion *Note: Ensure radiographic evidence of fracture healing and implant integrity present before proceeding to these exercises:* • Sit-to-stand exercises 10-20 reps BID • Dancing exercises 30 sec-2 min BID-TID • Stair Climbing 1 working up to 5 flights BID-TID • Light jogging 5-8 minutes BID Medications • NSAIDs—PRN Modalities • Cryotherapy PRN after exercises	• Goniometry—stifle ROM and other joints if applicable • Re-evaluate thigh muscle girth at 7-8 weeks postoperative • Response to activity and subjective pain level • Lameness score at a stance, walk, and trot • Weight	• Consistent partial weight-bearing on operated limb at a trot • Minimal to no pain with extensive activities

*This protocol is for use by licensed personnel who have been trained in canine rehabilitation. The therapist must keep in mind that these timelines are intended as guidelines and may be influenced by many factors affecting individualized patient care.

**This device is intended to minimize the risk of quadriceps contracture by controlling/preventing stifle extension.

Degenerative Lumbosacral Stenosis—Conservative Management*

Stage	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures
Mild Pain Goals: No spinal/ paraspinal pain Perform at all gaits and balancing activities without pain Eliminate limb muscle atrophy Strengthen paraspinal muscles	Therapeutic Exercises *Introduce gradually as tolerated by the patient* • Balance on a 360-degree balance board (all four limbs) or semi-inflated disk (hindlimbs) 5 min SID working up to 10 min BID • Aquatic therapy: Underwater Treadmill (UWTM) walking, 5-10 min SID steady speed working up to 20-30 min BID variable speed • Swimming 5 working up to 20 minutes adding jets 2-3 times per week • Walking working up to jogging through weave poles, gradually decreasing distance between poles, 3 working up to 10 laps through 6-8 poles SID-BID • Cavaletti rails 5 working up to 10 reps, increasing height and decreasing distance, SID-BID • Walking along the length of a foam pad or mattress 5 working up to 10 reps SID-BID • Walking up and down stairs, 1-2 working up to 5-8 flights SID-BID. *Add this exercise only if lumbosacral pain is well controlled* • Sit-to-stand 5 working up to 15-20 reps SID-BID • Balance with pelvic limbs on a high-traction surface and antebrachii or forepaws balancing on a physioroll or peanut. Apply gradually increasingly challenging displacement forces in multiple directions to encourage walking of the hindlimbs while balancing. 5-10 min SID-BID. *Select an appropriately sized physioroll to prevent spinal hyperextension with this activity.* *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in over-exertion and pain early in the program*	Therapeutic Exercises *Introduce gradually as tolerated by the patient* • Balance on a moderately unstable surface (all four limbs) or semi-inflated disk (hindlimbs) 5 min SID working up to 10 min BID • Walking working up to jogging in circles and figure eights, gradually decreasing size, 5-10 min SID-BID • Walking in sand, snow, low water or across a foam mattress 5-10 minutes 2-3 times per week. • Sit-to-stand 5 working up to 15-20 reps SID-BID • Walking up and down stairs, 1-2 working up to 5-8 flights SID-BID. *Add this exercise only if lumbosacral pain is well controlled* • Balance "rides" while standing in a cart, wagon, or wheelbarrow over increasingly variable terrain, 5-10 min SID. *Ensure the patient is supported at all times to prevent falling.* • Moderate leash walks 20-30 min BID, incorporating 5 minutes of mild-moderate inclines and 2-5 minutes jogging *Caution: Choose only 2-4 exercises per session. Doing all exercises may result in over-exertion and pain early in the program.*	• Subjective pain level at digital pressure L7-S1, dorsal tail elevation, back hyperextension, and rectal palpation • Hindlimb proprioception and postural reactions • Thigh circumference • Ability to navigate challenging obstacles while jogging/turning

Continued

Degenerative Lumbosacral Stenosis—Conservative Management*—cont'd

Stage	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures
	Modalities • Warm packs across L7-S1 BID-TID or before exercise PRN • TENS across L7-S1 15-20 min SID-EOD • Gentle massage of lumbar epaxial and limb musculature SID-BID • Extracorporeal shock wave treatment Medications • NSAIDs, NMDA antagonists and other analgesics PRN • Epidural corticosteroid injection protocol if needed	Modalities • Warm packs across L7-S1 BID-TID or before exercise PRN • Effleurage BID-TID before exercise PRN • Cryotherapy to paraspinal region of injury 20 min after exercise PRN Medications • NSAIDs, NMDA antagonists and other analgesics PRN	
Moderate Pain** Goals: Mild-to-minimal pain with activity Controlled navigation on challenging surfaces Prevent/minimize limb muscle atrophy	Therapeutic Exercises *Introduce gradually as tolerated by the patient* • Balance on a bi-directional balance board (all four limbs) or semi-inflated disk (hindlimbs) 5 min SID-BID • Aquatic therapy: Underwater Treadmill (UWTM) walking, 5-10 min working up to 15-20 min SID slow, steady pace • Flexor reflex exercises for affected limbs (10-15 reps BID-TID) • Walking through weave poles, gradually decreasing distance, 5-10 laps through 6-8 poles SID • Low Cavaletti rails 5-10 reps SID-BID • Sit-to-stand 5 working up to 15-20 reps SID-BID	Therapeutic Exercises *Introduce gradually as tolerated by the patient* • Balance with very gentle displacement forces applied across a mildly unstable surface (eg sofa cushion) 5 min SID-BID • Flexor reflex exercises for affected limbs (10-15 reps BID-TID) • Walking in circles and figure eights, gradually decreasing size, 5-10 min SID-BID • Walking in sand, snow, low water or across a foam mattress 1-3 min 2-3 times per week. • Sit-to-stand 5 working up to 15-20 reps SID-BID • Moderate leash walks 10-20 min BID, incorporating 5 minutes of mild inclines	• Subjective pain level at digital pressure L7-S1, dorsal tail elevation, back hyperextension, and rectal palpation • Hindlimb proprioception and postural reactions • Thigh circumference
Maintain normal limb ROM Early strengthening of paraspinal muscles	• Balance with pelvic limbs on a high-traction surface and antebrachii or forepaws balancing on a physioroll or peanut. Apply gradually increasingly challenging displacement forces in multiple directions to encourage walking of the hindlimbs while balancing. 5 min SID. *Select an appropriately sized physioroll to prevent spinal hyperextension with this activity.* *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in over-exertion and pain early in the program*	*Caution: Choose only 2-4 exercises per session. Doing all exercises may result in over-exertion and pain early in the program.* Modalities • Warm packs across L7-S1 BID-TID or before exercise PRN • Effleurage BID-TID before exercise PRN • Cryotherapy to paraspinal region of injury 20 min after exercise PRN	• Ability to navigate obstacles and mild inclines at a walk *If consistent pain control is achieved move to mild pain protocol*

	Modalities • Warm packs across L7-S1 BID-TID or before exercise PRN • TENS across L7-S1 15-20 min SID-EOD • Extracorporeal shock wave treatment • Therapeutic laser lumbar epaxial muscles SID • Gentle massage of lumbar epaxial and limb musculature SID-BID Medications • NSAIDs, NMDA antagonists and other analgesics PRN • Epidural corticosteroid injection protocol if needed • Muscle relaxants PRN if severe epaxial/pelvic limb muscle spasms	Medications • NSAIDs, NMDA antagonists, and other analgesics PRN • Muscle relaxants PRN if severe epaxial/pelvic limb muscle spasms	
Severe Pain** <u>Goals:</u> Pain and muscle spasm reduction Maintain normal limb ROM Increase affected limb muscle mass Maintenance of balance during turns and on a moderately unstable surface	Therapeutic Exercises *Introduce gradually as tolerated by the patient* • Aquatic therapy: Underwater Treadmill (UWTM) walking, 2-5 min working up to 10-15 min SID slow, steady pace • Flexor reflex exercises for affected limbs (10-15 reps BID-TID) • PROM pelvic limbs 10-15 reps BID-TID • Low Cavaletti Rails 5 reps SID-BID • Sit-to-stand 5 reps SID-BID *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in over-exertion and pain early in the program* Modalities • Warm packs across L7-S1 BID-TID or before exercise PRN • TENS across L7-S1 15-20 min SID-EOD • Therapeutic laser lumbar epaxial muscles SID • Gentle massage of lumbar epaxial (if tolerated) and limb musculature SID-BID Medications • NSAIDs, NMDA antagonists, and other analgesics PRN • Epidural corticosteroid injection protocol if needed • Muscle relaxants PRN if severe epaxial/pelvic limb muscle spasms	Therapeutic Exercises *Introduce gradually as tolerated by the patient* • Balance with very gentle displacement forces applied across a mildly unstable surface (eg sofa cushion) 5 min SID-BID • Flexor reflex exercises for affected limbs (10-15 reps BID-TID) • PROM pelvic limbs 10-15 reps BID-TID • Walking in circles and figure eights, gradually decreasing size, 5-10 min SID-BID • Walking in sand or on uneven ground 1-3 min 2-3 times per week. • Sit-to-stand 5 reps BID • Slow leash walks 5-10 min BID *Caution: Choose only 2-4 exercises per session. Doing all exercises may result in over-exertion and pain early in the program.* Modalities • Warm packs across L7-S1 BID-TID or before exercise PRN • Effleurage BID-TID before exercise PRN • Cryotherapy to paraspinal region of injury 20 min after exercise PRN Medications • NSAIDs, NMDA antagonists, and other analgesics PRN • Muscle relaxants PRN if severe epaxial/pelvic limb muscle spasms	• Response to activity and subjective pain level when active and at rest • Length of time with unsupported standing • Length of time walking in aquatic therapy and on dry land • Frequency of steps in which appropriate proprioception is used • Height of objects navigated cleanly • Thigh circumference/ muscle mass *If consistent pain control is achieved move to moderate pain protocol*

This protocol is for use by licensed personnel who have been trained in canine rehabilitation.

*This protocol is designed for patients for whom conservative management is appropriate. Patients that have sacral subluxation at L7-S1 or an extruded intervertebral disk causing significant cauda equina compression that are good surgical candidates would likely receive greatest benefit from surgical intervention.

**Patients with moderate to severe pain, paraparesis, or ataxia should have appropriate diagnostic imaging and surgery if indicated.

Elbow Dysplasia—Post-arthroscopy**

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Step 1 Postoperative to Early Toe-Touching at a Walk Goals: Pain control Swelling and edema control Maintain/ improve elbow postoperative ROM	Immediate to 48-72 hours postoperatively	Therapeutic Exercises • Slow leash walking only to go outside, no more than five minutes at a time, with a harness • Gentle PROM affected thoracic limb (with emphasis on elbow flexion/extension), 8-10 repetitions BID-TID Modalities • Gentle massage (effleurage, shoulder muscles) • Transcutaneous electrical stimulation across the elbow for pain relief (15-20 min SID-TID) • Cryotherapy to affected elbow (15-20 min BID-TID) • Therapeutic Laser SID Medications • NSAIDs/other analgesics • Oral chondroprotective agents • Injectable polysulfated glycosaminoglycans	Therapeutic Exercises • Slow leash walking only to go outside, no more than five minutes at a time, with a harness • Gentle PROM affected thoracic limb (with emphasis on elbow flexion/extension), 8-10 repetitions BID-TID Modalities • Gentle massage (effleurage) • Cryotherapy to affected elbow (15-20 min BID-TID) Medications • NSAIDs/other analgesics • Oral chondroprotective agents • Injectable polysulfated glycosaminoglycans	• Postoperative elbow PROM (other joints if applicable)—goniometry • Response to activity and subjective pain level • Gait evaluation at a walk • Weight	• Consistent toe-touching on operated limb • Adequate resting analgesia • Decreased perioperative swelling and no incisional drainage • Comfortable elbow ROM 80-100 degrees flexion, 135-150 degrees extension
Step 2 Early Weight-Bearing, Moderate Elbow Flexion Goals: Continue pain control Improve elbow ROM	48-72 hours to two weeks postoperatively	Therapeutic Exercises • Land Treadmill: Begin with slight incline or level surface, 5-10 minutes at a slow, steady pace BID with a harness • PROM within comfortable range affected thoracic limb (with emphasis on elbow flexion/ extension), 10-15 repetitions BID-TID	Therapeutic Exercises • Slow, steady leash walking on flat ground 5 min working up to 15 minutes at a time with a harness • PROM within comfortable range affected thoracic limb (with emphasis on elbow flexion/extension), 10-15 repetitions BID-TID	• Response to light activity and subjective pain level • Gait evaluation at walk • Goniometry—Elbow ROM and other joints if applicable	• Consistent partial weight-bearing with each stride on operated limb • Minimal pain with light activities • Incisional healing without complications

Increase toe-touching weight-bearing Prevent/minimize thoracic limb muscle atrophy		• Balance/weight-shifting exercises on a bi-directional board or soft foam pad 5-10 min BID-TID • "Shaking paw" exercises (begin training if applicable) 8-10 reps BID-TID • Flexor withdrawal stimulation 8-10 reps BID affected thoracic limb Modalities • Transcutaneous electrical stimulation across the elbow for pain relief (15-20 min PRN) • NMES muscles affected thoracic limb 15 min SID/PRN for atrophy prevention • Cryotherapy to affected elbow (15-20 min BID-TID PRN after exercise) • Therapeutic Laser SID Medications • NSAIDs/other analgesics • Oral chondroprotective agents • Injectable polysulfated glycosaminoglycans	• Balance/weight-shifting exercises for thoracic limbs on a semi-firm surface 5-10 min BID-TID • "Shaking paw" exercises (begin training if applicable) 8-10 reps BID-TID • Flexor withdrawal stimulation 8-10 reps BID affected thoracic limb Modalities • Gentle massage (effleurage, scapular muscles) • Cryotherapy to affected elbow (15-20 min BID-TID PRN after exercise) Medications • NSAIDs/other analgesics • Oral chondroprotective agents • Injectable polysulfated glycosaminoglycans	• Subjective thoracic limb muscle mass • Weight	• Elbow ROM 60-80 degrees flexion, 145-155 degrees extension
Step 3 Weight-Bearing at a Walk, Near-Normal Elbow Flexion	Two to four weeks postoperatively	Therapeutic Exercises • Land Treadmill: Begin with slight incline or level surface, 5-10 minutes at a slow, steady pace BID with a harness • PROM within comfortable range affected thoracic limb (with emphasis on elbow flexion/extension), 10-15 repetitions BID-TID • Aquatic Therapy: Underwater Treadmill (UWTM) slow walking 5-10 min SID-BID	Weight loss program can be initiated at this phase if warranted (minimizes loss of lean tissue mass). Therapeutic Exercises • Slow, steady leash walking on flat ground 5 min working up to 15 minutes at a time with a harness • PROM within comfortable range affected thoracic limb (with emphasis on elbow flexion/extension), 10-15 repetitions BID-TID	• Response to moderate activity and subjective pain level • Gait evaluation at walk	• Consistent weight-bearing on operated limb at walk • No evidence of pain with moderate activities

Continued

Elbow Dysplasia—Post-arthroscopy—cont'd**

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Goals: Minimize pain during exercises Achieve near-normal elbow ROM (unless significant osteoarthritis is present) Improve weight-bearing at a walk Improve operated limb muscle mass/strength		• Swimming 5-10 minutes 3-4 times per week with breaks (*Note: avoid this activity until 4-6 weeks postoperatively if excessive force observed with forelimb movements*) • Balance/weight-shifting exercises on a 360-degree balance board 5-10 min BID-TID • "Shaking paw" exercises 10-15 reps BID-TID. Add light weights to proximal antebrachium as tolerated for muscle strengthening. • Flexor withdrawal stimulation 8-10 reps BID affected thoracic limb *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program* Modalities • Heat therapy at elbow prior to PROM exercises 10 min (if no signs of acute inflammation present) • Transcutaneous electrical stimulation across the elbow for pain relief (15-20 min PRN) • NMES thoracic limb muscles affected thoracic limb 15 min PRN for atrophy prevention • Cryotherapy to affected elbow (15-20 min BID-TID PRN after exercise) • Therapeutic Laser PRN	• Balance/weight-shifting exercises for thoracic limbs on a semi-unstable surface 5-10 min BID-TID • "Shaking paw" exercises 10-15 reps BID-TID. Add light weights to proximal antebrachium as tolerated for muscle strengthening. • Flexor withdrawal stimulation 8-10 reps BID affected thoracic limb Modalities • Heat therapy at elbow prior to PROM exercises 10 min (if no signs of acute inflammation present) • Gentle massage (effleurage, scapular muscles) • Cryotherapy to affected elbow (15-20 min BID-TID PRN after exercise) Medications • NSAIDs/other analgesics • Oral chondroprotective agents • Injectable polysulfated glycosaminoglycans	• Goniometry—Elbow ROM and other joints if applicable • Subjective thoracic limb muscle mass • Weight	• Elbow PROM 40-60 degrees flexion, >150 degrees extension

		Medications • NSAIDs/other analgesics • Oral chondroprotective agents • Injectable polysulfated glycosaminoglycans			
Step 4 Consistent Weight-Bearing at a Walk, Normal Elbow Flexion Goals: Normal limb use at a walk Early weight-bearing at a trot Improve thoracic limb muscle mass and strength Improve stamina and endurance	Four to eight weeks postoperatively	Therapeutic Exercises • Land Treadmill: Level surface working up to slight decline, walking 10-15 minutes SID-BID with a harness • PROM affected thoracic limb (with emphasis on elbow flexion/extension), 10-15 repetitions PRN if ROM is not within normal limits • Aquatic Therapy: Underwater Treadmill (UWTM) walking 10-15 min SID-BID, gradually introducing light weights/flotation device on distal affected thoracic limb in the water • Swimming 10-15 minutes 3-4 times per week with breaks, gradually introducing light weights/flotation device on distal affected thoracic limb in the water • Balance/weight-shifting exercises on a 360-degree balance board 5-10 min BID-TID • "Shaking paw" exercises 10-15 reps BID-TID. Light weights to distal antebrachium as tolerated for muscle strengthening • Wheelbarrowing 5-8 reps of 10-15 seconds BID. *Begin at 6-8 weeks postoperatively, as feasible depending on patient weight* • Low Cavaletti Rails 5-10 reps BID *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program*	Continue weight loss program if needed Therapeutic Exercises • Slow, steady leash walking on flat ground 5 min working up to 15 min at a time with a harness • PROM within comfortable range affected thoracic limb (with emphasis on elbow flexion/extension), 10-15 repetitions BID-TID • Balance/weight-shifting exercises for thoracic limbs on a semi-unstable surface 5-10 min BID-TID • "Shaking paw" exercises 10-15 reps BID-TID. Light weights to distal antebrachium as tolerated for muscle strengthening • Wheelbarrowing 5-8 reps of 10-15 seconds BID. *Begin at 6-8 weeks postoperatively, as feasible depending on patient weight* *Caution: Choose only 2-4 exercises per session. Doing all exercises may result in lameness and pain early in the program* Modalities • Heat therapy at elbow prior to PROM exercises 10 min • Cryotherapy to affected elbow (15-20 min BID-TID PRN after exercise)	• Response to moderate/heavy activity and subjective pain level • Goniometry—Elbow ROM and other joints if applicable • Gait evaluation at walk and trot • Subjective thoracic limb muscle mass • Weight	• Minimal to no lameness at a walk • Partial weight-bearing at a trot • No evidence of pain with moderate-intense activities • Normal elbow range of motion

Continued

Elbow Dysplasia—Post-arthroscopy—cont'd**

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
		Modalities • Heat therapy at elbow prior to PROM exercises 10 min • Transcutaneous electrical stimulation across the elbow for pain relief (15-20 min PRN) • Cryotherapy to affected elbow (15-20 min BID-TID PRN after exercise) • Therapeutic Laser PRN Medications • NSAIDs PRN • Oral chondroprotective agents • Injectable polysulfated glycosaminoglycans	Medications • NSAIDs PRN • Oral chondroprotective agents • Injectable polysulfated glycosaminoglycans		
Step 5 Consistent Weight-Bearing at a Trot Goals: Trot working up to faster gaits with minimal to no lameness Continue improving muscle mass, strength, and power Continue improving stamina and endurance	Eight to twelve weeks postoperatively and beyond	Therapeutic Exercises • Land Treadmill: Trotting at a level surface working up to a decline, 5-15 min SID-BID with a harness • PROM affected thoracic limb (with emphasis on elbow flexion/extension), 10-15 repetitions PRN if ROM is not within normal limits • Aquatic Therapy: Underwater Treadmill (UWTM) walking 20-30 min SID-BID at moderate/variable speeds, light weights/flotation device on distal affected thoracic limb in the water • Swimming 15-20 minutes 3-4 times per week with breaks, light weights/flotation device on distal affected thoracic limb in the water	Continue weight loss program if needed Therapeutic Exercises • Slow, steady leash walking on flat ground 5 min working up to 15 minutes at a time with a harness • PROM within comfortable range affected thoracic limb (with emphasis on elbow flexion/extension), 10-15 repetitions BID-TID • Balance/weight-shifting exercises for thoracic limbs on a semi-unstable surface 5-10 min BID-TID	• Response to increasing activity and subjective pain level • Gait evaluation at walk and trot • Subjective thoracic limb muscle mass evaluation every 2 weeks until symmetry achieved • Weight	Progressions may be continued as long as lameness or pain with activity is not observed. If this occurs, decrease activity by 50% for 1 week and then increase again by 10-20% weekly.

Return to as normal activity as possible	• Balance/weight-shifting exercises on a 360-degree balance board 10 min BID-TID • "Shaking paw" exercises 10-15 reps BID-TID. Weights to distal antebrachium as tolerated for muscle strengthening • Wheelbarrowing 5-8 reps of 10-15 seconds BID. *Begin at 6-8 weeks postoperatively, as feasible depending on patient weight* • Climbing up and down stairs (slow, controlled on harness and leash) 1-2 flights working up to 5-6 flights BID • Cavaletti Rails 10-15 reps BID *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program* Modalities • Heat therapy at elbow prior to PROM exercises 10 min PRN • Cryotherapy to affected elbow (15-20 min BID-TID PRN after exercise) • Extracorporeal shock wave Medications • NSAIDs PRN • Oral chondroprotective agents • Injectable polysulfated glycosaminoglycans	• "Shaking paw" exercises 10-15 reps BID-TID. Weights to distal antebrachium as tolerated for muscle strengthening • Wheelbarrowing 5-8 reps of 10-15 seconds BID. *Begin at 6-8 weeks postoperatively, as feasible depending on patient weight* • Climbing up and down stairs (slow, controlled on harness and leash) 1-2 flights working up to 5-6 flights BID • Controlled ball play 5 min working up to 15 min SID-BID *Caution: Choose only 2-4 exercises per session. Doing all exercises may result in lameness and pain early in the program* Modalities • Heat therapy at elbow prior to PROM exercises 10 min PRN • Cryotherapy to affected elbow (15-20 min BID-TID PRN after exercise) Medications • NSAIDs PRN • Oral chondroprotective agents • Injectable polysulfated glycosaminoglycans

This protocol is for use only by licensed personnel who have been trained in canine rehabilitation.

*The therapist must keep in mind that these timelines are intended as guidelines and may be influenced by many factors affecting individualized patient care.

**This condition is comprised of one or more of the following: osteochondritis dissecans of the medial humeral condyle, fragmented medial coronoid process, ununited anconeal process.

Fibrocartilaginous Embolic Myelopathy**

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Initial Weight Support Goals: Appreciable limb tone Weight-bearing in standing with minimal support Minimize limb muscle atrophy Independent urination and defecation	Immediate to three to seven days post-injury	Therapeutic Exercises • Assisted standing with support as needed on a stable, high-traction surface (*beginning 24 hours post-injury*) • PROM with bicycling movements all joints of pelvic (+/– thoracic) limbs (10-15 reps BID-TID) • Aquatic therapy: Underwater Treadmill (UWTM) standing, 3-5 min SID-BID, with personnel assistance in the water as needed • Flexor reflex exercises for affected limbs (10-15 reps BID-TID) Modalities • Cryotherapy to paraspinal region of injury 20 min TID if pain assessed • TENS to paraspinal region of injury 20 min SID-BID if pain assessed • NMES to pelvic limb (+/– thoracic limb) musculature for contraction 15 min SID • Gentle massage of limb musculature +/– tapotement BID as tolerated Medications • Muscle relaxants BID-TID PRN Other • Bladder expression TID-QID PRN • Use of soft bedding with rotation every 4-6 hours to prevent decubital ulcers	Therapeutic Exercises • Assisted standing with support as needed on a stable, high-traction surface (*beginning 24 hours post-injury*) • PROM with bicycling movements all joints of pelvic (+/– thoracic) limbs (10-15 reps BID-TID) • Flexor reflex exercises for affected limbs (10-15 reps BID-TID) Modalities • Cryotherapy to paraspinal region of injury 20 min TID if needed • Effleurage BID-TID Medications • Muscle relaxants BID-TID PRN Other • Bladder expression TID-QID PRN • Use of soft bedding with rotation every 4-6 hours to prevent decubital ulcers	• Subjective comfort level • Pelvic (and thoracic, if affected) limb muscle tone • Thigh circumference • Degree of support required in standing	• Standing in weight-bearing with minimal support • Initial voluntary stepping movement when attempting to walk

Weight Support to Initial Motor Function Goals: Pain-free activity Full weight-bearing in standing Minimize limb muscle atrophy Movement of all four limbs at a slow, supported walk Self-rotation	One to two weeks post-injury	Therapeutic Exercises • Assisted standing and weight-shifting/rhythmic stabilization, gradually decreasing support (eg physioball beneath abdomen, standing in water) • PROM with bicycling movements all joints of pelvic (+/– thoracic) limbs (10-15 reps BID-TID) • Aquatic therapy: Supported swimming 2-5 minutes working up to 10 minutes with supported standing during breaks. Personnel accompaniment in water • Aquatic therapy: Underwater Treadmill (UWTM) assisted walking, 5-15 min SID-BID • Turning exercises during standing with positive reinforcement held at various locations (concentrate efforts on the weaker side), encouraging stepping movements towards a reward • Slow leash walking with sling support 5 minutes TID, encouraging stepping Modalities • Gentle massage of affected limb musculature BID-TID • NMES to affected limb musculature for contraction 15 min EOD-SID Medications • Muscle relaxants BID-TID PRN Other • Bladder expression TID-QID PRN • Use of soft bedding with rotation every 4-6 hours if not self-rotating	Therapeutic Exercises • Assisted standing and weight-shifting/rhythmic stabilization, gradually decreasing support (eg physioball beneath abdomen, standing on non-slick surface in the bathtub) • PROM with bicycling movements all joints of pelvic (+/– thoracic) limbs (10-15 reps BID-TID) • Slow leash walking with sling support 5 minutes TID, encouraging stepping • Flexor reflex exercises for affected limbs (10-15 reps BID-TID) Modalities • Gentle massage of hindlimb musculature BID-TID Medications • Muscle relaxants BID-TID PRN Other • Bladder expression TID-QID PRN • Use of soft bedding with rotation every 4-6 hours if not self-rotating	• Response to activity and subjective pain/effort level • Length of time with unsupported standing • Number of steps in which gait patterning is used • Thigh circumference/muscle mass	• Limb voluntary motor activity • Unsupported standing

Continued

Fibrocartilaginous Embolic Myelopathy—cont'd**

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Initial Motor Function to Good Motor Function with Proprioceptive Deficits Goals: Improve proprioception during walking activity Maintain normal limb ROM Increase affected limb muscle mass Maintenance of balance during turns and on a moderately unstable surface	Two to four weeks post-injury	Therapeutic Exercises • Assisted standing and weight-shifting on a bi-directional balance board • PROM with bicycling movements all joints of pelvic (+/– thoracic) limbs (10-15 reps PRN based on ROM) • Aquatic therapy: Swimming 10-15 minutes with supported standing during breaks • Aquatic therapy: Underwater Treadmill (UWTM) walking, 10-15 minutes SID. Use flotation devices/resistance bands to assist with proprioceptive placing if needed. • Gait patterning on a ground treadmill, 5-10 minutes SID with resistance band use PRN • Turning exercises during standing with positive reinforcement held at various locations (concentrate efforts on the weaker side) • Turning exercises during walking: circles and figure eights 5-10 reps SID-BID • Low Cavaletti rails 5-10 reps SID-BID • Sit-to-stand exercises 5-10 reps BID with hindquarters on an elevated surface or with sling support *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in over-exertion and pain early in the program* Modalities • NMES to hindlimb musculature for contraction 15 min EOD-SID PRN	Therapeutic Exercises • Assisted standing and weight-shifting on a moderately unstable surface • PROM with bicycling movements all joints of pelvic (+/– thoracic) limbs (10-15 reps BID-TID PRN) • Slow leash walking with sling available 5-10 minutes BID-TID • Turning exercises during standing with positive reinforcement held at various locations (concentrate efforts on the weaker side) • Turning exercises during walking: circles and figure eights 5-10 reps SID-BID • Walking across an unstable surface such as a mattress 5-10 reps BID • Walking over 5-8 evenly spaced low obstacles 5-10 reps BID *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in over-exertion and pain early in the program*	• Response to activity and subjective pain level • Length of time with unsupported standing • Length of time walking in aquatic therapy and on dry land • Frequency of steps in which appropriate proprioception is used • Height of Cavaletti rails navigated • Thigh circumference/ muscle mass	• Consistent strong limb movements at a walk • Minimal over-exertion with moderate activities

Good motor function with proprioceptive deficits to near normal gait Goals: Consistent proprioception with moderate- to high-intensity activity Affected limb muscle mass approaching normal Maintain balance during turns and negotiating obstacles while walking Consistent gait-patterning in early trotting	Four to six weeks post-injury	Therapeutic Exercises • Assisted standing and weight-shifting on a 360-degree balance board • Aquatic therapy: Swimming 20-30 minutes with supported standing during breaks • Aquatic therapy: Underwater Treadmill (UWTM) walking, 15-20 minutes • Gait patterning on a ground treadmill, 5-10 minutes with resistance band use PRN. Introduce trotting for 1-2 minutes. • Trotting 2-3 minutes over even ground BID • Turning exercises during walking: weave poles gradually decreasing in distance 5-10 reps SID-BID • Cavaletti rails 10 reps SID-BID • Stair Climbing 1 working up to 5 flights SID-BID • Sit-to-stand exercises, 5-10 reps SID-BID *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program*	Therapeutic Exercises • Assisted standing and weight-shifting on an unstable surface • Turning exercises during walking: weave poles gradually decreasing in distance 5-10 reps SID-BID • Stair Climbing 1 working up to 5 flights SID-BID • Sit-to-stand exercises, rising from an elevated surface 5-10 reps SID-BID • Leash walking 10-15 min BID-TID with 5 minutes inclines/declines • Trotting 2-3 minutes over even ground BID • Walking across an unstable surface such as a mattress 10-15 reps BID • Walking over 5-8 evenly spaced low obstacles 10-15 reps BID *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program*	• Response to activity and subjective pain level • Length of time with unsupported standing • Walking endurance • Tightness of turns in which balance is maintained • Frequency of steps in which appropriate proprioception is used • Height of Cavaletti rails navigated • Thigh circumference/ muscle mass	• Consistent pelvic limb proprioception at a walk • Minimal to no over-exertion or pain with extensive activities • Normal gait patterning at a trot

Continued

Fibrocartilaginous Embolic Myelopathy—cont'd**

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Near Normal Gait to Normal Gait Goals: Elimination of proprioceptive deficits during high-intensity activity Normal affected limb muscle mass Maintain balance during turns and negotiating obstacles while trotting	Six or more weeks post-injury	Therapeutic Exercises • Assisted standing and weight-shifting on a 360-degree balance board • Aquatic therapy: Swimming 20-30 minutes • Aquatic therapy: Underwater Treadmill (UWTM) walking, 20-30 minutes at variable speeds • Gait patterning on a ground treadmill, 5-10 minutes at a trot • Turning exercises with weave poles 10-20 reps SID-BID at a trot • Cavaletti rails 10-15 reps SID-BID at a trot • Stair Climbing 5-10 repetitions SID-BID • Sit-to-stand exercises 10-20 reps SID-BID • Controlled ball play *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program*	Therapeutic Exercises • Assisted standing and weight-shifting on an unstable surface • Turning exercises with weave poles 10-20 reps SID-BID at a trot • Stair Climbing 5-10 repetitions SID-BID • Sit-to-stand exercises 10-20 reps SID-BID • Controlled ball play • Leash walking 20-30 minutes BID with 10 minutes variable inclines and 5-10 minutes working up to longer periods trotting • Trotting across an unstable surface such as a mattress 10-15 reps BID • Trotting over 5-8 evenly spaced low obstacles 10-15 reps BID *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program*	• Response to activity and subjective pain level • Walking and trotting endurance • Tightness of turns in which balance is maintained • Height and distance of Cavaletti rails navigated • Pelvic limb thigh circumference/ muscle mass	• Minimal to no over-exertion with extensive activities • Normal gait and proprioception at a trot and at faster speeds

This protocol is for use by licensed personnel who have been trained in canine rehabilitation.

*The therapist must keep in mind that these timelines are intended as guidelines. Rate of recovery will vary widely among patients for reasons that include initial status of nociception and degree of spinal cord injury. Approximately 50% of patients show improvement in the first few weeks.

**Fibrocartilaginous embolism is often difficult to differentiate from acute intervertebral disk extrusion. Appropriate diagnostics should be performed to differentiate the conditions to determine the need for surgical decompression. Caution should be taken and progression of rehabilitation should be slower in patients for which the latter condition may be suspected.

Femoral Head and Neck Ostectomy

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Non-Weight-Bearing to Toe-Touching	Immediate to 48-72 hours postoperative	Therapeutic Exercises • Slow, gentle hip pseudoarthrosis PROM for operated limb focusing on extension (10 reps TID-QID beginning immediately postoperative while recovering from anesthesia) • Slow leash walking with sling support available, only to go outside (up to 5 minutes, TID-QID) • Balance exercises on a soft foam pad or bi-directional balance board for weight-bearing Medications • NSAIDs • Opioids: injectable/CRI Modalities • Gentle massage around the pseudoarthrosis, thigh, and lumbosacral regions • Transcutaneous electrical stimulation for pain relief (15-20 min SID-TID) • Cryotherapy (15-20 min TID after activities)—*first session immediately postoperative in combination with slow PROM while recovering from anesthesia* • Therapeutic laser therapy SID	Inpatient status preferred during this phase If Home: Therapeutic Exercises • Slow, gentle hip pseudoarthrosis PROM for operated limb focusing on extension (10 reps TID-QID beginning immediately postoperative) • Slow leash walking with sling support available, only to go outside (up to 5 minutes, TID-QID) • Balance exercises on a semi-firm surface for weight-bearing Medications • NSAIDs • Opioids Modalities • Gentle massage around the pseudoarthrosis, thigh, and lumbosacral regions • Cryotherapy (15-20 min TID after activities)—*first session immediately postoperative*	• Postoperative bilateral "hip" PROM and other joints as applicable via goniometry • Postoperative bilateral thigh circumference • Response to activity and subjective pain level • Lameness score at a stance and walk • Weight	• Early toe-touching • Adequate resting analgesia • Decreased perioperative swelling and lack of incisional drainage

Continued

Femoral Head and Neck Ostectomy—cont'd

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Early Weight-Bearing	72 hours to two weeks postoperative	Therapeutic Exercises • PROM and flexion/extension hip stretches of operated limb (10-15 reps BID-TID). Bicycling and flexor reflex exercises • Slow, controlled walking on a land treadmill, 5-10 min including mild incline settings to encourage hip extension and target gluteal muscles • Balance exercises on a soft foam pad or bi-directional balance board for weight-bearing BID-TID Modalities • Heat therapy before activity (10-15 min BID-TID, NOT within 72 hours after surgery or if CS of acute inflammation are still present) • Therapeutic ultrasound SID • Massage • Therapeutic laser therapy PRN • Cryotherapy (15-20 min BID) following exercises	Therapeutic Exercises • PROM and flexion/extension hip stretches of operated limb (10-15 reps BID-TID) • Slow, controlled leash walking, 5-20 min including mild inclines to encourage hip extension and target gluteal muscles • Balance exercises on a soft foam pad for weight-bearing BID-TID Medications • NSAIDs—PRN • Opioids—PRN Modalities • Heat therapy (10-15 min BID-TID, prior to exercises, NOT within 72 hours after surgery or if CS of acute inflammation are still present) • Cryotherapy (15-20 min BID) following exercises	• Goniometry—Hip ROM and other joints if applicable • Response to activity and subjective pain level • Lameness score at a stance and walk • Weight	• Consistent partial weight-bearing on operated limb on all strides at a walk • Minimal pain with light activities • Incisional healing without complications
Consistent Weight-Bearing at a Walk	Two to four weeks postoperative	Therapeutic Exercises • PROM and flexion/extension hip stretches of operated limb (10-15 reps SID-BID) • Controlled walking on a land treadmill, 10-15 min with increased incline angle and speed SID • Balance exercises on an inflatable disk or 360 degree wobble board for weight-bearing 5 min BID-TID	Therapeutic Exercises • PROM and flexion/extension hip stretches of operated limb (10-15 reps BID-TID) • Leash walks 15-20 min including 5-10 min of inclines • Balance exercises on an inflatable disk for weight-bearing BID-TID • Sit-to-stand exercises 5-10 reps BID	• Goniometry—Hip ROM and other joints if applicable • Re-evaluate thigh muscle girth at 3-4 weeks postoperative • Response to activity and subjective pain level	• Consistent weight-bearing on operated limb on all strides at a walk, consistent partial weight-bearing at a trot

		• Encourage increased weight-bearing on operated limb (examples: initiating dancing exercises as tolerated 5 min BID-TID, applying weight on operated limb at 3-5% body weight or syringe cap under contralateral foot) • Sit-to-stand exercises 5-10 reps BID • Aquatic therapy: UWTM walking 5-10 minutes once incision is sealed SID-BID • Swimming 3-5 days per week • Cavaletti Rails 5-10 reps BID Modalities • Heat therapy before activity • Therapeutic ultrasound PRN • Massage PRN • Therapeutic laser therapy PRN • Cryotherapy (15-20 min BID) following exercises	• Light jogging 3-5 min per day • Stairs: 1 flight SID-BID Medications • NSAIDs—PRN Modalities • Heat therapy (10 min) before activity • Cryotherapy (15-20 min) following exercises	• Lameness score at a stance, walk and trot • Weight	• Minimal pain with light activities
Consistent Weight-Bearing at a Trot	Five to eight weeks postoperative	Therapeutic Exercises • PROM and flexion/extension hip stretches of operated limb PRN • Controlled walking or light jogging on a land treadmill, 15-20 min with increased incline angle and speed SID • Balance exercises on an inflatable disk or 360 degree wobble board for weight-bearing 10 min BID-TID • Sit-to-stand exercises 10-20 reps BID • Aquatic therapy: UWTM walking 15-30 minutes SID-BID • Swimming 2-5 days per week Modalities • Heat therapy PRN before activity • Cryotherapy PRN after exercises	Therapeutic Exercises • PROM and flexion/extension hip stretches of operated limb PRN • Leash walks 20-30 min including up to 10-15 min of inclines, may use weights on affected limb or pulled with a harness as tolerated/required • Incorporate challenging surfaces to walks, eg snow, sand, when possible • Controlled ball-playing with gradually increasing times and distances • Sit-to-stand exercises 10-20 reps BID • Light jogging 3-5 min per day • Stairs: 2-4 flights SID-BID Medications • NSAIDs—PRN	• Goniometry—Hip ROM and other joints if applicable • Re-evaluate thigh muscle girth at 7-8 weeks postoperative • Response to activity and subjective pain level • Lameness score at a stance, walk, and trot • Weight	• Consistent weight-bearing on operated limb at a trot • Minimal to no pain with moderate to extensive activities

Continued

Femoral Head and Neck Ostectomy—cont'd

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
			Modalities • Heat therapy PRN before activity • Cryotherapy PRN after exercises		
Trotting at Speed with Minimal to No Lameness	Nine weeks postoperative and beyond	Aquatic therapy as desired; otherwise exercises may be continued as part of a Home Exercise Program.	Therapeutic Exercises • PROM for operated limb PRN • Leash walks at times and distances tolerated, including fast walks up inclined surfaces • Sit-to-stand exercises 20-30 reps as needed • Jogging: working up from 10-15 minutes per day • Stairs: walking and trotting, increasing number of flights as tolerated • Swimming or walking in mid-to-upper-thigh-level water • Ball-playing with gradually increasing times and distances, becoming more vigorous over time Medications • NSAIDs—PRN	• Goniometry every 3-4 weeks if needed • Re-evaluate thigh muscle girth every 3-4 weeks PRN • Response to activity and subjective pain level • Lameness score at a stance, walk, and trot • Weight	• Consistent weight-bearing on operated limb at a trot; permanent mild gait deficits may persist • Minimal to no pain with extensive activities

*This protocol is for use by licensed personnel who have been trained in canine rehabilitation. The therapist must keep in mind that these timelines are intended as guidelines and may be influenced by many factors affecting individualized patient care. Patients receiving femoral head and neck surgery as a result of trauma, such as fracture of the acetabulum, generally require longer to recover than those receiving surgery as a result of severe hip dysplasia.

Hip Luxation—Craniodorsal, Closed Reduction**

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Immobilization Goals Minimize post-procedural pain Maintain coxofemoral joint in coaptation/ reduction	Immediately to 2 weeks post-reduction	*The patient's affected pelvic limb should be placed in an Ehmer sling for 2 weeks after closed reduction, to maintain the limb abducted and internally rotated at the hip joint. This is necessary in hopes of maintaining hip reduction through early healing of joint capsule and periarticular soft tissues.* Therapeutic Exercises • Slow, controlled leash walks with Ehmer sling in place, 5 minutes only to go outside Modalities • Cryotherapy at coxofemoral joint for 48-72 hours after closed reduction, 20 min TID • Heat therapy at coxofemoral joint (only after acute inflammation has subsided, at least 72 hours after reduction) 10 min BID-TID • Massage of lumbar spinal and accessible thigh muscles (while limb is immobilized) • TENS across coxofemoral joint 20 min BID-TID Medications • NSAIDs/other analgesics	*The patient's affected pelvic limb should be placed in an Ehmer sling for 2 weeks after closed reduction, to maintain the limb abducted and internally rotated at the hip joint. This is necessary in hopes of maintaining hip reduction through early healing of joint capsule and periarticular soft tissues.* Therapeutic Exercises • Slow, controlled leash walks with Ehmer sling in place, 5 minutes only to go outside Modalities • Cryotherapy at coxofemoral joint for 48-72 hours after closed reduction, 20 min TID • Heat therapy at coxofemoral joint (only after acute inflammation has subsided, at least 72 hours after reduction) 10 min BID-TID • Massage of lumbar spinal and accessible thigh muscles (while limb is immobilized) Medications • NSAIDs/other analgesics	• Morbidity associated with the sling apparatus • Range of motion of pelvic limb joints after immobilization period • Thigh circumference of affected limb after immobilization period • Degree of weight-bearing after immobilization period • Weight	• Well-maintained hip reduction with movement within the sagittal plane, indicating appropriate capsular/soft tissue healing under way • Adequate analgesia after closed reduction

Continued

Hip Luxation—Craniodorsal, Closed Reduction—cont'd**

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Early Weight-Bearing Goals Minimize pain with early activity Improve weight-bearing of affected limb Maintain hip in reduction with mild activity Improve range of motion Increase muscle mass	Two to four weeks post-reduction	Therapeutic Exercises *Care must be taken with all exercises to ensure external rotation and adduction of the hip do not occur at this stage.* • Slow, controlled leash walks, 5 min TID-QID on flat surface or mild incline, support as needed • PROM and very gentle flexion/extension stretches of affected hip and other joints as needed 10-15 reps SID-BID. *It is critical to maintain movement in the sagittal plane* • Aquatic Therapy: Underwater Treadmill (UWTM) walking 5 working up to 10 min SID-BID at a slow, steady pace with support as needed, beginning 4 weeks after reduction • Balance on a bi-directional rocker board with hindlimbs at a wide base (in slight abduction), 5-10 min SID-BID Modalities • Heat therapy at coxofemoral joint and thigh muscles 10 min BID-TID before exercise • Massage of lumbar spinal and thigh muscles • Therapeutic ultrasound (continuous) to coxofemoral joint SID • Therapeutic laser hip PRN • Cryotherapy at coxofemoral joint 20 min PRN after exercise • NMES affected thigh muscles 15 min EOD-SID PRN Medications • NSAIDs/other analgesics PRN	Therapeutic Exercises *Care must be taken with all exercises to ensure external rotation and adduction of the hip do not occur at this stage.* • Slow, controlled leash walks, 5 min TID-QID on flat surface or mild incline, support as needed • PROM of affected hip and other joints as needed (10-15 reps SID-BID. *It is critical to maintain movement in the sagittal plane* • Balance on a semi-firm surface such as a sofa cushion with hindlimbs at a wide base (in slight abduction), 5-10 min SID-BID Modalities • Heat therapy at coxofemoral joint and thigh muscles 10 min BID-TID before exercise • Massage—effleurage • Cryotherapy at coxofemoral joint 20 min PRN after exercise Medications • NSAIDs/other analgesics PRN	• Goniometry—Hip ROM (flexion and extension only) and other joints if applicable • Response to light activity and subjective pain level • Thigh muscle circumference (evaluate at 2 and 4 weeks post reduction) • Lameness score at a stance and walk • Weight	• Consistent partial weight-bearing on affected limb on all strides at a walk • Minimal pain with light activities • Maintenance of hip reduction at current activity level • Hip PROM: <60 degrees flexion and >130-140 degrees extension

Consistent Weight-Bearing at a Walk <u>Goals</u> Minimize pain with increased activity Improve weight-bearing of affected limb Maintain hip in reduction Improve range of motion Increase muscle mass	Four to eight weeks post-reduction	Therapeutic Exercises *Care must be taken with all exercises to ensure external rotation and adduction of the hip do not occur at this stage.* • Slow, controlled leash walks, 10-15 working up to 20 min TID-QID on flat surface or mild incline • PROM, bicycling, and flexion/extension stretches of affected hip and other joints as needed 10-15 reps SID-BID. *It is critical to maintain movement in the sagittal plane* • Land Treadmill: 5 minutes SID walking on flat surface working up to slight inclines • Aquatic Therapy: Underwater Treadmill (UWTM) 10-20 min SID-BID at a slow, steady pace with support as needed • Balance on a 360-degree balance board with hindlimbs at a normal or slightly increased width (never adducted) 5-10 min SID-BID • Cavaletti Rails 5-10 laps BID • Sit-to-Stand 5-10 reps BID • Stair climbing 1-2 flights BID *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in over-exertion and pain early in the program* Modalities • Heat therapy at coxofemoral joint and thigh muscles 10 min BID-TID before exercise • Therapeutic ultrasound (continuous) to coxofemoral joint PRN • Therapeutic laser hip PRN • Cryotherapy at coxofemoral joint 20 min PRN after exercise Medications • NSAIDs/other analgesics PRN	Therapeutic Exercises *Care must be taken with all exercises to ensure external rotation and adduction of the hip do not occur at this stage.* • Slow, controlled leash walks, 10-15 working up to 20 min TID-QID on flat surface or mild incline • PROM, bicycling and flexion/extension stretches of affected hip and other joints as needed 10-15 reps SID-BID. *It is critical to maintain movement in the sagittal plane* • Balance on a moderately unstable surface with hindlimbs at a normal or slightly increased width (never adducted) 5-10 min SID-BID • Sit-to-stand 5-10 reps BID • Stair climbing 1-2 flights BID • Light jogging 1-2 min per day *Caution: Choose only 2-4 exercises per session. Doing all exercises may result in over-exertion and pain early in the program* Modalities • Heat therapy at coxofemoral joint and thigh muscles 10 min BID-TID before exercise • Cryotherapy at coxofemoral joint 20 min PRN after exercise Medications • NSAIDs/other analgesics PRN	• Goniometry—Hip ROM and other joints if applicable • Thigh muscle circumference (re-evaluate every 2 weeks) • Response to activity and subjective pain level • Lameness score at a stance, walk and trot • Weight	• Consistent weight-bearing on affected limb on all strides at a walk, early weight bearing at a trot • Minimal pain with moderate activities • Hip PROM: 50-55 degrees flexion and 140-150 degrees extension

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Hip Luxation—Craniodorsal, Closed Reduction**—cont'd

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Consistent Weight-Bearing at a Trot Goals Minimal to no pain with intense activity Normal weight-bearing of affected limb at all strides Normal hip range of motion Maintain hip in reduction through all movements Normal muscle mass	Eight to twelve weeks post-reduction	Therapeutic Exercises • Slow, controlled leash walks, 10-15 working up to 20 min TID-QID on flat surface or mild incline • PROM, bicycling and flexion/extension stretches of affected hip and other joints as needed 10-15 reps SID-BID. *It is critical to maintain movement in the sagittal plane* • Aquatic Therapy: Underwater Treadmill (UWTM) 20-30 minutes at variable speeds • Land Treadmill: 5-10 minutes SID walking with inclines, working up to use of resistance bands and weights on affected limb as tolerated. Incorporate 1-2 minutes jogging. • Balance on a 360-degree balance board 10 min BID • "Limbo" or tunnel crawl 5-10 reps SID-BID • Cavaletti Rails 10-20 laps BID • Weave poles 5-10 laps of 6-8 poles BID, gradually decreasing distance and increasing speed of navigation • Sit-to-stand 10-20 reps BID • Stair climbing 3-5 flights BID *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in over-exertion and pain early in the program* Modalities • Heat therapy PRN before activity • Cryotherapy PRN after exercises	Therapeutic Exercises • PROM and flexion/extension hip stretches of affected limb PRN • Leash walks 20-30 min including up to 10-15 min of mild-moderate inclines, may use weights on affected limb or pulled with a harness as tolerated/required • Incorporate challenging surfaces to walks, eg snow, sand, across a foam mattress, when possible 3-5 min BID • Controlled ball-playing with gradually increasing times and distances • Sit-to-stand 10-20 reps BID • Light jogging 3-5 min per day • Stair climbing: 3-5 flights SID-BID *Caution: Choose only 2-4 exercises per session. Doing all exercises may result in over-exertion and pain early in the program* Modalities • Heat therapy PRN before activity • Cryotherapy PRN after exercises	• Goniometry—Hip ROM and other joints if applicable • Thigh muscle circumference (re-evaluate every 2 weeks) • Response to activity and subjective pain level • Lameness score at a stance, walk, and trot • Weight	• Consistent weight-bearing on affected limb on all strides at a trot • Minimal pain with moderate to intense activities • Hip PROM normal

*This protocol is for use by licensed personnel who have been trained in canine rehabilitation. The therapist must keep in mind that these timelines are intended as guidelines and may be influenced by many factors affecting individualized patient care.

**Closed reduction is not indicated in cases of hip dysplasia, femoral head fracture, or significant osteoarthritis of the hip. Chronic luxation warrants evaluation of the cartilage. If concurrent injuries preclude limb use, surgery may be required for earlier return of hip function.

Osteoarthritis

Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures
Therapeutic Exercises • Passive Range of Motion within comfortable range of affected joint(s), 15-20 repetitions, SID-BID • Balance/weight shifting on surface with stability appropriate to the patient's capability 5-10 min BID • Controlled stair climbing 1-3 flights per session BID-TID (to add to program only as appropriate) • Sit-to-stand exercises (if pelvic limb(s) affected) 10-15 reps BID • Cavaletti Rails 10-15 reps BID • Treadmill Walking or Jogging Downhill (for OA involving pelvic limbs) or Uphill (for OA involving thoracic limbs)—5-10% decline walking at a slow-to-medium walking speed, 5 min SID-TID • Aquatic Therapy: Underwater Treadmill Walking 5-10 min working up to 20-30 min SID-BID depending on patient tolerance and muscle strength/endurance • Swimming 5 working up to 20 minutes 2-4 times per week *The mainstay of exercises for osteoarthritic patients is low impact, frequent activity of shorter duration; activity should not be of sufficient duration to exacerbate the patient's lameness. No more than 2-4 exercises should be performed per session in order to prevent pain due to excessive activity. At least two days of rest each week are recommended, in which exercises should consist only of PROM, stretching, and short leash walks*	Therapeutic Exercises • Passive Range of Motion within comfortable range of affected joint(s), 15-20 repetitions, SID-BID • Balance/weight shifting on surface with stability appropriate to the patient's capability 5-10 min BID • Controlled leash walks with mild inclines 10-30 minutes as tolerated TID-QID • Controlled stair or ramp climbing 1-3 flights per session BID-TID (to add to program only as appropriate) • Walking on the side of inclines with affected side higher 5 min BID • Sit-to-stand exercises (if pelvic limb(s) affected) 10-15 reps BID • Trotting on lead (as tolerated, to add to program only if appropriate) 2-5 min SID *The mainstay of exercises for osteoarthritic patients is more frequent activity of shorter duration; activity should not be of sufficient duration to exacerbate the patient's lameness. No more than 2-4 exercises should be performed per session in order to prevent pain due to excessive activity. At least two days of rest each week are recommended, in which exercises should consist only of PROM, stretching, and short leash walks*	• Goniometric range of motion measurements for affected joint(s) • Bilateral limb circumference • Force plate evaluation • Stance analysis • Subjective gait analysis (using tools such as VAS) • Individualized functional measures set for each patient at the beginning of treatment, ie:

Continued

Osteoarthritis—cont'd

Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures
Modalities • Massage: effleurage and massage of peri-articular muscle groups of affected limbs: 10 minutes at the start of each session • Transcutaneous electrical stimulation across affected joint(s) for pain relief (15-20 min SID) • NMES for muscle strengthening (15 min SID PRN) • Therapeutic Laser 10 J/cm^2 to affected joint(s) • Extracorporeal shock wave treatment • Cryotherapy (15-20 min BID-TID after exercise sessions) *Cryotherapy may also be utilized prior to exercise and stretching to improve pain-free range of motion* Medications • Injectable polysulfated glycosaminoglycan agents • Oral chondroprotective agents • Omega-3 Fatty Acids (EPA/DHA) • NSAIDs PRN, ideally no more often than every other day long term • Opioids/Acetaminophen/NMDA Antagonists • Intra-articular corticosteroids; or hyaluronic acid, platelet rich plasma, and/or stem cell therapy Other • Weight management or weight loss program as appropriate with goal of BCS 4/9 to 5/9	Modalities • Massage: effleurage and massage of peri-articular muscle groups of affected limbs: 10 minutes before PROM and exercises • Cryotherapy (15-20 min BID-TID after exercise sessions) *Cryotherapy may also be used prior to exercise and stretching to improve pain-free range of motion* Medications • Injectable polysulfated glycosaminoglycan agents • Oral chondroprotective agents • Omega-3 Fatty Acids (EPA/DHA) • NSAIDs PRN, ideally no more often than every other day long term • Opioids/Acetaminophen/NMDA Antagonists Other • Weight management or weight loss program as appropriate with goal of BCS 4/9 to 5/9	• Difficulty rising from a sit or lie down position • Difficulty navigating stairs • Weight

This protocol is for use only by licensed personnel who have been trained in canine rehabilitation.
The therapist must keep in mind that these timelines are intended as guidelines and may be influenced by many factors affecting individualized patient care.
Osteoarthritis is a disease characterized by exacerbation and remission. The rehabilitation program should be appropriately adjusted to accommodate the varying phases/degrees of this condition.

Patellar Luxation Repair

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Step 1 Postoperative to Early Toe-Touching at a Walk Goals: Pain control Swelling and edema control Maintain/ improve stifle postoperative ROM	Immediate to 48-72 hours postoperatively	Therapeutic Exercises • Slow leash walking only to go outside, no more than five minutes at a time • Gentle PROM affected stifle within easy comfortable range, 8-10 repetitions BID-TID *NOTE: It is critical that motion is limited to the sagittal plane.* Modalities • Gentle massage (effleurage, thigh muscles) • Transcutaneous electrical stimulation for pain relief (15-20 min SID-TID) • Cryotherapy to affected stifle (15-20 min BID-TID) • Therapeutic Laser SID Medications • NSAIDs/other analgesics • Oral chondroprotective agents/ injectable polysulfated glycosaminoglycans if patellofemoral OA exists	Inpatient status preferred during this phase If home: Therapeutic Exercises • Slow leash walking only to go outside, no more than five minutes at a time Modalities • Gentle massage (effleurage) • Cryotherapy to affected stifle (15-20 min BID-TID) Medications • NSAIDs/other analgesics • Oral chondroprotective agents/injectable polysulfated glycosaminoglycans if patellofemoral OA exists	• Postoperative bilateral stifle ROM (other joints if applicable)—goniometry • Postoperative bilateral thigh circumference • Response to activity and subjective pain level • Gait evaluation at a walk • Weight	• Consistent toe-touching on operated limb • Adequate resting analgesia. • Decreased perioperative swelling and no incisional drainage. • Comfortable ROM 70-90 degrees flexion, 120-135 degrees extension
Step 2 Early Weight-Bearing Goals: Continue pain control Reduce swelling Improve stifle ROM	48-72 hours to two weeks postoperatively	Therapeutic Exercises • Slow, steady leash walking 5 working up to 10 minutes at a time with sling support • Gentle PROM affected stifle within easy comfortable range, 10 repetitions BID-TID. Include hip/tarsal PROM of operated limb. *NOTE: It is critical that motion is limited to the sagittal plane.*	Therapeutic Exercises • Slow, steady leash walking 5 working up to 10 minutes at a time with sling support • Balance/weight-shifting exercises on a semi-firm surface 5-10 min BID-TID	• Response to activity and subjective pain level • Gait evaluation at walk and trot • Goniometry—Stifle ROM and other joints if applicable	• Consistent partial weight-bearing with each stride on operated limb • Minimal pain with light activities

Continued

Patellar Luxation Repair—cont'd

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Increase toe-touching weight-bearing Prevent/minimize muscle atrophy		• Balance/weight-shifting exercises on a bi-directional board or soft foam pad 5-10 min BID-TID Modalities • Heat therapy prior to PROM exercises 10 min, only if acute inflammation is absent • Gentle massage (effleurage, thigh muscles) prior to PROM • Transcutaneous electrical stimulation across stifle for pain relief (15-20 min SID-TID) • NMES 15 min for affected quadriceps and hamstring muscle group strengthening SID • Cryotherapy to affected stifle (15-20 min PRN) • Therapeutic Laser SID Medications • NSAIDs/other analgesics • Oral chondroprotective agents/injectable polysulfated glycosaminoglycans if patellofemoral OA exists	Modalities • Gentle massage (effleurage) • Cryotherapy to affected stifle (15-20 min PRN) Medications • NSAIDs/other analgesics • Oral chondroprotective agents/injectable polysulfated glycosaminoglycans if patellofemoral OA exists	• Thigh circumference at 2 weeks postoperatively • Weight	• Incisional healing without complications • ROM 60-80 degrees flexion, 130-150 degrees extension
Step 3 Consistent Weight-Bearing at a Walk Goals: Achieve normal stifle ROM Improve weight-bearing at a walk	Two to five weeks postoperatively	Therapeutic Exercises • Slow consistent leash walking—add 5 min every week if progressing well BID-QID • PROM all joints of affected pelvic limb 10 repetitions BID-TID (PRN). *Maintain motion in the sagittal plane.* • Balance/weight-shifting exercises on a 360-degree balance board/unstable surface 5-10 min BID-TID	Weight loss program can be initiated at this phase if warranted (minimizes loss of lean tissue mass). Therapeutic Exercises • PROM all joints of affected pelvic limb 10 repetitions BID-TID (PRN). *Maintain motion in the sagittal plane.*	• Response to activity and subjective pain level • Gait evaluation at walk and trot • Goniometry—Stifle ROM and other joints if applicable	• Consistent weight-bearing on operated limb at walk • Early partial weight-bearing of operated limb at a trot • No evidence of pain with moderate activities

Early weight-bearing at a trot Improve operated limb muscle mass/strength	• Sit-to-stand exercises 5-10 reps BID. *Use a sling or begin with hindquarters on an elevated surface if necessary. Begin at 4 weeks* • Low Cavaletti rails 5-10 reps BID, beginning at 4 weeks • Aquatic Therapy: Underwater Treadmill (UWTM) Walking 5-10 min SID-BID at a slow, steady pace • Land Treadmill (LTM): 5-10 min SID-BID at a slow, steady pace at a level plane. *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program* Modalities • Heat therapy prior to PROM exercises 10 min • Gentle massage (effleurage, thigh muscles) prior to PROM • Transcutaneous electrical stimulation across stifle for pain relief (15-20 min SID-TID PRN) • NMES 15 min for affected quadriceps and hamstring muscle group strengthening PRN • Cryotherapy to affected stifle (15-20 min after exercises PRN) Medications • NSAIDs PRN • Oral chondroprotective agents/ injectable polysulfated glycosaminoglycans if patellofemoral OA exists	• Slow consistent leash walking—add 5 min every week if progressing well BID-QID • 5 minutes of gentle uphill inclines *added to walks* • Sit-to-stand exercises 5-10 reps BID. *Use a sling or begin with hindquarters on an elevated surface if necessary* Modalities • Heat therapy prior to PROM exercises 10 min • Gentle massage (effleurage, thigh muscles) prior to PROM • Cryotherapy to affected stifle (15-20 min after exercises PRN) Medications • NSAIDs—PRN • Oral chondroprotective agents/injectable polysulfated glycosaminoglycans if patellofemoral OA exists	• Reassess thigh circumference at 3 and 5 weeks postoperatively • Weight	• Stifle PROM 40-60 degrees flexion, >150 degrees extension

Continued

Patellar Luxation Repair—cont'd

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Step 4 Consistent Partial Weight-Bearing at a Trot Goals: Normal limb use at a walk Improve ability to use the limb at a trot Improve muscle mass and strength Improve stamina and endurance	Five to eight weeks postoperatively	Therapeutic Exercises • PROM/stretching all joints of affected pelvic limb 10 repetitions BID-TID (PRN). *Maintain motion in the sagittal plane.* • Balance/weight-shifting exercises on a 360-degree balance board/unstable surface 5-10 min BID-TID • Sit-to-stand exercises 10-15 reps BID • Cavaletti rails 10-15 reps BID • Weave poles (decreasing distance and increasing speed adjusted to performance) 10-15 reps BID • Aquatic Therapy: Underwater Treadmill (UWTM) walking 15-20 min SID-BID • Swimming 5-10 min SID • Land Treadmill (LTM): 5-10 min SID-BID at a mild incline. Resistance band use (*in sagittal plane ONLY*) and leg weights as applicable for muscle mass/strength improvement in the operated limb	Continue weight loss program if needed Therapeutic Exercises • PROM/stretching all joints of affected pelvic limb 10 repetitions PRN. *Maintain motion in the sagittal plane.* • Controlled leash walking—add 5 min every week if progressing well BID-QID • 5 minutes of moderate uphill inclines on walks • 5 minutes circling, increasing tightness of circles every 4-5 sessions *added to walks* • 2-3 minutes trotting added to walks • "Dancing" exercises 5-10 reps of 5-10-second intervals BID-TID if acceptable to owner • Walking up and down stairs, 1-2 flights BID-TID • Sit-to-stand exercises 10-15 reps BID	• Response to moderate/heavy activity and subjective pain level • Goniometry—Stifle ROM and other joints if applicable • Gait evaluation at walk and trot • Reassess thigh circumference at 7-8 weeks postoperatively • Weight	• Minimal to no lameness at a walk. • Consistent partial weight-bearing at a trot of normal speed • No evidence of pain with activities • Normal stifle range of motion

		Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program Modalities • Heat therapy prior to exercises 10 min PRN • NMES 15 min for affected quadriceps and hamstring muscle group strengthening PRN • Cryotherapy to affected stifle (15-20 min after exercises PRN) Medications • NSAIDs PRN • Oral chondroprotective agents/ injectable polysulfated glycosaminoglycans if patellofemoral OA exists	*Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program* Modalities • Heat therapy prior to exercises 10 min PRN • Cryotherapy to affected stifle (15-20 min after exercises PRN) Medications • NSAIDs—PRN • Oral chondroprotective agents/injectable polysulfated glycosaminoglycans if patellofemoral OA exists		
Step 5 Trotting at Speed with Minimal to No Lameness Goals: Continue improving speed at a lope and slow gallop Continue improving muscle mass, strength, and power Continue improving stamina and endurance	Eight weeks postoperatively and beyond	Therapeutic Exercises • Walking up and down stairs, 3-5 flights BID-TID building reps weekly as tolerated • Land treadmill 10-15 minutes with inclines, adding 2-3 minutes of jogging and increasing jogging time weekly as tolerated. Add leg weights and/or resistance bands as needed for muscle mass/strength development. • Sit-to-stand exercises 15-20 reps BID • Cavaletti rails 10-15 reps BID, increasing height and decreasing distance as appropriate	Therapeutic Exercises • Leash walks up to 30 min BID-TID • Trotting up and down inclines, 3-5 min added to walks • Trotting in circles and figure 8s, 3-5 min BID • Controlled ball play, 5-10 min SID-BID • Walking up and down stairs, 3-5 flights BID-TID building reps weekly as tolerated • "Dancing" exercises 5-10 reps of 15-20-second intervals BID-TID if acceptable to owner	• Response to increasing activity and subjective pain level • Gait evaluation at walk and trot • Evaluate thigh circumference every 2 weeks until symmetry achieved • Weight	Progressions may be continued as long as lameness or pain with activity is not observed. If this occurs, decrease activity by 50% for 1 week and increase again by 10-20% weekly.

Continued

Patellar Luxation Repair—cont'd

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Return to as normal activity as possible		• Weave poles (decreasing distance and increasing speed adjusted to performance) 15-20 reps BID • Aquatic therapy—UWTM for conditioning, consider progressively increasing speed and use of jets when available • Swimming 10-15 minutes 3-4 times per week *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program* Modalities • Heat therapy prior to exercises 10 min PRN • Cryotherapy to affected stifle (15-20 min after exercises PRN) Medications • NSAIDs PRN • Oral chondroprotective agents/ injectable polysulfated glycosaminoglycans if patellofemoral OA exists	• Sit-to-stand exercises 15-20 reps BID *Caution: Choose only 2-3 exercises per session. Doing all exercises may result in lameness and pain early in the program* Modalities • Heat therapy prior to exercises 10 min PRN • Cryotherapy to affected stifle (15-20 min after exercises PRN) Medications • NSAIDs PRN • Oral chondroprotective agents/injectable polysulfated glycosaminoglycans if patellofemoral OA exists		

This protocol is for use only by licensed personnel who have been trained in canine rehabilitation.

*The therapist must keep in mind that these timelines are intended as guidelines and may be influenced by many factors affecting individualized patient care.

Thoracolumbar IVDE (postoperative)

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Rest Goals: Pain control Appreciable hindlimb tone Weight-bearing in standing with minimal support Minimize hindlimb muscle atrophy Independent urination and defecation	Immediate to five-seven days postoperatively	Therapeutic Exercises • Assisted standing with abdominal support as needed on a stable, high-traction surface (*beginning 24 hours postoperatively*) • PROM with bicycling movements all joints of pelvic limbs (10-15 reps BID-TID) Modalities • TENS, premodulated or interferential electrical stimulation across incision site, 15-20 min SID • NMES to hindlimb musculature for contraction 15 min SID • Cryotherapy at thoracolumbar incision site, 20 min BID-TID • Gentle massage of hindlimb musculature +/– tapotement BID as tolerated • Therapeutic laser therapy of epaxial musculature SID PRN Other • Bladder expression TID-QID PRN • Use of soft bedding with rotation every 4-6 hours to prevent decubital ulcers	Therapeutic Exercises • Assisted standing with abdominal support as needed on a stable, high-traction surface (*beginning 24 hours postoperatively*) • PROM with bicycling movements all joints of pelvic limbs (10-15 reps BID-TID) Modalities • Cryotherapy at thoracolumbar incision site, 20 min BID-TID • Effleurage BID-TID Medications • NSAIDs (if corticosteroids are not used)/opioids • NMDA receptor antagonists • Muscle relaxants BID-TID PRN Other • Bladder expression TID-QID PRN • Use of soft bedding with rotation every 4-6 hours to prevent decubital ulcers	• Subjective pain level • Hindlimb muscle tone • Degree of support required in standing	• Postoperative comfort • Incisional integrity maintained • Standing in weight-bearing with minimal support

Continued

Thoracolumbar IVDE (Postoperative)—cont'd

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Weight Support to Initial Motor Function Goals: Continued pain control Full weight-bearing in standing Minimized hindlimb muscle atrophy Movement of all four limbs at a slow, supported walk Self-rotation	One to two weeks postoperatively	Therapeutic Exercises • Assisted standing and weight-shifting/rhythmic stabilization, gradually decreasing support (eg physioball beneath abdomen, standing in water) • PROM with bicycling movements all joints of pelvic limbs (10-15 reps BID-TID) • Aquatic therapy: Swimming 2-5 minutes working up to 10 minutes with supported standing during breaks • Aquatic therapy: Underwater Treadmill (UWTM) walking, begin at 3-5 min up to 10-12 min. Ensure incision is sealed before beginning aquatic therapy • Turning exercises during standing with positive reinforcement held at various locations (concentrate efforts on the weaker side) Modalities • Gentle massage of hindlimb musculature BID-TID • NMES to hindlimb musculature for contraction 15 min EOD-SID PRN	Therapeutic Exercises • Assisted standing and weight-shifting/rhythmic stabilization, gradually decreasing support (eg physioball beneath abdomen) • PROM with bicycling movements all joints of pelvic limbs (10-15 reps BID-TID) • Slow leash walking with sling support 5 minutes TID Modalities • Gentle massage of hindlimb musculature BID-TID • Cryotherapy at thoracolumbar incision site, 20 min PRN after exercise Medications • NSAIDs (if corticosteroids are not used)/opioids • NMDA receptor antagonists Other • Bladder expression TID-QID PRN • Use of soft bedding with rotation every 4-6 hours to prevent decubital ulcers	• Response to activity and subjective pain level • Length of time with unsupported standing • Number of steps in which gait patterning is used • Hindlimb thigh circumference/ muscle mass	• Pelvic limb voluntary motor activity • Minimal pain with light activities • Unsupported standing

		• NMES to epaxial muscles for contraction 15 min EOD-SID PRN • Cryotherapy at thoracolumbar incision site, 20 min PRN after exercise Other • Bladder expression TID-QID PRN • Use of soft bedding with rotation every 4-6 hours to prevent decubital ulcers			
Initial Motor Function to Good Motor Function with Proprioceptive Deficits <u>Goals:</u> Improve proprioception during walking activity Maintain normal pelvic limb ROM Increase pelvic limb muscle mass Maintenance of balance during turns and on a moderately unstable surface	Two to four weeks postoperatively	Therapeutic Exercises • Assisted standing and weight-shifting on a bi-directional balance board • PROM with bicycling movements all joints of pelvic limbs (10-15 reps PRN based on ROM) • Aquatic therapy: Swimming 10-15 minutes with supported standing during breaks • Aquatic therapy: Underwater Treadmill (UWTM) walking, 10-15 minutes SID • Gait patterning on a ground treadmill, 5-10 minutes SID with resistance band use PRN • Turning exercises during standing with positive reinforcement held at various locations (concentrate efforts on the weaker side) • Turning exercises during walking: weave poles gradually decreasing in distance 5-10 reps SID-BID	Therapeutic Exercises • Assisted standing and weight-shifting on a moderately unstable surface • PROM with bicycling movements all joints of pelvic limbs (10-15 reps PRN based on ROM) • Slow leash walking with sling available 10 minutes BID-TID • Turning exercises during standing with positive reinforcement held at various locations (concentrate efforts on the weaker side) • Turning exercises during walking: weave poles gradually decreasing in distance 5-10 reps SID-BID Modalities • Cryotherapy at thoracolumbar incision site, 20 min PRN after exercise Medications • NSAIDs PRN • NMDA receptor antagonists	• Response to activity and subjective pain level • Length of time with unsupported standing • Length of time walking in aquatic therapy and on dry land • Frequency of steps in which appropriate proprioception is used • Height of Cavaletti rails navigated • Pelvic limb thigh circumference/ muscle mass	• Consistent strong pelvic limb movements at a walk • Minimal pain with moderate activities

Continued

Thoracolumbar IVDE (postoperative)—cont'd

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
		• Low Cavaletti rails 5-10 reps SID-BID *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program* Modalities • NMES to hindlimb musculature for contraction 15 min EOD-SID PRN • NMES to epaxial muscles for contraction 15 min EOD-SID PRN • Cryotherapy at thoracolumbar incision site, 20 min PRN after exercise			
Good motor function with mild proprioceptive deficits to near normal gait Goals: Consistent proprioception with moderate- to high-intensity activity	Four to six postoperatively	Therapeutic Exercises • Assisted standing and weight-shifting on a 360-degree balance board • Aquatic therapy: Swimming 20-30 minutes with supported standing during breaks • Aquatic therapy: Underwater Treadmill (UWTM) walking, 15-20 minutes	Therapeutic Exercises • Assisted standing and weight-shifting on an unstable surface • Turning exercises during walking: weave poles gradually decreasing in distance 5-10 reps SID-BID • Stair Climbing 1 working up to 5 flights SID-BID • Sit-to-stand exercises, rising from an elevated surface 5-10 reps SID-BID	• Response to activity and subjective pain level • Length of time with unsupported standing • Walking endurance • Tightness of turns in which balance is maintained	• Consistent pelvic limb proprioception at a walk • Minimal to no pain with extensive activities • Normal gait patterning at a trot

Pelvic limb muscle mass approaching normal Maintain balance during turns and negotiating obstacles while walking		• Gait patterning on a ground treadmill, 5-10 minutes with resistance band use PRN • Turning exercises during walking: weave poles gradually decreasing in distance 5-10 reps SID-BID • Cavaletti rails 5-10 reps SID-BID • Stair Climbing 1 working up to 5 flights SID-BID • Sit-to-stand exercises, rising from an elevated surface 5-10 reps SID-BID *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program* Modalities • Cryotherapy at thoracolumbar incision site, 20 min PRN after exercises	• Leash walking 10-15 min BID-TID with 5 minutes inclines/declines Modalities • Cryotherapy at thoracolumbar incision site, 20 min PRN after exercise Medications • NSAIDs PRN • NMDA receptor antagonists	• Frequency of steps in which appropriate proprioception is used • Height of Cavaletti rails navigated • Pelvic limb thigh circumference/ muscle mass	
Near Normal Gait to Normal Gait <u>Goals:</u> Elimination of proprioceptive deficits during high-intensity activity	Six or more weeks postoperatively	Therapeutic Exercises • Assisted standing and weight-shifting on a 360-degree balance board • Aquatic therapy: Swimming 20-30 minutes • Aquatic therapy: Underwater Treadmill (UWTM) walking, 20-30 minutes at variable speeds	Therapeutic Exercises • Assisted standing and weight-shifting on an unstable surface • Turning exercises with weave poles 10-20 reps SID-BID at a trot • Stair Climbing 5-10 repetitions SID-BID • Sit-to-stand exercises 10-20 reps SID-BID	• Response to activity and subjective pain level • Walking and trotting endurance • Tightness of turns in which balance is maintained	• Minimal to no pain with extensive activities • Normal gait and proprioception at a trot and at faster speeds

Continued

Thoracolumbar IVDE (postoperative)—cont'd

Phase	Expected Timeframe*	Rehabilitation Clinic Program	Home Program	Outcome Assessment Measures	Criteria for Movement to Next Phase
Normal pelvic limb muscle mass Maintain balance during turns and negotiating obstacles while trotting		• Gait patterning on a ground treadmill, 5-10 minutes at a trot • Turning exercises with weave poles 10-20 reps SID-BID at a trot • Cavaletti rails 10-20 reps SID-BID at a trot • Stair Climbing 5-10 repetitions SID-BID • Sit-to-stand exercises 10-20 reps SID-BID • Controlled ball play *Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program*	• Controlled ball play • Leash walking 20-30 minutes BID with 10 minutes variable inclines and 5-10 minutes working up to longer periods trotting	• Height and distance of Cavaletti rails navigated • Pelvic limb thigh circumference/ muscle mass	

This protocol is for use by licensed personnel who have been trained in canine rehabilitation.

*The therapist must keep in mind that these timelines are intended as guidelines. Rate of recovery will vary widely among patients for reasons that include status of nociception and function prior to and immediately after surgery.

Total Hip Replacement

Phase	Expected Timeframe*	Rehabilitation Clinic Program**	Home Program**	Outcome Assessment Measures	Criteria for Movement to Next Phase
Non-Weight-Bearing to Toe-Touching Goals: Pain control Swelling and edema control Early limb use Slow/minimize muscle atrophy	Immediate to 2 weeks postoperatively	Therapeutic Exercises • Slow, gentle PROM for operated limb (8-10 reps flexion and extension BID). *Sagittal plane only—abduction and adduction of the hip must be STRICTLY AVOIDED* • Slow leash walking with firm sling support, only to go outside (up to 5 minutes, TID-QID) • Balance exercises on a soft foam pad or bi-directional balance board for early toe-touching weight-bearing (begin at early toe-touching 3-5 days after surgery, use assisted sling support) Modalities • Gentle massage around the operated hip, thigh, and lumbosacral regions, avoiding the incision • Transcutaneous electrical stimulation to hip for pain relief (15-20 min SID-BID) • Cryotherapy to hip (15-20 min TID for 48-72 hours then PRN after light activity)—first session immediately postoperatively • Therapeutic laser therapy SID • NMES—quadriceps and hamstrings muscle groups (15 min SID), begin 5-7 days postoperatively after acute inflammation has been addressed Medications • NSAIDs • Opioids • NMDA Antagonists	Therapeutic Exercises • Slow, gentle PROM for stifle and tarsus of the operated limb (8-10 reps flexion and extension TID-QID) • Slow leash walking with firm sling support, only to go outside (up to 5 minutes, TID-QID) • Weight-shifting exercises on a stable surface for early toe-touching weight-bearing (begin at early toe-touching 3-5 days after surgery, use assisted sling support) Modalities • Effleurage, avoiding incision, BID-TID • Cryotherapy to hip (15-20 min TID for 48-72 hours then PRN after light activity) Medications • NSAIDs • Opioids • NMDA Antagonists	• Postoperative hip sagittal plane PROM and other joints as applicable via goniometry • Postoperative bilateral thigh circumference • Response to minimal activity and subjective pain level • Weight	• Early toe-touching • Adequate resting analgesia • Decreased perioperative swelling and no incisional drainage.

Continued

Total Hip Replacement—cont'd

Phase	Expected Timeframe*	Rehabilitation Clinic Program**	Home Program**	Outcome Assessment Measures	Criteria for Movement to Next Phase
Toe-Touching to Consistent Partial Weight-Bearing at a Walk Goals: Continued pain control Prevention of further muscle atrophy, early strengthening Improved limb function at a walk	2-12 weeks postoperatively	Therapeutic Exercises • Stretching and PROM with bicycling of the operated limb (8-10 reps flexion and extension BID before activities). *Sagittal plane only—abduction and adduction of the hip must be STRICTLY AVOIDED* • Slow leash walking with firm sling support, 3-5 min working up to 10 min steady walking TID • Balance and weight-shifting exercises on a soft foam pad or bi-directional balance board (use assisted sling support) • Aquatic therapy: Underwater Treadmill (UWTM) walking: begin at 3-5 min working up to 10-15 min SID-BID. *Begin at 3-4 weeks postoperatively* • Low Cavaletti Rails: 8-10 reps BID. *Begin at 4-6 weeks postoperatively, working up to higher rails by 8-12 weeks postoperatively* • Sit-to-Stand with hindquarters on an elevated surface, 5-10 reps BID-TID. *Begin at 8-12 weeks postoperatively* *Caution: Choose only 1-2 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program*	Therapeutic Exercises • Stretching and PROM with bicycling of the operated limb (8-10 reps flexion and extension BID-TID before activities)—*abduction and adduction of the hip must be STRICTLY AVOIDED* • Slow leash walking with firm sling support, 3-5 min working up to 10 min steady walking TID • Balance and weight-shifting exercises on a semi-firm stable surface (use assisted sling support) • Flexor reflex exercises of operated hindlimb • Sit-to-Stand with hindquarters on an elevated surface, 5-10 reps BID-TID. *Begin at 8-12 weeks postoperatively* *Caution: Choose only 1-2 exercises per session. Doing all exercises may result in lameness and pain early in the program* Modalities • Cryotherapy to hip (15-20 min PRN after light activity)	• Goniometry—Hip ROM and other joints if applicable • Response to activity and subjective pain level • Bilateral thigh circumference • Lameness score at a stance and walk • Weight	• Consistent partial weight-bearing on operated limb on all strides at a walk • Minimal pain with light activities • Incisional healing without complications

		Modalities • Transcutaneous electrical stimulation to hip for pain relief (15-20 min SID-BID PRN) • Cryotherapy to hip (15-20 min PRN after light activity) • NMES—quadriceps and hamstrings muscle groups (15 min EOD-SID) Medications • NSAIDs SID-PRN • Chondroprotective agents as needed for other joints	Medications • NSAIDs SID-PRN • Chondroprotective agents as needed for other joints		
Consistent Weight-Bearing at a Walk to Early Trotting <u>Goals:</u> Muscle strengthening Normal limb function at a walk, improved function at faster gaits Near-normal hip PROM in flexion/ extension	12-16 weeks postoperatively	Therapeutic Exercises • Stretching of the operated limb (8-10 reps BID-TID before activities) • 3-5 min leash walk with 30 sec-1 min trotting as a warm-up • Land treadmill walking 5-10 min SID-BID on a flat surface, with resistance band applied in the sagittal plane to strengthen operated limb • Balance and weight-shifting exercises on a 360-degree balance board, 10 min SID-BID (have sling support available if needed) • Aquatic therapy: Underwater Treadmill (UWTM) walking: 15-20 min SID-BID • Swimming: 5-10 min working up to 20 min SID with breaks • Cavaletti Rails: 10-15 reps BID • Limbo crawling: 8-10 reps BID • Sit-to-Stand 10-15 reps BID-TID • Controlled stair walking on leash: 1-2 flights SID-BID	Therapeutic Exercises • Stretching of the operated limb (8-10 reps BID-TID before activities). • Slow leash walking 15-20 min BID. Incorporate 5 min light inclines and 3-5 min slow trotting on a flat surface • Balance and weight-shifting exercises on a semi-unstable surface (have sling support available if needed) • Sit-to-Stand 10-15 reps BID-TID • Controlled stair walking on leash: 1-2 flights SID-BID *Caution: Choose only 2-3 exercises per session. Doing all exercises may result in lameness and pain early in the program*	• Goniometry—Hip ROM and other joints if applicable • Bilateral thigh muscle girth • Response to activity and subjective pain level • Lameness score at a stance, walk, and trot • Weight	• Consistent weight-bearing on operated limb on all strides at a walk, consistent partial weight bearing at a trot • Minimal pain with moderate activities

Continued

Total Hip Replacement—cont'd

Phase	Expected Timeframe*	Rehabilitation Clinic Program**	Home Program**	Outcome Assessment Measures	Criteria for Movement to Next Phase
		Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program Modalities • Heat therapy to hip (10 min PRN with stretching before exercises) • Cryotherapy to hip (15-20 min PRN after exercises) Medications • NSAIDs PRN • Chondroprotective agents as needed for other joints	Modalities • Heat therapy to hip (10 min PRN with stretching before exercises) • Cryotherapy to hip (15-20 min PRN after exercises) Medications • NSAIDs PRN • Chondroprotective agents as needed for other joints		
Early to Consistent Weight-Bearing at a Trot Goals: Hindlimb muscular symmetry Normal limb function at a walk and trot Normal hip PROM	>16 weeks postoperatively	Therapeutic Exercises • Stretching of the operated limb (8-10 reps PRN before activities). • 5 min leash walk with 1-2 min trotting as a warm-up • Land treadmill walking 10-15 min SID-BID on a flat surface, with resistance band applied in the sagittal plane and leg weights as needed for atrophy on operated limb • Balance and weight-shifting exercises on a 360-degree balance board, 10 min SID-BID • Aquatic therapy: Underwater Treadmill (UWTM) walking: 15-20 min SID-BID, variable speeds • Swimming: 20-30 min SID with breaks • Cavaletti Rails: 10-15 reps BID • Limbo crawling: 10-15 reps BID	Therapeutic Exercises • Stretching of the operated limb (8-10 reps PRN before activities) • Slow leash walking 15-20 min BID. Incorporate 5-min moderate inclines and gradually increase slow trotting on a flat surface • Balance and weight-shifting exercises on a semi-unstable surface (leg weights as needed for atrophy on operated limb) • Sit-to-Stand 15-20 reps BID-TID • Controlled stair walking on leash: 3-5 flights SID-BID	• Goniometry—Hip ROM and other joints if applicable • Bilateral thigh muscle girth • Response to moderate-intense activity and subjective pain level • Lameness score at a stance, walk and trot • Weight	• Consistent weight-bearing on operated limb at all gaits • Minimal to no pain with moderate to extensive activities Hindlimb muscle subjective symmetry

- Sit-to-Stand 15-20 reps BID-TID
- Controlled stair walking on leash: 3-5 flights SID-BID

Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program.

Activity should continue to be gradually increased based on each patient's individual progress. Jumping and sharp turns should not be allowed until return to full function, strength, and endurance are observed.

Modalities

- Heat therapy to hip (10 min PRN with stretching before exercises)
- Cryotherapy to hip (15-20 min PRN after exercises)

Medications

- NSAIDs PRN

- Controlled ball play, 5-10 min SID-BID

Caution: Choose only 2-3 exercises per session. Doing all exercises may result in lameness and pain early in the program.

Activity should continue to be gradually increased based on each patient's individual progress. Jumping and sharp turns should not be allowed until return to full function, strength, and endurance are observed.

Modalities

- Heat therapy to hip (10 min PRN with stretching before exercises)
- Cryotherapy to hip (15-20 min PRN after exercises)

Medications

- NSAIDs PRN

*This protocol is for use by licensed personnel who have been trained in canine rehabilitation. The therapist must keep in mind that these timelines are intended as guidelines and may be influenced by many factors affecting individualized patient care.

**Strict exercise restriction, with the exception of controlled rehabilitation exercises, should be maintained for 8-12 weeks following this procedure.



APPENDIX 2

Joint Motions and Ranges

Joints	Joint Motions	Normal Range of Motion for Dogs (degrees)
Forelimb Joints		
Shoulder (including scapular motion)	Flexion	30-60
	Extension	160-170
	Abduction	40-50
	Adduction	40-50
	Internal rotation	40-50
	External rotation	40-50
Elbow	Flexion	20-40
	Extension	160-170
	Hyperextension or overextension	
Radioulnar	Pronation	40-50
	Supination	80-90
Carpus (Wrist)	Flexion	20-35
	Hyperextension	190-200
	Radial or medial deviation	5-15
	Ulnar or lateral deviation	10-20
Hip	Flexion	55
	Extension	160-165
	Abduction with a flexed hip	120 (Stifle at 90)
	Adduction with a flexed hip	65 (Stifle at 90)
	Abduction with an extended hip	85
	Adduction with an extended hip	63
	Internal rotation	55
	External rotation	50
Stifle (knee)	Flexion	45
	Extension	160-170
Talocrural, tarsocrural, ankle	Flexion	40
	Extension	170

NOTE: Presently, the tendency is to call the straight joint position 180° rather than 0° being established. Range of motion is expressed in degrees unless noted otherwise.

Goniometry of the Forelimb and Hindlimb

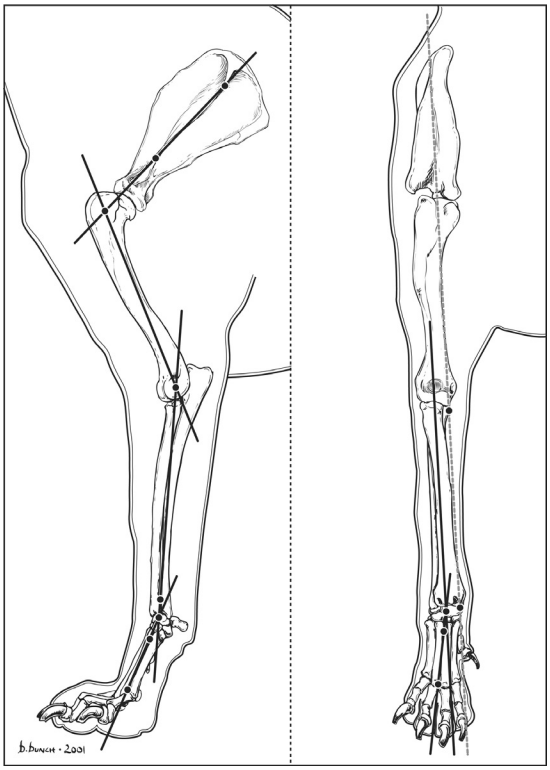


Figure 1 Goniometry of the forelimb. Carpal flexion and extension are measured as the angles formed by the long axis of metacarpal bones III and IV and the line joining the cranial to caudal midpoint of the antebrachium at the level of the ulnar styloid process and the lateral humeral epicondyle. Elbow flexion and extension are measured as the angles formed by the line joining the cranial to caudal midpoint of the antebrachium at the level of the ulnar styloid process and the lateral humeral epicondyle and a line joining the lateral epicondyle to the point of insertion of the infraspinatus muscle on the greater tubercle of the humerus. Shoulder flexion and extension are measured as the angles formed by the line joining the lateral humeral epicondyle and the point of insertion of infraspinatus muscle and the spine of the scapula. Carpal varus and valgus are measured as the angles formed by the long axis of metacarpals III and IV and the long axis of the medial border of the radius.

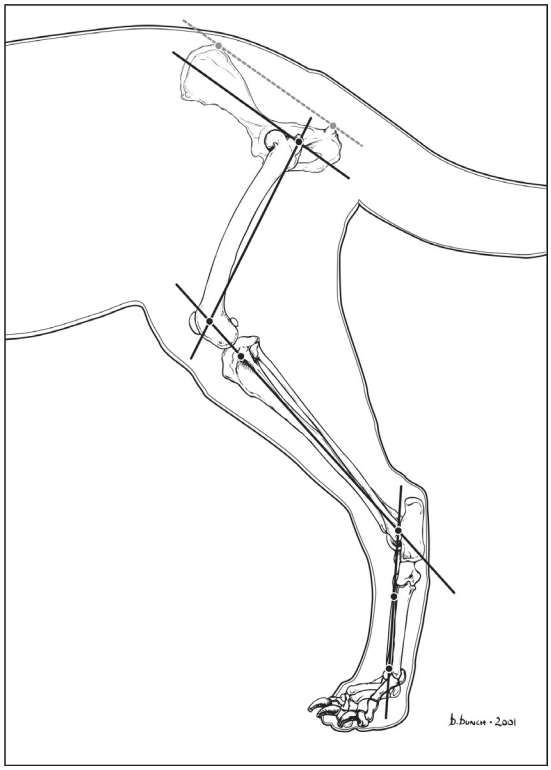


Figure 2 Goniometry of the hindlimb. Tarsal flexion and extension are measured as the angles formed by the long axis of metatarsal bones III and IV and the long axis of the tibial shaft. Flexion and extension of the stifle joint are measured as the angles formed by the long axis of the tibial shaft and the line joining the lateral femoral epicondyle and greater trochanter. Hip joint flexion and extension are measured as the angles formed by the line joining the lateral femoral epicondyle of the femur and greater trochanter and a line joining the tuber sacrale and ischiadicum.

The figures and Table 1 used by permission from Jaegger G, Marcellin-Little DJ, Levine D: Reliability of goniometry in Laborador retrievers, *Am J Vet Res* 63: 979-986, 2002

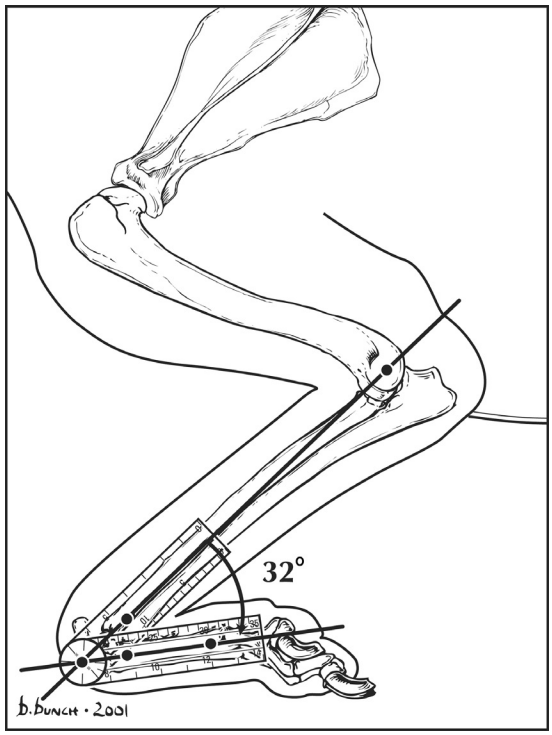


Figure 3 Carpal flexion.

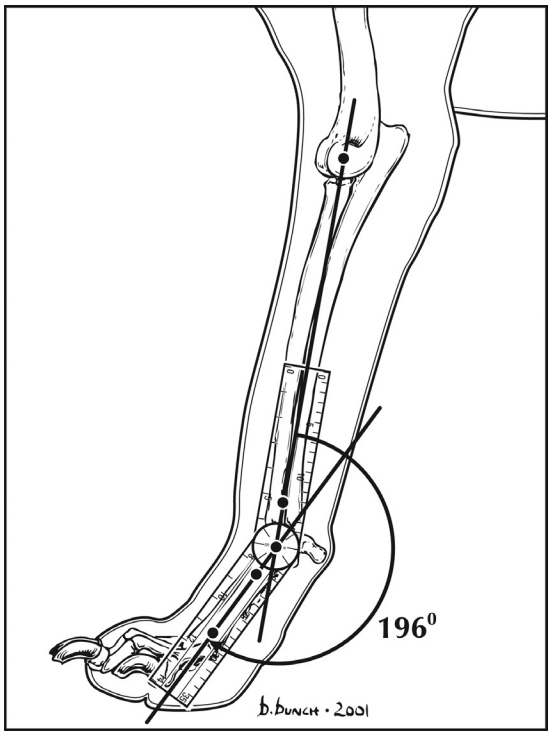


Figure 4 Carpal extension.

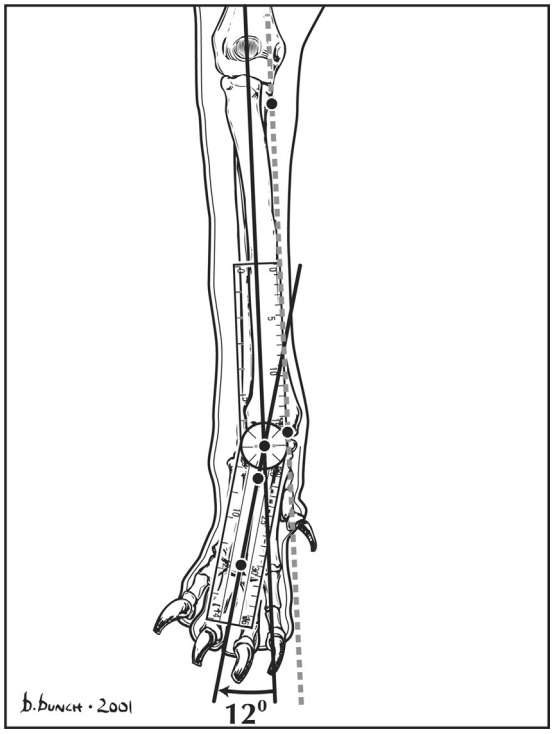


Figure 5 Carpal valgus.

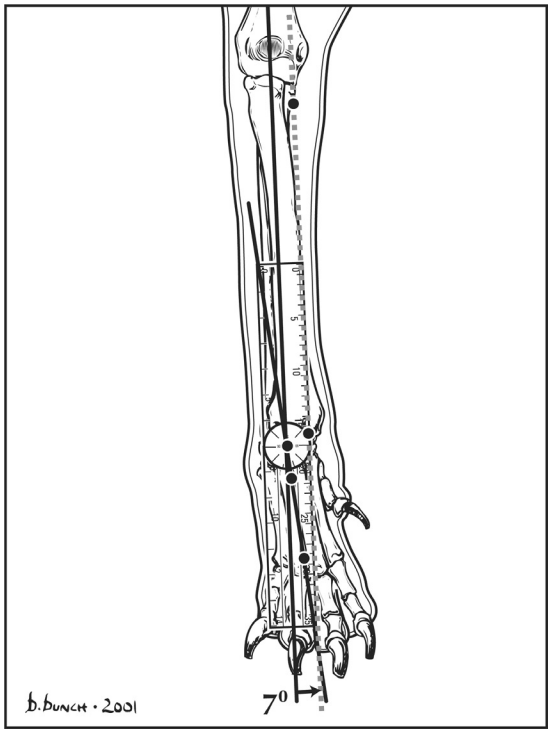


Figure 6 Carpal varus.

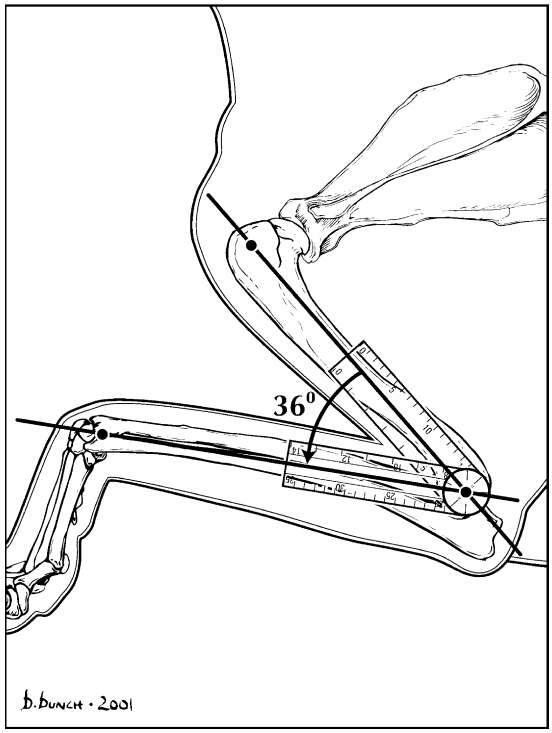


Figure 7 Elbow flexion.

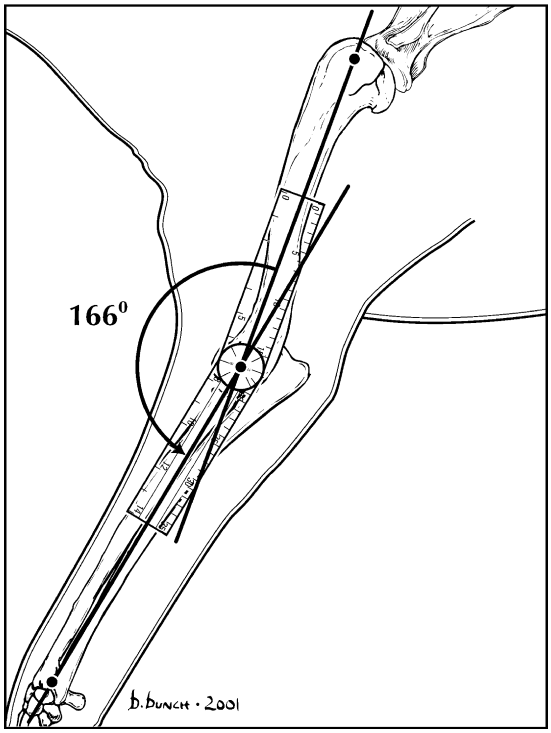


Figure 8 Elbow extension.

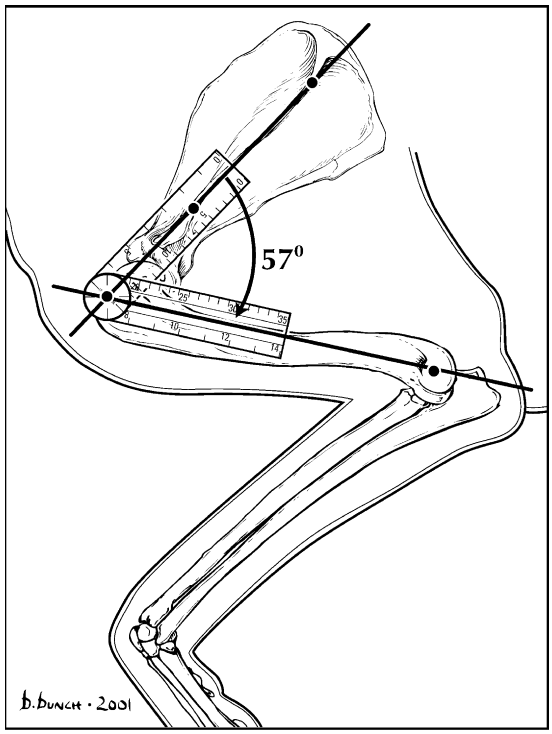


Figure 9 Shoulder flexion.

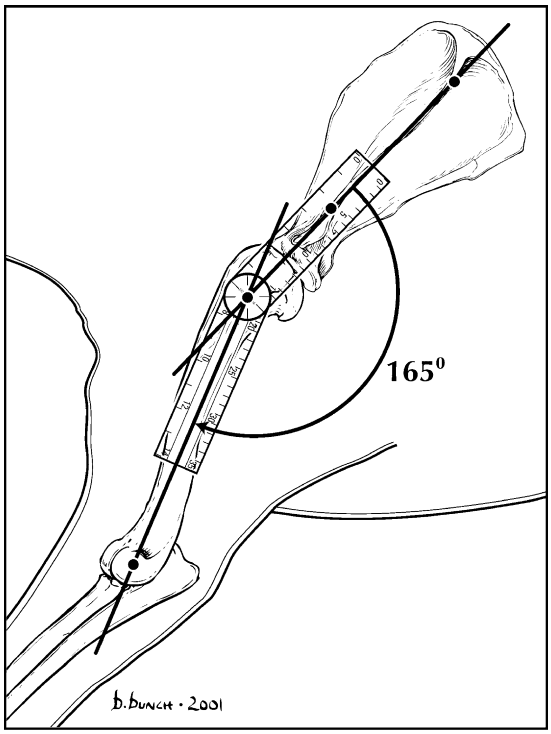


Figure 10 Shoulder extension.

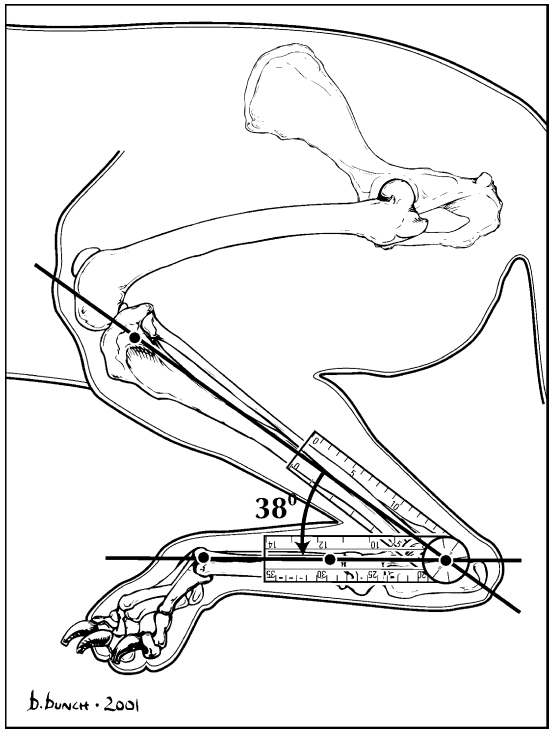


Figure 11 Tarsal flexion.

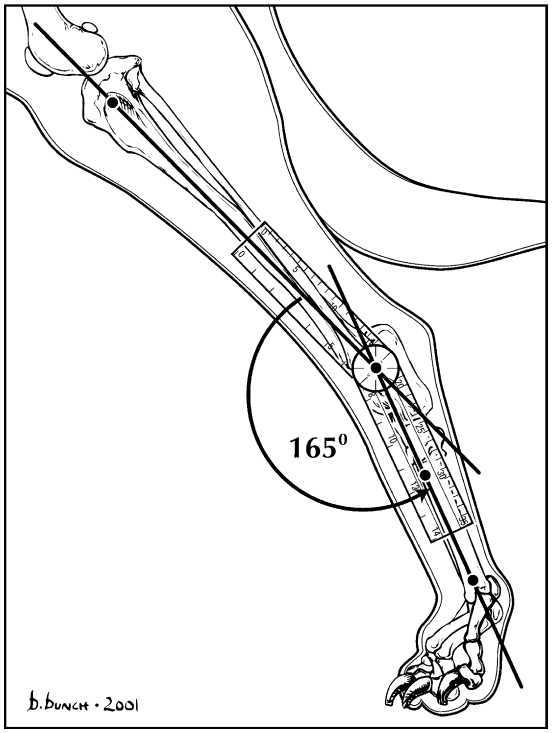


Figure 12 Tarsal extension.

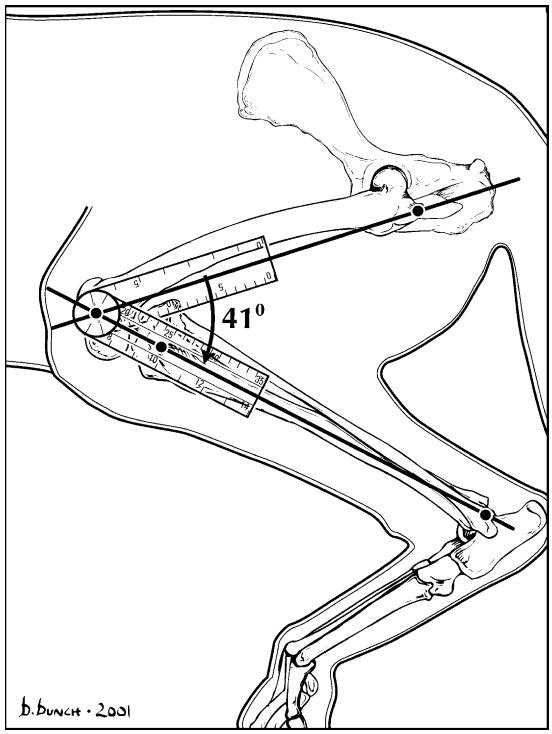


Figure 13 Stifle joint flexion.

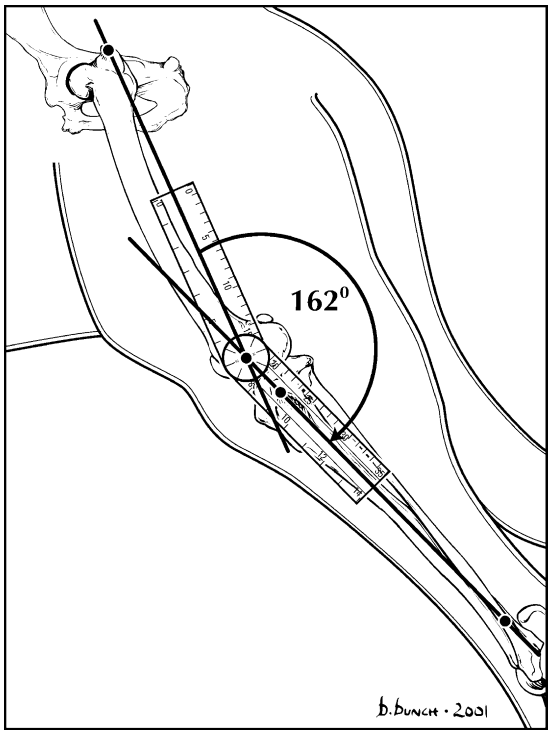


Figure 14 Stifle joint extension.

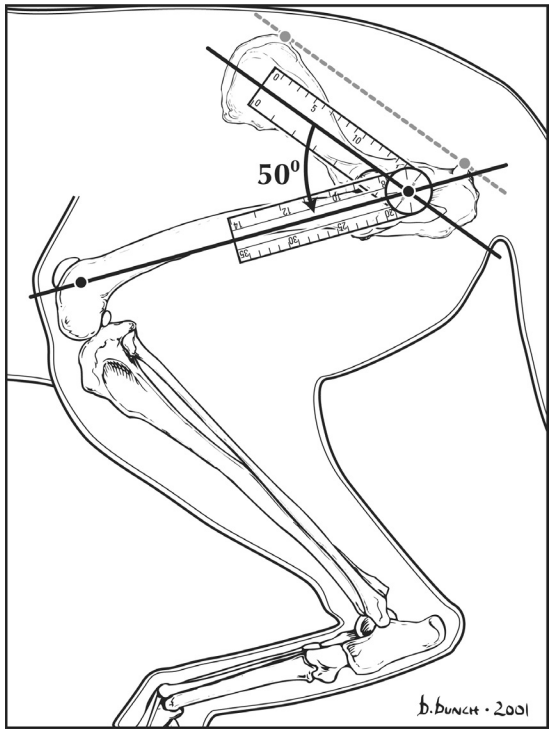


Figure 15 Hip joint flexion.

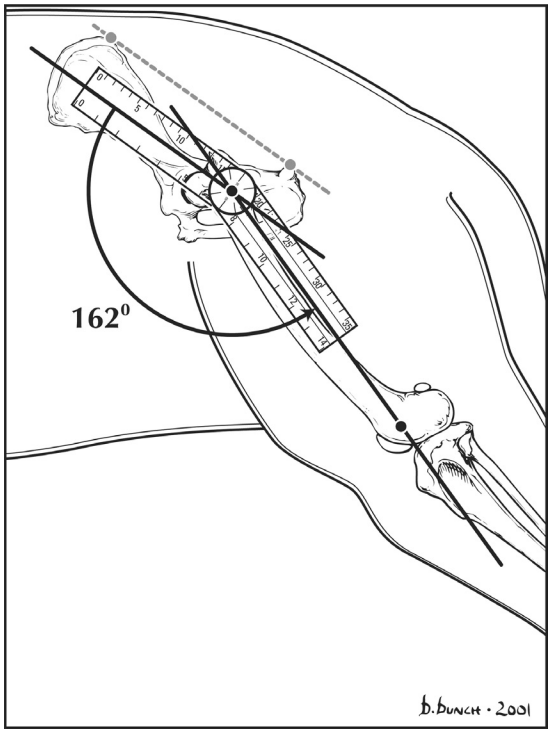


Figure 16 Hip joint extension.

Table 1 Range of Motion (degrees) of Various Appendicular Joints Measured by Goniometry in 16 Healthy Labrador Retrievers

Joint	Position	Mean	SD	95% CI of the Mean	Median
Carpus	Flexion	32	2	31-34	32
Extension	196	2	194-197	196	
Valgus	12	2	11-13	12	
Varus	7	1	6-8	7	
Elbow	Flexion	36	2	34-38	36
Extension	165	2	164-167	166	
Shoulder	Flexion	57	2	54-59	57
Extension	165	2	164-167	165	
Tarsus	Flexion	39	2	37-40	38
Extension	164	2	162-165	165	
Stifle	Flexion	42	2	40-43	41
Extension	162	3	160-164	162	
Hip	Flexion	50	2	48-52	50
Extension	162	3	160-164	162	

CI = Confidence interval.



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WEB APPENDIX 1

Comparative Myology of the Dog

Muscle	Correlated Human Muscle	Joint Action in the Forelimb	Main Cranial, Dorsal, or Proximal Attachment	Main Caudal, Ventral, or Distal Attachment	Fiber Direction from Proximal to Distal Attachment (listed in order of importance)	Innervation
FORELIMB						
Trunk to Forelimb						
Cutaneous trunci	No correlate; however, like the platysma in the neck	None: Twitch the skin over the scapular and shoulder area	In the superficial fascia of the dorsal, lateral, and ventral walls of thorax and abdomen	Medial side of the forelimb		Lateral thoracic nerve
Trapezius						
Cervical	Upper trapezius	Elevate the scapula cranially and dorsally; abduct the forelimb	Median or mid-dorsal raphe of the neck and supraspinous ligament	Dorsal aspect of the spine of the scapula	Caudoventral	Accessory nerve Cranial XI
Thoracic	Lower trapezius	Depress the scapula; abduct the forelimb	Supraspinous ligament and dorsal spinous processes of T3-T8 or T9	Middle part of the spine of the scapula	Cranioventral	Accessory nerve Cranial XI
The fibrous band in between the cervical and thoracic parts is in the position of the middle trapezius.						
Rhomboideus		Elevate the scapula and forelimb; pull the scapula to the trunk				Ventral rami of cervical spinal nerves
Rhomboideus capitis: two parts		Elevate the scapula and forelimb; pull the scapula to the trunk	Nuchal crest of the occipital bone	Cranial dorsal border of the scapula	Craniodorsal	
Rhomboideus cervicis	Rhomboid minor	Elevate the scapula and forelimb; pull the scapula to the trunk	Median fibrous raphe of neck, spinous processes of T1-T3	Caudal (base) of the scapula, medially and laterally	Craniodorsal	
Rhomboideus thoracis	Rhomboid major	Joint action in the forelimb	Spinous process of T4-T7	Caudal (base of scapula, medially and laterally)	Craniodorsal	

Serratus ventralis	Serratus anterior	Carry the trunk and shoulder forward and back, support the trunk, depress the scapula	Transverse processes of C3-C7 and ventral ribs 1-7 or 8	Medial scapula, dorsal one third	Craniodorsal and cranioventral	Ventral rami of cervical spinal nerves and long thoracic nerve
Brachiocephalicus: two parts	Two parts are divided by a fibrous clavicular intersection					Accessory nerve Ventral rami of cervical spinal nerves
Cleidobrachialis	Anterior deltoid	Extend the shoulder; advance the forelimb	Ventral aspect of the clavicle	Cranial surface, distal third of the humerus, between the biceps brachii medially and the brachialis laterally	Distomedial	
Cleidocephalicus—neck muscle		Draw the head to the side; extend the neck	Dorsal aspect of the clavicle	Cranial half middorsal fibrous raphe and nuchal crest of occipital bone	Dorsocranial	
Superficial pectoral: two parts—						
Descending pectoral portion	Part of pectoralis major	Adduct the shoulder, prevent abduction in weight-bearing; extend the shoulder; draw trunk sideward	Ventral sternum, most cranial aspect (ventral to the transverse pectoral muscle)	All but most distal part of crest of greater tubercle of humerus	Distolateral	Cranial pectoral nerves
Transverse pectoral portion	Part of pectoralis major	As above, plus flex or extend shoulder, depending on position	Ventral sternum, cranial half dorsal to the descending pectoral muscle	All but most distal part of crest of greater tubercle of humerus	Distolateral	Cranial pectoral nerves
Deep pectoral	Part of pectoralis major	Flex the shoulder; move the trunk cranially during weight-bearing; support the trunk with the serratus ventralis	Ventral aspect of the sternum, dorsal to the superficial pectoral muscle cranially	Major portion—lesser tubercle of humerus, aponeurosis to the greater tubercle and crest of humerus Caudal portion—medial brachial fascia	Craniodisto-lateral	Caudal pectoral nerves

Continued

Muscle	Correlated Human Muscle	Joint Action in the Forelimb	Main Cranial, Dorsal, or Proximal Attachment	Main Caudal, Ventral, or Distal Attachment	Fiber Direction from Proximal to Distal Attachment (listed in order of importance)	Innervation
Latissimus dorsi	Latissimus dorsi	Flex the shoulder; draw the trunk cranially in weight bearing; adduct the shoulder; support the trunk	Thoracolumbar fascia from spinous processes of most caudal seven or eight thoracic and lumbar vertebrae; most caudal two or three ribs	Teres major tuberosity on the humerus and teres major tendon; joins tip of the muscle cutaneous trunci laterally	Craniolatero-distal	Thoracodorsal nerve
SHOULDER: WITHIN FORELIMB						
Deltoideus: two parts	Deltoid			The two parts of the deltoid blend near the shoulder joint and attach to the deltoid tuberosity		
Spinous head	Posterior deltoid	Shoulder flexion	Scapular spine, blending with the infraspinatus		Distolateral	Axillary nerve
Acromial head	Middle deltoid	Shoulder abduction	Acromion		Laterodistal	Axillary nerve
Infraspinatus	Infraspinatus	Shoulder flexion or extension depending on position; shoulder abduction and lateral rotation; stabilize lateral shoulder	Infraspinous fossa	Lateral side of the greater tubercle of the humerus	Laterodistal	Suprascapular nerve
Teres minor	Teres minor	Shoulder flexion and lateral rotation	Infraglenoid tubercle	Infraglenoid tuberosity of the humerus	Laterodistal	Axillary nerve
Supraspinatus	Supraspinatus	Extend the shoulder; stabilizes and prevents collapse of the shoulder	Supraspinous fossa, wider and larger than the infraspinatus	Greater tubercle of the humerus by a thick tendon	Distally on the cranial aspect of the shoulder, then medially	Suprascapular nerve
Subscapularis	Subscapularis	Adduct, extends, and stabilizes the shoulder medially	Subscapular fossa	Lesser tubercle of the humerus	Craniodistal	Subscapular nerve

Teres major	Teres major	Shoulder flexion and medial rotation	Caudal angle and border of the scapula and caudal subscapularis	Teres major tuberosity of the humerus	Cranial and then distal	Axillary nerve
Coracobrachialis	Coracobrachialis	Shoulder adduction and extension	Coracoid process of the scapula	Crest of the lesser tubercle of humerus, proximal to teres major tuberosity	Distolateral	Musculocutaneous nerve
Biceps brachii—one head	Biceps brachii	Shoulder extension and elbow flexion; some passive stability in weight bearing, preventing shoulder flexion	Supraglenoid tubercle of the scapula	Ulnar tuberosity on proximal cranial ulnar Radial tuberosity on proximal cranial radius (weaker part)	Distomedial	Musculocutaneous nerve
Brachialis	Brachialis	Elbow flexion	Proximal third of the lateral humerus (brachialis groove)	Ulnar tuberosity on proximal cranial ulnar	Distolateral	Musculocutaneous nerve
Triceps brachii—four heads	Triceps brachii—three heads	Elbow extension; helps tense the antebrachial fascia				Radial nerve
Long head	Long head	Elbow extension, shoulder flexion	Caudal border of the scapula	Olecranon tuber or tuberosity	Caudodistal	
Lateral head	Lateral head	Elbow extension	Tricipital line of the humerus—covers the accessory head	Olecranon tuber, lateral to long head	Caudodistal	
Accessory head	None	Elbow extension	Neck of the humerus	Olecranon tuber, between lateral and medial heads	Caudodistal	
Medial head	Medial head	Elbow extension	Crest of lesser tubercle near teres major tuberosity	Olecranon	Caudodistal	
Anconeus	Anconeus	Elbow extension; helps tense the antebrachial fascia	Lateral supracondylar crest; lateral and part of medial epicondyles of the humerus	Lateral surface, proximal end of ulna	Caudal	Radial nerve

Continued

Muscle	Correlated Human Muscle	Joint Action in the Forelimb	Main Cranial, Dorsal, or Proximal Attachment	Main Caudal, Ventral, or Distal Attachment	Fiber Direction from Proximal to Distal Attachment (listed in order of importance)	Innervation
Tensor fasciae antebrachii	None	Elbow extension; chief tensor of the antebrachial fascia	Fascia covering the latissimus dorsi; very thin	Olecranon and the medial fascia of the forearm	Distolateral	Radial nerve
CRANIAL AND LATERAL FOREARM OR ANTEBRACHIUM						
Brachioradialis	Brachioradialis	Supination; rotation of the radius dorsolaterally	Lateral supracondylar crest of the humerus; thin and inconstant muscle	Distal fourth of the radius	Medial then distal	Radial nerve
Extensor carpi radialis	Extensor carpi radialis longus and brevis	Carpal extension; elbow flexion	Lateral supracondylar crest	Small tuberosities on dorsal bases of MCs II and III		Radial nerve
Common digital extensor	Extensor digitorum communis	Extension of digits II-V; carpal extension	Lateral epicondyle of the humerus	Extensor processes of the distal phalanges of digits II-V	Distomedial	Radial nerve
Lateral digital extensor	None	Extension of digits III-V; carpal extension	Lateral epicondyle of the humerus	Proximal ends, all phalanges III-V; extensor processes of distal phalanges of III-V	Distomedial	Radial nerve
Ulnaris lateralis	Extensor carpi ulnaris	Carpal ulnar deviation or abduction; weak carpal flexion	Lateral epicondyle of the humerus	Lateral aspect, MC V; accessory carpal bone	Distolateral	Radial nerve
Supinator	Supinator	Supination; elbow flexion	Lateral epicondyle of the humerus	Cranial surface, proximal fourth of the radius	Distomedial	Radial nerve
CAUDAL AND MEDIAL FOREARM OR ANTEBRACHIUM						
Pronator teres	Pronator teres	Pronation, elbow flexion	Medial epicondyle of the humerus	Medial border of the radius between middle and proximal thirds	Distolateral	Median nerve

Flexor carpi ulnaris: two parts	Flexor carpi ulnaris	Carpal flexion, carpal abduction	Proximally the ulnar head is more cranial and distally the humeral head is more cranial			Ulnar nerve
Ulnar head			Caudal border and medial surface of the olecranon	Accessory carpal bone		
Humeral head			Medial epicondyle of the humerus	Accessory carpal bone		
Flexor carpi radialis	Flexor carpi radialis	Carpal flexion	Medial epicondyle of the humerus	Palmar aspect of proximal metacarpals I and II		Median nerve
Superficial digital flexor	Flexor digitorum superficialis	Flexion of PIP and MCP of II-V; carpal flexion	Medial epicondyle of the humerus and medial border of radius	Palmar surface, base of middle phalanges of II-V		Median nerve
Deep digital flexor: three parts	Flexor digitorum profundus	Flexion of digits; carpal flexion				
Humeral head			Medial epicondyle of the humerus—radial, deep, and medial bellies	For all three heads: palmar surface of the base (proximal end) of the distal phalanx of digits II-V and a weak tendon to digit I		Median nerve to radial head, deep and medial parts of humeral head
Ulnar head	None		Proximal three fourths of caudal border of the ulna			Ulnar nerve to ulnar head and lateral part of humeral head
Radial head	None		Middle third, medial border of the radius			
Pronator quadratus	Pronator quadratus	Pronation	Medial surface of the ulna, caudal to the interosseous membrane	Caudal surface of the radius except proximal and distal ends	Distomedial	Median nerve

Continued

Muscle	Correlated Human Muscle	Joint Action in the Forelimb	Main Cranial, Dorsal, or Proximal Attachment	Main Caudal, Ventral, or Distal Attachment	Fiber Direction from Proximal to Distal Attachment (listed in order of importance)	Innervation
HINDLIMB						
Caudal Thigh Muscles						
Biceps femoris: two or three heads—longest and widest thigh muscle in the hindlimb	Biceps femoris	Cranial—hip and stifle extension Caudal—stifle flexion, tarsocrural extension, hip (hindlimb) abduction	Sacrotuberous ligament and ischiatic tuberosity	Patella; patellar ligament and cranial border of tibia via fascia lata and crural fascia; tibial body via crural fascia; calcaneal tuberosity	Distocranio-lateral	Sciatic nerve
Caudal crural abductor muscle	None	Stifle flexion and hip (hindlimb) abduction with caudal biceps femoris muscle	Sacrotuberous ligament near ischiatic tuberosity	Via crural fascia to the digital extensor	Distocranio-lateral	Sciatic nerve
Semitendinosus	Semitendinosus	Hip and tarsocrural extension; stifle flexion	Ischiatic tuberosity	Medial tibial body and calcaneal tuberosity via crural fascia	Distocranio-medial	Sciatic nerve
Semimembranosus: two bellies	Semimembranosus	Hip extension Femoral belly—stifle extension Tibial belly—stifle flexion or extension, depending on stifle position	Ischiatic tuberosity	Medial lip, distal shaft, caudal rough surface of the femur—distal to pectineus muscle; medial aspect, proximal part, medial tibial condyle	Distocranio-medial	Sciatic nerve
MEDIAL THIGH MUSCLES						
Sartorius	Sartorius					
Cranial part		Hip flexion, stifle extension	Iliac crest, thoracolumbar fascia	Patella, with rectus femoris	Distocaudal	Femoral nerve
Caudal part		Hip flexion, stifle flexion	Cranial VIS, adjacent ventral ilium	Cranial border, medial tibia, with gracilis		
Pectineus	Pectineus	Hip or hindlimb adduction	Body of pubis, iliopubic eminence to pubic tubercle	Medial lip, distal shaft, caudal rough surface of the femur, proximal to semimembranosus		Obturator nerve

Adductor: two parts, which may or may not be distinguishable	Adductors magnus, brevis, and longus	Hip or hindlimb adduction, hip extension	Entire symphysis pelvis via symphysial tendon, adjacent ischiatic arch, ventral pubis and ischium	Entire lateral lip of the caudal rough surface of the femur	Distocranio-lateral	Obturator nerve
LATERAL THIGH MUSCLES						
Tensor fasciae latae	Tensor fascia lata/ tensor fasciae latae	Tense the fascia lata, hip flexion, stifle extension	Cranial VIS and adjacent ilium (=tuber coxae), aponeurosis of middle gluteal muscle	Via fascia lata, connects to biceps femoris and quadriceps femoris muscle to patella	Distocaudal	Cranial gluteal nerve
Superficial gluteal	Gluteus maximus	Hip extension, hip or hindlimb abduction	Lateral border of sacrum, caudal 1 or 1st vertebra of the tail, sacrotuberous ligament, cranial DIS via deep gluteal fascia	Third trochanter, fusing with aponeurosis of tensor fasciae latae—synovial bursa deep to attachment in one third of dogs	Caudolatero-distal	Caudal gluteal nerve
Middle gluteal	Gluteus medius	Hip extension, abduction, internal rotation	Crest and gluteal or lateral surface of the ilium	Greater or major trochanter	Caudolatero-distal	Cranial gluteal nerve
Piriformis—may actually be part of the middle gluteal or deep gluteal muscle	Piriformis	Hip extension	Lateral surface of S3 and C1	Greater trochanter with middle gluteal muscle	Caudolatero-distal	Caudal gluteal nerve
Deep gluteal	Gluteus minimus	Hip extension, abduction, internal rotation	Body of ilium, ischiatic spine	Greater trochanter, cranial aspect	Caudodistal	Cranial gluteal nerve
CRANIAL THIGH MUSCLES						
Iliopsoas	Iliopsoas	Hip flexion				Ventral rami of lumbar and femoral nerves

Continued

Muscle	Correlated Human Muscle	Joint Action in the Forelimb	Main Cranial, Dorsal, or Proximal Attachment	Main Caudal, Ventral, or Distal Attachment	Fiber Direction from Proximal to Distal Attachment (listed in order of importance)	Innervation
Psoas major	Psoas major	Hip flexion, vertebral column flexion, lumbar spine extension with ventral tilting of the pelvis	Transverse processes and bodies of the lumbar vertebrae	Lesser trochanter with the iliacus	Caudoventro-lateral	
Iliacus	Iliacus	Hip flexion	Smooth ventral surface of the ilium	Lesser trochanter with the psoas major	Caudoventro-lateral	
Quadriceps femoris	Quadriceps femoris	See actions below				
Rectus femoris	Rectus femoris	Stifle extension, hip flexion	Lateral ilium, cranial to the acetabulum —bursa occasionally deep to this	Tibial tuberosity	Distocranio-lateral	Femoral nerve
Vastus lateralis	Vastus lateralis	Stifle extension	Proximal part of the lateral lip of the caudal rough surface of the femur	Tibial tuberosity—bursa usually deep to tendon attachment	Distocranio-medial	Femoral nerve
Vastus intermedius	Vastus intermedius	Stifle extension	Lateral side of the proximal femur, with the vastus lateralis	Tibial tuberosity	Distocranio-medial	Femoral nerve
CRANIAL THIGH MUSCLES						
Vastus medialis	Vastus medialis	Stifle extension	Medial side, proximal end of the cranial end, medial lip, caudal rough surface of the femur	Tibial tuberosity—bursa usually deep femur; proximal attachment	Distocranio-lateral to tendon	Femoral nerve
Articularis genu	Articularis genu	Stifle extension; tenses the proximal pouch of the stifle joint capsule	Cranial surface of femur, proximal to the trochlea	Tibial tuberosity		Femoral nerve

CRANIOLATERAL MUSCLES OF THE LEG OR CRUS

Cranial tibial	Anterior tibialis/ tibialis anterior	Talocrural flexion; rotates the hindpaw so the plantar surface of the paw faces medially	Extensor groove and adjacent articular margin of tibia, lateral edge of the cranial border of the tibia	Plantar surface of base of MT I and II after coursing deep to the crural extensor retinaculum	Distomedial	Deep peroneal nerve
Long digital extensor	Extensor digitorum longus	DIP and PIP extension II-V, talocrural flexion	Extensor fossa on the femur lateral to the trochlea	Extensor processes on the dorsum, distal phalanges of digits II-V coursing deep to the crural and tarsal extensor retinacula	Distomedial	Peroneal nerve
Extensor digiti I longus	Extensor hallucis longus and part of the extensor digitorum longus	Digit II extension, digit I extension	Cranial border of the fibula between proximal and middle third of the interosseous membrane cranial to the peroneus brevis	Fascia and tendon of the lateral digital extensor of MTP joint II; aponeurosis of the metatarsus, rudiment of digit I or hallux	Distomedial	Deep peroneal nerve
Lateral digital extensor	Peroneus tertius	Digit V extension and abduction	Proximal third of the fibula	Extensor process on the dorsum of middle phalanx of digits V coursing deep to the crural and tarsal extensor retinacula		Superficial peroneal nerve
Peroneus longus	Peroneus longus	Talocrural flexion, rotates the hindpaw so the plantar surface of the paw faces laterally	Lateral tibial condyle, proximal fibula, lateral collateral ligament of the stifle, which then attaches to the lateral femoral epicondyle	Fourth tarsal bone/ quartal bone, plantar aspect of the base of the MTs after coursing deep to the abductor digiti V muscle	Distal on the leg then medial on the plantar hindpaw	Common peroneal nerve, deep peroneal nerve
Peroneus brevis	Peroneus brevis	Talocrural flexion	Lateral surface of the distal two thirds of the fibula and tibia	Proximal end of MT V in a common sheath with the extensor digitorum lateralis after coursing deep to the lateral collateral ligament of the ankle	Distal and slightly medial	Superficial peroneal nerve

Continued

Muscle	Correlated Human Muscle	Joint Action in the Forelimb	Main Cranial, Dorsal, or Proximal Attachment	Main Caudal, Ventral, or Distal Attachment	Fiber Direction from Proximal to Distal Attachment (listed in order of importance)	Innervation
CAUDAL MUSCLES OF THE LEG						
Popliteus	Popliteus	Stifle internal rotation/ leg internal rotation	Lateral condyle of the femur	Proximal third caudal surface of tibia, with a sesamoid at the muscle–tendon junction	Distomedial	Tibial nerve
Gastrocnemius: two heads	Gastrocnemius—two heads	Talocrural extension; stifle flexion	Medial and lateral supracondylar tuberosities of the femur; each tendon has a sesamoid bone or fabella that articulates with a femoral condyle	Dorsal surface of the calcaneal tuberosity by the common calcaneal tendon to provide the largest portion of the tendon		Tibial nerve
Superficial digital flexor	Flexor digitorum brevis	MTP and PIP joints of II-V; stifle flexion, talocrural extension	Lateral supracondylar tuberosity of the femur	Calcaneal tuberosity and bases of middle phalanges digits II-V		Tibial nerve
Deep digital flexor	Flexor digitorum longus	Digit flexion; talocrural extension by both parts	See each part separately below	Two heads join at the distal tarsals and then attach to the plantar surface of phalanges I-V	Distomedial the base of distal	Tibial nerve
Lateral digital flexor (flexor hallucis longus)	Flexor hallucis longus		Caudolateral border of the proximal two thirds of tibia, proximal half of fibula, and adjacent interosseous membrane	Before joining the medial digital flexor muscle, the tendon is bound in a groove at the sustentaculum tali		
Medial digital flexor (flexor digitorum longus)	Flexor digitorum longus		Caudomedial border of the tibia, popliteal line head of the fibula	Tendon lies on the caudomedial side of the tibia before joining the lateral digital flexor muscle		

Caudal tibial	Posterior tibialis/ tibialis posterior	Rotation of the hindpaw so the plantar paw faces medially; talocrural extension	Medial proximal fibula	Medial ligamentous masses of the tarsus after coursing as a very thin tendon, lying cranial to medial digital flexor at the talocrural joint	Distomedial	Tibial nerve
DEEP DORSAL MUSCLES OF THE NECK						
Splenius	Splenius	Extension, ipsilateral side bend, and ipsilateral rotation of the neck, raising the head and neck, stabilization of T1	Spinous processes of T1-T2, ligamentum nuchae cranial to T1, median dorsal raphe of cervical spine, by aponeurosis of cranial part of thoracolumbar fascia, extending to T5-T6	Mastoid part of the temporal bone with the longissimus capitis, dorsal nuchal line of occiput, at times to the wing of the atlas	Cranioventral	Dorsal rami of cervical nerves
DEEP VENTRAL MUSCLES OF THE NECK						
Scalenus: two parts	Scalenes	Ipsilateral side bend of the neck, pulls the neck down, inspiration				Ventral rami of cervical and thoracic spinal nerves
Supracostal scalenus—superficial part (two or three parts)			Lateral surfaces of ribs 1-9	Transverse processes of C4 and C5 and sometimes C3	Craniomedio-dorsal	
Primae costae scalenus—deep part (three parts)			As three portions from the cranial border of rib 1	Superficial part to transverse process of C3 through C5; two deep parts to transverse processes of C6 and C7	Craniomedio-dorsal	
Longus capitis	Longus capitis	AO flexion, pulls the neck down	Caudal surfaces of the transverse processes of cervical vertebrae	Ventral surface of the basioccipital bone of the skull	Cranial	Ventral rami of cervical spinal nerves

Continued

Muscle	Correlated Human Muscle	Joint Action in the Forelimb	Main Cranial, Dorsal, or Proximal Attachment	Main Caudal, Ventral, or Distal Attachment	Fiber Direction from Proximal to Distal Attachment (listed in order of importance)	Innervation
Longus colli	Longus colli	Neck flexion, pulls the neck down	Ventral convex surfaces of T1-T6 vertebrae Ventral borders of transverse processes of C6-C3 Ventral surface of the angle of cranial ribs	Ventral border of the wing of C6 and the transverse process of C7 Ventral aspect of the next cranial vertebra Ventral aspect of the atlas	Cranial Craniomedial Craniomedial	Ventral rami of cervical spinal nerves
LATERAL AND VENTRAL THORACIC WALL MUSCLES						
External intercostals	External intercostals	Draw ribs together, breathing	Caudal border of a rib	Cranial border of the next caudal rib	Caudoventral	Intercostal nerves
Internal intercostals	Internal intercostals	Draw ribs together, breathing	Extending from the vertebral column to the distal ends of the rib—cranial border of a rib	Caudal border of the next cranial rib	Cranioventral	Intercostal nerves
MUSCLES OF THE ABDOMINAL WALL AND DIAPHRAGM						
Rectus abdominis	Rectus abdominis	Flexion of the spine, bring pelvis forward (i.e., posterior pelvic tilt); abdominal press functions—expiration, urination, defecation, parturition; support of abdominal viscera	Sternum, rib 1, and 1st costal cartilage, and a fleshy attachment on the sternal portion of the 9th costal cartilage	Pecten ossis pubis, between the two iliopectineal eminences	Caudal	Intercostal, costoabdominal, ilioinguinal nerves, medial branches
External abdominal oblique abdominis: two parts	External oblique abdominis	Flexion of the spine, ipsilateral side bend, functions of abdominal press, contralateral rotation of the spine, support of abdominal viscera	Last ribs, thorocolumbar fascia	Linea alba, cranial pubic ligament	Caudoventral	Intercostal nerves 3 or 4-12, costoabdominal, iliohypogastric and ilioinguinal nerves, lateral branches

Internal abdominal oblique: abdominis three parts	Internal oblique abdominis	Flexion of the spine, ipsilateral side bend, functions of abdominal press, support of abdominal viscera	Thoracolumbar fascia caudal to rib 12 with the pars lumbalis	Rectus abdominis and linea alba	Cranioventral	Caudal intercostal nerve and medial branches of the costoabdominal, iliohypogastric, and ilioinguinal nerves
Transversus abdominis	Transversus abdominis	Abdominal press, support of abdominal viscera	Costal cartilage of rib 8, transverse process of L7, and tuber coxae	Linea alba	Ventral	Caudal intercostal nerve and medial branches of the costoabdominal nerve
Diaphragm	Diaphragm	Inspiration, support of abdominal viscera	Ventral surfaces of lumbar vertebrae, ribs, and sternum	Central tendon	Craniomedial	Phrenic nerve, ventral rami of C5-C7
DEEP DORSAL MUSCLES OF THE NECK AND TRUNK						
Iliocostalis system: two parts	Iliocostalis	Ipsilateral side bend; stabilizes the spine, expiration by pulling the ribs caudally				Dorsal rami of thoracic and lumbar nerves
Iliocostalis thoracis	Iliocostalis thoracis		Cranial borders of vertebral ends of ribs 2-12	Costal angles of the ribs, transverse process of C7	Cranial	
Iliocostalis lumborum	Iliocostalis lumborum		Pelvic surface of the wing of the ilium, the iliac crest, and the intermuscular septum between the iliocostalis and longissimus muscles	Lumbar vertebrae and last 5 ribs	Cranioventral	
LONGISSIMUS SYSTEM: THREE PARTS—EXTENT IS FROM THE ILIAC CREST TO C7						
Longissimus thoracis et lumborum: two parts—strongest muscle of the trunk in the thoracic and lumbar region		Extension of the spine, raising the cranial portion of the body from the pelvis; stabilizes the spine, raises the caudal body with the hindlimbs			Craniolateral	Dorsal rami of thoracic and lumbar nerves

Continued

Muscle	Correlated Human Muscle	Joint Action in the Forelimb	Main Cranial, Dorsal, or Proximal Attachment	Main Caudal, Ventral, or Distal Attachment	Fiber Direction from Proximal to Distal Attachment (listed in order of importance)	Innervation
Longissimus thoracis	Longissimus thoracis		Spinous processes and supraspinous ligament of thoracic vertebrae	Medial tendons to the accessory processes of T6-T13 and caudal ends of transverse processes of T1-T5 Lateral tendons to the groove adjacent to the costal tubercle of ribs 6-13 Cranially, both attach to the costal tubercles of ribs 1-5		
Longissimus lumborum	Longissimus lumborum		Iliac crest and ventral surface of the ilium, supraspinous ligament of lumbar vertebrae	Dorsal aspects and cranial articular processes of lumbar vertebrae, disk of lumbosacral joint		
Longissimus cervicis	Longissimus cervicis	Extension of the neck, ipsilateral side bend and rotation	Spinous processes and supraspinous ligament of cranial thoracic and caudal cervical vertebrae	Transverse processes of cranial cervical vertebrae		Dorsal rami of cervical and thoracic nerves
Longissimus capitis	Longissimus capitis	AO extension, ipsilateral side bend and rotation	Transverse processes of T1-T3, caudal articular processes of C3-C7	Mastoid process of the temporal bone, united with splenius at the level of the atlas		Dorsal rami of cervical nerves
TRANSVERSOSPINALIS SYSTEM—JOINS ONE OR MORE VERTEBRAE						
	Transversus spinalis					Dorsal rami

Spinalis et semispinalis thoracis et cervicis: two parts	Spinalis and semispinalis thoracis	Neck extension, raises the neck, stabilizes the spine	Dorsal aspect of the longissimus thoracis muscle, spinous processes and accessory processes of cranial lumbar vertebrae and C7-T6 and mammillary processes of T13-L2, iliac crest, spinous processes of T13-S2	Thoracis portion—spinous processes of thoracic vertebrae Cervicis portion—spinous processes of C2-C5 vertebrae	Craniomedial	Dorsal rami of thoracic nerves Dorsal rami of cervical nerves
Spinalis et semispinalis thoracis—lateral						
Spinalis cervicis—medial	Spinalis cervicis					
Semispinalis capitis: two parts	Semispinalis capitis	Neck extension, ipsilateral side bend, stabilize the neck				Dorsal rami of cervical nerves
Multifidus: three parts	Multifidus	Stabilize the vertebral column			Craniomedial	Dorsal rami of cervical, thoracic, and lumbar nerves
Multifidus lumborum	Multifidus lumborum		Dorsal sacrum, mammillary processes of L7-T12	Spinous processes of T9-L6		Dorsal rami of lumbar and thoracic nerves
Multifidus thoracis	Multifidus thoracis		Mammillary and transverse processes of T3-T11	Spinous processes of T7-T8		Dorsal rami of thoracic nerves
Multifidus cervicis	Multifidus cervicis		Articular process of T2	Spinous process of axis		Dorsal rami of cervical nerves

Continued

Muscle	Correlated Human Muscle	Joint Action in the Forelimb	Main Cranial, Dorsal, or Proximal Attachment	Main Caudal, Ventral, or Distal Attachment	Fiber Direction from Proximal to Distal Attachment (listed in order of importance)	Innervation
MUSCLES OF THE TAIL						
Sacrocaudalis dorsalis lateralis	None	Tail extension or lifting, ipsilateral side bend	Aponeurosis of the longissimus, mammillary processes L1-L6 and Cd1-Cd8 articular processes of the sacrum	Mammillary processes of Cd vertebrae Continuation of the longissimus muscle	Caudomedial	Dorsal rami of caudal nerves
Sacrocaudalis dorsalis medialis	None	Tail extension or lifting, ipsilateral side bend	Spine and dorsolateral aspects of cranial vertebrae	Dorsolateral aspects of caudal vertebrae	Caudolateral	Dorsal rami of caudal nerves
Sacrocaudalis ventralis lateralis	None	Tail flexion, ipsilateral side bend	Ventral surface of L7 and the sacrum, ventral surfaces of transverse processes of Cd vertebrae	Ventrolateral tubercle of more caudal vertebrae	Caudomedial	Ventral rami of caudal nerves
Sacrocaudalis ventralis medialis	None	Tail flexion, ipsilateral side bend	Ventral surface of S3 and Cd vertebrae	Hemal process of the next caudal vertebrae	Caudomedial	Ventral rami of caudal nerves

AA, Atlantoaxial; *AO,* atlantooccipital; *caudal DIS,* caudal dorsal iliac spine; *C,* cervical spinal nerve; *caudal VIS,* caudal ventral iliac spine; *Cd,* caudal; *CN,* cranial nerve; *cranial DIS,* cranial dorsal iliac spine; *cranial VIS,* cranial ventral iliac spine; *Cx,* coccygeal; *L,* lumbar spinal nerve; *MC,* metacarpal bone; *MCP,* metacarpophalangeal joint; *MT,* metatarsal bone; *MTP,* metatarsophalangeal joint; *PIP/DIP,* proximal/distal interphalangeal joint; *S,* sacral spinal joint.



WEB APPENDIX 2

Main Ligament Attachments and Function

Name of Ligament	Correlated Human Ligament	Proximal Attachment	Distal Attachment	Fiber Direction	Motion Restricted by Ligament/Function
FORELIMB SHOULDER JOINT					
Transverse humeral retinaculum	Transverse humeral	Medial side of greater tubercle	Lateral side of lesser tubercle	Transverse/ horizontal	Stabilizes the biceps brachii, proximal tendon
Medial glenohumeral	Glenohumeral	Medial lip of glenoid fossa	Greater tubercle	Distal	Stabilizes humeral head, preventing medial dislocation
Lateral glenohumeral	Glenohumeral glenoid fossa	Lateral lip of glenoid fossa	Lesser tubercle	Distolateral	Stabilizes humeral head, preventing lateral dislocation
ELBOW JOINT					
Medial collateral	Lateral or radial collateral	Humeral medial epicondyle	Radial tuberosity and radius and ulna in the interosseous space by crura	Distal	Limits elbow valgus
Lateral collateral	Medial or ulnar collateral	Humeral lateral epicondyle	Ulna with the annular ligament and area distal to the radial neck	Distal	Limits elbow varus
RADIOULNAR JOINTS					
Annular	Annular	Lateral extremity of the radial notch of the ulna	Medial extremity of the radial notch of the ulna	Circular	Stabilizes radial head position
Interosseous	Interosseous	Radius	Ulna	Between ulna and radius	Stabilizes radius and ulna in radioulnar pronation
Radioulnar	Distal radioulnar	Distal, lateral radius	Distal, medial ulna	Transverse	Stabilize the distal radioulnar joint
CARPAL JOINTS					
Flexor retinaculum/ transverse palmar carpal	Transverse carpal	Accessory carpal and styloid process of radius	Palmar projections of radius and proximal carpals	Transverse	Two layers stabilize tendons of the superficial and deep digital flexors
Short radial collateral	Radial collateral of the wrist				
Straight part		Tubercle proximal to radial styloid process	Medial radial carpal	Distal	Valgus deviation
Oblique part		Radial styloid process	Palmomedial surface of radial carpal	Distal	Valgus deviation
Short ulnar collateral	Ulnar or medial collateral	Ulnar styloid process	Ulnar carpal	Distal	Varus deviation

Dorsal radiocarpal	Radial or lateral collateral	Dorsolateral radius	Dorsoproximal ulnar carpal	Distolateral	Carpal flexion
Palmar carpal	Palmar carpal ligaments	Palmar aspect of radius, ulna, and carpals	Distally on the carpals and metacarpals	Distal, distolateral, distomedial	Carpal extension
DIGITAL JOINTS					
Collaterals of the MCP joints	Collaterals of the MCP joints	Distal ends of metacarpals	Proximal ends of proximal phalanges	Vertical in weight-bearing	Medial and lateral MCP joint motion
Collaterals of the PIP joints	Collaterals of the PIP joints	Distal ends of proximal phalanges	Collateral tubercles on the proximal ends of the middle phalanges and distal phalanges in digit I	Vertical in weight-bearing	Medial and lateral PIP motion
Collaterals of the DIP joints	Collaterals of the DIP joints	Distal ends of middle phalanges	Sides of the ungual crests of the distal phalanx	Caudodistal	Medial and lateral PIP motion
HINDLIMB HIP JOINT					
Transverse acetabular	Transverse acetabular	One side of acetabular notch	Other side of acetabular notch	Caudal	Completes the acetabular lip to secure femoral head in the acetabulum
Ligament of the femoral head	Ligamentum teres or ligamentum capitis femoris	Fovea in the femoral head	Acetabular fossa	Mediocaudal	Maintains hip reduction
STIFLE JOINT					
Cranial cruciate	Anterior cruciate	Intercondylar fossa, caudomedial part of the femoral lateral condyle	Cranial intercondylar area of the tibia	Craniodistal	Stifle cranial glide during extension, also internal rotation
Caudal cruciate	Posterior cruciate	Intercondylar fossa, femoral medial condyle	Medial edge, popliteal notch of tibia, caudal to the caudal attachment of the medial meniscus	Caudodistal	Stifle caudal glide during flexion
Medial collateral	Medial or tibial collateral	Medial femoral epicondyle	Medial tibial border, distal to the condyle Attaches to the medial meniscus	Distocranial	Stifle valgus and extension
Lateral collateral	Lateral or fibular collateral	Lateral femoral epicondyle	Fibular head and adjacent tibial condyle	Distal (vertical instance)	Stifle varus and extension
Patellar ligament	Patellar ligament (patellar tendon)	Quadriceps tendon and patella	Tibial tuberosity	Distal	Transfer force of quadriceps muscle to the tibia

Continued

Name of Ligament	Correlated Human Ligament	Proximal Attachment	Distal Attachment	Fiber Direction	Motion Restricted by Ligament/Function
TALOCURAL JOINT					
Medial collateral	Medial or tibial collateral				Stabilization of lateral motion of the tarsus
Long part		Medial aspect of the medial malleolus	Firmly to tarsal I and the central tarsal and loosely to MTs I-II and the talus	Distocranial	
Short part: two parts		Medial aspect of the medial malleolus	1. Tarsal I and MTs, sustentaculum tali (deep to long part) 2. Talus	Distocranial Caudal	
Lateral collateral	Lateral or fibular collateral				Stabilization of internal motion of the tarsus
Long part		Lateral malleolus	Calcaneus, tarsal IV, and base of MT V	Distocranial	
Short part: two parts		Lateral malleolus	1. Tuber calcanei 2. Talus	Distocaudal Caudodistal	
TARSUS					
Distal extensor retinaculum—distal transverse ligament of tarsus	None	Calcaneus	Surrounds the long digital extensor to attach to the calcaneus	Circular	Secure the long digital extensor muscle
Dorsal ligaments—many, named by connecting bones	Many dorsal ligaments	From one tarsal bone	Another tarsal bone or MT bone	Different for each ligament	Stabilize the dorsal tarsus Limit tarsal extension
Talocalcaneal	Subtalar interosseous	Talus—distal surface	Calcaneus—proximal surface	Near vertical	Stabilize the talocalcaneal articulation
Plantar ligaments—thicker and stronger than dorsal talocrural ligaments	Long and short plantar ligaments				Support the joints of the plantar hindpaw Increase stability Limit dorsiflexion
Medial plantar		Sustentaculum tali, plantar surface	Central tarsal and tarsometatarsal joint capsule	Distolateral	

Middle plantar—most commonly injured plantar ligament		Body of the calcaneus, plantar aspect	Tarsal bone IV and bases of MTs IV and V	Distal	
Lateral plantar		Caudolateral calcaneus	Long part of lateral collateral ligament of the talocrural joint and base of MT V	Distal	
DIGITAL JOINTS					
Collaterals of the MTP joints	Collaterals of the MTP joints	Distal ends of MTs	Proximal ends of proximal phalanges	Vertical in weight-bearing	Abduction and adduction MTP motion
Collaterals of the PIP joints	Collaterals of the PIP joints	Distal ends of proximal phalanges	Collateral tubercles on the proximal ends of the middle phalanges and distal phalanx in digit I	Vertical in weight-bearing	Abduction and adduction PIP motion
Collaterals of the DIP joints	Collaterals of the DIP joints	Distal ends of middle phalanges	Sides of the ungual crests of the distal phalanx	Caudodistal	Abduction and adduction PIP motion
Proximal and distal digital annular		One side of phalanx	Other side of phalanx crossing the plantar surface	Mediolateral	Secure the flexor tendons close to the phalanges
SPINE					
AO Joint					
Dorsal AO ligament or membrane	Posterior AO	Dorsal edge of foramen magnum	Cranial border of the arch of the atlas	Craniocaudal	AO flexion Punctures are made through this membrane to drain cerebrospinal fluid
Ventral AO ligament or membrane	Anterior AO	Ventral edge of foramen magnum	Body of the atlas	Craniocaudal	AO extension
Lateral AO ligament	Lateral AO	Lateral part of the dorsal arch of the atlas	Jugular process of the occipital bone	Cranioventro-lateral	AO contralateral side bend
AA Joint					
Dorsal AA membrane	Posterior AA	Caudal margins of the sides and body of the atlas	Cranial margin of the same areas of the axis	Craniocaudal	AA contralateral side bend
Apical ligament of the dens	Apical	Dens of the axis	Foramen magnum, occipital bone medial to the caudal parts of the occipital condyles	Cranial and craniolateral	Stabilizes the dens
Transverse ligament of the atlas	Transverse portion of the cruciform ligament	Internal surface of the ventral arch of the atlas	Internal surface of the other side of the arch of the atlas	Mediolateral	Stabilizes the dens

Continued

Name of Ligament	Correlated Human Ligament	Proximal Attachment	Distal Attachment	Fiber Direction	Motion Restricted by Ligament/Function
INTERVERTEBRAL JOINTS					
Ligamentum nuchae/nuchal ligament	Ligamentum nuchae	Caudal part of the spinous process of C2	Tip of spinous process of T1	Craniocaudal	Stabilizes the cervical spine
Supraspinous	Supraspinous	Spinous process, T1 being the most cranial	Each sequential spinous process to Cd3	Craniocaudal	Limits separation of the spinous processes during spine flexion motion
Ventral longitudinal	Anterior longitudinal	Ventral surfaces of bodies of vertebrae, C2 being the most cranial Best developed caudal to the middle thorax	Ventral surfaces of bodies of vertebrae, the sacrum being the most caudal	Craniocaudal	Limits spine extension
Dorsal longitudinal	Posterior longitudinal	Dorsal surfaces of bodies of vertebrae, C2 being the most cranial Heavier than the ventral longitudinal ligament	Dorsal surfaces of bodies of vertebrae, the end of the vertebral canal in the Cd region being the most caudal	Craniocaudal	Limits spine flexion
Interspinous	Interspinous	Cranial border of a spinous process	Caudal border of a spinous process	Craniocaudal	Limits spine flexion
Intertransverse	Intertransverse	Transverse process of a caudal vertebra	Transverse process of an adjacent cranial vertebra	Craniolateral	Limits side bend
Yellow/ interarcuate/ ligamentum flavum	Ligamentum flavum	Loose thin elastic ligament attached to the arch of a vertebra	Arch of an adjacent vertebra	Craniocaudal	Limits spine flexion

LIGAMENTS OF THE RIBS					
Ligament of the head		Head of the rib	Lateral part of the intervertebral disk and body adjacent to the disk	Mediolateral	Stabilizes the rib
Ligament of the tubercle/dorsal costotransverse ligament		Distal to the articular capsule of the tubercle	Transverse process of the vertebra corresponding to the rib	Mediolateral	Stabilizes the rib
Ligament of the neck		Neck of the rib	Ventral surface of transverse process and adjacent lateral surface of the body of the vertebra	Medioventral	Stabilizes the rib
SACROILIAC JOINT					
Ventral sacroiliac	Ventral sacroiliac	Sacrum, ventral surface	Ilium, ventral surface	From the sacrum, dorsolateral and medial	Stabilizes the sacroiliac joint, prevents ventral displacement of the sacrum
Dorsal sacroiliac	Dorsal sacroiliac	Caudal dorsal iliac spine	Caudal two thirds of the lateral border of the sacrum or even Cd1	Caudomedial portion and dorsocaudal portion	Stabilizes the sacroiliac joint
Sacrotuberous	Sacrotuberous	Caudolateral apex of the sacrum and Cd1	Lateral angle of the ischiatic tuberosity	Caudolatero-ventral	Stabilizes the sacrum and pelvis Muscle attachment site

AA, Atlantoaxial; *AO,* atlantooccipital; *Cd,* caudal; *DIP,* distal interphalangeal; *L,* lumbar; *MCP,* metacarpophalangeal; *MTP,* metatarsophalangeal; *PIP,* proximal interphalangeal; *S,* sacral.



WEB APPENDIX 3

Manufacturers of Supplies and Equipment

Aquatic Therapy

Aqua Paws Underwater Treadmill System Platinum
http://www.fernovetsystems.com/smallanimal/ap_2chamber.htm
1 (888) 206-7802

Canine Aquatherapy (Underwater Treadmills, Pools)
http://www.canineaquatherapy.com/main/Main/main.html
1 (604) 888-2525

Hydro Physio
http://www.hydrophysio.com
1 (866) 385-7676

Water Walker Treadmill (underwater treadmill)
http://www.animalrehab.co.uk/animalrehab/Water_Walker/
1 (877) 426-9255

Hydro Hound
http://www.hydrohound.com/
1 (541) 798-0110

Paws Aboard Life Jackets
http://www.pawsaboard.com/dog-lifejacket-pet-life-jacket.asp
1 (317) 222-1112

Hydro Horse
http://horsetreadmills.com/
1 (800) 405-1099

Physio-Tech Aqua Treadmill
http://www.physio-treadmill.egah.de/
+49 40232384

Aquatic Access Inc. (dog lift for pools)
www.aquaticaccess.com
1 (502) 425-5817

VetSystems Whirlpool
http://www.vetsystems.net
1 (843) 572-0708

Cryotherapy

Canine Icers
http://www.canineicer.com
1 (434) 975-5939

Grampa's Garden
http://www.grampasgarden.com/
1 (207) 373-0770

Canine Chillows
http://www.eoco.org.uk/chillowspets.shtml
+44 1256 332737

Pro-Kold Ice Wraps
http://www.equinetherapyproducts.com/store/product/4
1 (800) 425-9506

Ice Horse
http://www.mackinnonicehorse.com/
1 (800) 786-6633

Game Ready Vasopneumatic Compressive Devices
http://www.gamereadyvet.com/
1 (888) 426-3732

DVDs/Books

Building the Canine Athlete
http://www.caninesports.com
1 (443) 535-9144

Wizards of Paws
http://www.wizardofpaws.net
1 (860) 537-1081

Long Term Care Pets
http://www.activitytherapy.com/pet.htm
1 (973) 729-6601

Sound Therapy
http://www.soundtherapy4pets.com/pet-therapy.php
+44 1244 371 473

Pet Therapy Services
http://www.pettherapyservices.org/products.html
chris@pettherapyservices.org

Electrical Stimulation

Intellect Vet Electrotherapy System
http://www.fernovetsystems.com/equine/es_intelect.htm
1 (888) 206-7802

Matrix Therapy Products
http://therapyproducts.net/
1 (503) 632-7187

Alpha Stim SCS
www.midwestmicrocurrent.com/vetindex.html
1 (888) 583-0944

Kits/Equipment

Canine Rehab Products
http://www.canine rehabproducts.com
1 (413) 243-0253

Balanced Canine Products
http://www.balancedcanineproducts.com
1 (954) 655-2584

Animal Therapy Systems
http://www.animaltherapysystems.com/products/
1 (800) 544-8957

Jog A Dog Treadmill
http://www.jogadog.com/
1 (800) 756-4364

FitPAWS Canine Conditioning Equipment
http://www.balldynamics.com/fitpaws.php
1 (800) 752-2255

DogTread Treadmill
http://www.petzenproducts.com/health_d353.htm
1 (801) 866-3955

Lasers

K Laser
http://www.k-laserusa.com/your-k-laser/animal-veterinary/
1 (877) 776-8246

Low Level Laser Therapy
http://www.respondsystems.com
1 (800) 722-1228

Low Level Laser Therapy System
http://www.danscottandassociates.com
1 (888) 866-6736

Pulsing Magnetic Therapy
http://www.respondsystems.com
1 (800) 722-1228

Emerson Worldwide (lasers, infrared, magnetics, etc.)
http://www.emersonww.com/
1 (888) 974-5687

Companion Therapy Laser
www.companiontherapy.com
1 (302) 709-0408

SpectraVET Therapeutic Lasers
www.spectravet.com
1 (704) 263-8101

Irradia Education & Resources (laser therapy education)
www.irradia.us
1 (704) 263-5525

Splints, Braces, Orthotics, Prosthetics, and Supports

Animal Ortho Care, LLC
http://www.animalorthocare.com
1 (703) 474-6204

A-Trac Dynamic Brace
http://www.woundwear.com
1 (800) 443-4055

Dynasplint
http://www.dynasplint.com
1 (800) 638-6771

Jorvet Pet Brace
http://www.jorvet.com
1 (800) 525-5614

Orthovet Splints
http://www.orthovet.com
1 (541) 544-2435

Tammy and Teddy's Booties and More
http://tammyandteddys.com
1 (860) 749-6552

Thera-Paw Pet Products
http://www.therapaw.com
1 (800) 525-5614

Canine Orthopedic Leg Braces
http://www.dogbrace.com/
1 (209) 688-4047

Bottoms Up Leash
http://www.bottomsupleash.com/
1 (800) 805-2001

Walkabout Harnesses
http://www.walkaboutharnesses.com/
1 (800) 779-0439

Ace Ortho Solutions
http://www.aceorthosolutions.com
1 (308) 423-2690

Help 'Em Up Harness
http://www.helpemup.com/
1 (303) 447-2201

K-9 Orthotics & Prosthetics, Inc.
www.k-9orthotics.com
1 (902) 865-5596

Ortho Pet Custom Prosthetics and Orthotics
http://www.orthopets.com/
1 (303) 953-2545

Sky Prosthetics, Inc.
http://www.skyprosthetics.com
1 (866) 832-1269

Ortho Dog Mobility Braces
http://www.orthodog.com/
1 (828) 298-0885

Biko Canine Physio Brace
http://www.biko.co.at/
http://www.biko.co.at/english/default.html
+43 699 15200080

Thermotherapy

Infrared Therapeutic Pet Pads and Beds
http://www.mtavet.com
1 (866) 311-1303

Therapeutic Pet Beds
http://www.thermotexusa.com/products-pets.htm
1 (513) 942-2770

Wheelchairs

Doggon' Wheels
http://www.doggon.com
1 (800) 736-4466

Eddie's Wheels for Pets
http://www.eddieswheels.com
1 (888) 211-2700

Tammy and Teddy's Booties and more
http://tammyandteddys.com
1 (860) 749-6552

Treadmills, wheelchairs, etc.
http://ablepet.com/Therapeutic-Exercise/CanineRehab Products.html
1 (413) 243-0253

K9 carts
http://www.k9-carts.com/
1 (800) 578-6960

Animal Therapy Products
http://www.animaltherapyproducts.com/
1 (904) 779-2590

Dewey's Wheelchairs for Dogs
http://www.wheelchairsfordogs.com/
1 (877) 312-2122

Walkin' Wheels Wheelchairs for Dogs
http://www.walkinwheels.com/
1 (603) 577-8854

Canine Wheelchair Project
web.mac.com/gbertocci
canine.biomechanics@gmail.com

Wraps (Incontinence) & Modified Items

Sam's Dog Hut
http://www.samsdoghut.com
1 (888) 908-4888

DogLeggs
http://www.dogleggs.com
1 (800) 313-1218

Advanced Canine
http://www.dogpt.com/catalog.htm
1 (908) 447-3876

Hot or Cold Leg Wraps
http://www.animaltherapysystems.com/rejuvgelz/
1 (800) 544-8957

The Femwrap (for urinary incontinence)
http://www.arwi.com/products.html
1 (919) 861-5868

Rubber Dog Boots
http://www.pawzdogboots.com
1 (646) 301-8959

Pet Therapy Pads & Pillows
http://www.pettherapypadsandpillows.com/productpage .htm
1-866-595-7749
gonerennie@aol.com

Puppy Stairs
http://www.puppystairs.com
1 (866) 767-4551

Medipaw Protective Boot
http://www.medivetproducts.com/
1 (800) 245-3413

The Anxiety Wrap
http://www.anxietywrap.com/
1 (260) 344-1217

Miscellaneous

Magnetic Therapy
http://www.therionmagnetics.com/magnetic-pet-products .html
1 (888) 636-9700

Horse Magnetic Equipment
http://www.equinemagnetic.com/Pet.html
1 (800) 731-8463

Magnetic Pet Bed
http://magnapak-magnetic-mattress-health-therapy.com/catalog/index.php
1 (519) 471-8611

Ramps, Beds, Vests
http://ablepet.com/Therapeutic-Exercise/CanineRehabProducts.html
+61 (3) 9793 6188

Shampoo for Dog Skin Conditions
http://www.ravenpetproducts.com/
1 (800) 888-3647

Equineox Technologies (hyperbaric chamber)
http://www.equineox.com/index2.html
1 (604) 465-3134

Equine Therapy Products
http://www.equinetherapyproducts.com/
1 (800) 425-9506

Equine Health Care
http://www.equinehealthcare.com/
1 (859) 223-1326

Handicapped Pets
http://handicappedpets.com/www/index.php/pet-products-summary.html
1 (888) 811-7387

Senior Pet Products
http://www.seniorpetproducts.com/
1 (800) 523-7979

Scout's Store ("special needs" pet products)
http://www.scoutshouse.com/store/
1 (800) 558-8951

FitPAWS Canine Conditioning Equipment
http://www.balldynamics.com/fitpaws.php
1 (800) 752-2255

Lite Gait Mobility Training
http://litegait.com/litegaitv.html
1 (800) 332-9255

GAITRite Gait Analysis Software
http://gaitfour.com/site/
1 (610) 449-4879

Motion Imaging and Analysis
http://www.mi-as.com/
1 (805) 577-0463

Ruff Wear Performance Dog Gear
www.ruffwear.com
1 (888) 783-3932



A Glossary of Terms in Physical Rehabilitation

accessory joint motions: See *arthrokinematic joint motions.*

active assisted range of motion: Occurs when some degree of muscle activity assists joint range of motion activities.

active range of motion: The motion of a joint that may be achieved by active muscle contraction.

activities of daily living: Activities involved in self-care, communication, and mobility. Examples are climbing stairs, the ability to eliminate independently, and other tasks necessary for normal independent function.

aerobic activity or conditioning: The performance of exercise to increase endurance.

aerobic capacity: A measure of the ability to perform work or participate in activity over time using a body's oxygen uptake, delivery, and energy release mechanisms.

affective: Relating to the expression of emotion; for example, affective disorder.

afferent: Proceeding from the peripheral to the central nervous system.

airway clearance techniques: A broad group of activities used to manage or prevent consequences of acute and chronic lung diseases and impairments, including those associated with surgery.

algometer (pressure): An instrument for measuring the degree of sensitivity to a painful stimulus.

ambulation: Walking, with or without the use of assistive devices.

amplitude: The maximum difference between an alternating current's peak and average values.

anaerobic threshold: The point during exercise at which a person cannot supply enough oxygen to meet the demands of the body.

angiogenesis: Angiogenesis is the physiologic process through which new blood vessels form from preexisting vessels.

anterior: In front; ventral.

approximation: Bringing together two joint surfaces.

arrhythmia: An irregular or abnormal heart rhythm.

arthrokinematic joint motion: Describing the motion of a joint without regard to the forces producing that motion or resulting from it; describing the structure and shape of joint surfaces. Examples of these motions are glide (slide), roll, spin, distraction or traction, and compression or approximation.

ASIF: Association for the Study of Internal Fixation.

assistive, adaptive, supportive, and protective devices: A variety of implements or equipment used to aid individuals in performing tasks or movements.

atelectasis: Airlessness of the lungs caused by failure of expansion or resorption of air from the alveoli.

athermal: Not using heat, describing, for example, a modality such as pulsed ultrasound.

atlantoaxial luxation: Occurs when instability of the joint allows the axis to luxate dorsally, relative to the atlas, and compress the spinal cord. Congenital in some toy breeds of dogs.

autogenic drainage: Airway clearance through the patient's own efforts (such as coughing).

axonotmesis: The axon integrity is lost, but the endoneurium and Schwann cell sheath it lies within are still intact, providing the opportunity for regeneration back to the correct target. Successful regeneration may occur, particularly if the axon is damaged close to its target.

balance: The ability of an individual to maintain the body in equilibrium with gravity both statically (e.g., while stationary) and dynamically (e.g., while walking).

ballistic stretching: A form of stretching in which a series of quick movements are used to stretch the muscles and connective tissues.

beam nonuniformity ratio (BNR): Regarding therapeutic ultrasound, compares the maximal intensity from the transducer to the average intensity. This ratio should be low (between 2 and 6) to indicate that the energy distribution is relatively uniform.

biofeedback: A training technique that enables an individual to gain some element of voluntary control over muscular or autonomic nervous system functions using a device that produces auditory or visual stimuli.

biomechanical: Describing the action of forces on the body, especially as they affect the musculoskeletal system.

body mechanics: The interrelationships of the muscles and joints as they maintain or adjust posture in response to environmental forces.

carpal hyperextension: A problem of the carpal joints in which the palmar carpal ligaments and palmar carpal

fibrocartilage are damaged and the dog develops a palmigrade stance.

case management: The coordination of patient care or client activities.

cauda equina syndrome: A progressive neurologic syndrome characterized by lumbar pain, fecal and urinary incontinence, and possible progressive neurologic deficits caused by soft and hard tissue proliferation associated with lumbosacral intervertebral disk disease.

caudal: In animals, a directional term meaning "toward the tail."

caudal cervical stenotic myelopathy: See *cervical spondylomyelopathy.*

cervical spondylomyelopathy: A disorder caused by abnormal development of the cervical vertebrae resulting in compression of the spinal cord. Relatively common in Great Danes and Doberman pinschers. Sometimes also called caudal cervical stenotic myelopathy or wobbler syndrome.

chondroprotectant: Various compounds that are proposed to have a positive effect on the health and metabolism of chondrocytes and synoviocytes.

cicatrix: Scar; the fibrous tissue replacing the normal tissues destroyed by injury or disease.

circulation: The passage of blood through the heart, blood vessels, organs, and tissues; it also describes the oxygen delivery system.

client: An individual, business, agency, or other organizational entity receiving consultative services.

clinical indications: The patient factors (symptoms, impairments, deficits, and so forth) that suggest that a particular kind of care (examination, intervention) would be appropriate.

closed kinetic chain exercise: Exercise with foot fixed against a resistance.

coaptation device: A bandage, splint, or cast used to immobilize joints and tissues.

coherence: Light waves that are "in phase" with one another.

collagenase: Collagenases are enzymes that break the peptide bonds in collagen.

collimated light: Light with parallel light rays that therefore spreads slowly as it propagates.

compression therapy: Treatment using devices or techniques that decrease the density of a part of the body through the application of pressure.

compressive joint motion: Compressive or approximation accessory movements of joint surfaces.

conduction: Transfer of heat by direct contact between two objects, for example, a hot pack.

conduction velocity: The speed at which electrical energy is transmitted.

consultation: The provision by a physical therapist or veterinarian of a professional, expert opinion or of advice.

continuous passive motion (CPM): The use of a device that allows a joint (such as the knee) to be exercised without the involvement of the patient, often in the early postoperative period.

contracture, joint: Inability to move a joint through its full range of motion.

contrast bath: Alternately immersing the patient or body part in cold and hot water, most appropriate during the early subacute phase of tissue healing.

convection: Transfer of heat using a liquid or gas medium.

cosmesis: A concern in therapeutics, especially in surgical operations, for the appearance of the patient.

cranial: In relation to animals, a directional term indicating "toward the head."

cranial cruciate ligament (CCL): Analogous to the anterior cruciate ligament in humans.

cranial nerve: One of the 12 paired nerves (such as olfactory and optic) that emerge from or enter the brain.

critical inquiry: The process of applying the principles of scientific methods to read and interpret professional literature; participate in research activities; and analyze patient care outcomes, new concepts, and findings.

cryokinetics: The combination of cryotherapy with motion (passive, active-assisted, active) to facilitate normal, pain-free movement and to promote reduction of edema.

cryotherapy: Therapeutic application of cold (such as ice).

cyanosis: A bluish or purplish discoloration of the skin or mucous membranes caused by a severe oxygen deficiency.

cyclooxygenase (COX): An enzyme necessary for the production of prostaglandins from arachidonic acid. Two forms of the enzyme are recognized, COX-1 and COX-2. COX-2 is produced in response to inflammation.

debridement: Excision of contused and necrotic tissue from the surface of a wound. *Autolytic:* Self-debridement, that is, removal of contused or necrotic tissue through the action of enzymes in the tissue. *Sharp:* Debridement using a sharp instrument.

deficit: A shortfall in amount or quality. *Developmental:* Difference between expected and actual (lower) performance in an aspect of development (e.g., motor). *Receptive:* A shortfall in the skills involving reception (e.g., in vision, in hearing).

degenerative lumbosacral stenosis: A condition in which soft tissue and bony changes, possibly in conjunction with abnormal motion of the lumbosacral joint, impinge on the nerve roots of the cauda equina.

degenerative myelopathy: A poorly understood neurologic condition associated with progressive motor weakness caused by progressive spinal cord degeneration.

developmental delay: The failure to reach expected age-specific performance in one or more areas of development (such as motor or sensory-perceptual).

diagnosis: A label encompassing a cluster of signs and symptoms, syndromes, or categories. It is also the decision reached as a result of the diagnostic process, which is the evaluation of information obtained from the patient examination organized into clusters, syndromes, or categories.

discospondylitis: An infection of the intervertebral disk and adjacent vertebral bodies, resulting in an inflammatory response surrounding the associated spinal cord or nerve roots.

dislocation: A disturbance or disarrangement of the usual relationship of bones as they enter into the formation of a joint.

dispersion: The property by which light is spread out according to its color as it passes through an object.

distraction joint motion: Distraction or traction motions are tensile (pulling apart) movements between bones.

divergence: The beam divergence of a laser beam is a measure for how fast the beam expands. Beams with very small divergence (i.e., with approximately constant beam radius over significant propagation distances) are called *collimated beams.*

dolorimeter: A device to measure pain.

dorsal: In animals, a directional term meaning "toward the spine."

dressing: A material (such as a topical agent or gauze) applied to a lesion.

duty cycle: Refers to the percentage of time that ultrasound is emitted during one pulse period. Duty cycle = pulse duration (time on)/the pulse period (time on + time off).

dynamometry: Measuring the degree of muscular power.

dyspnea: Shortness of breath; subjective difficulty or distress in breathing frequently manifested by rapid, shallow breaths; usually associated with serious disease of the heart or lungs.

edema: An accumulation of fluid, often occurring as part of the inflammatory process after trauma.

efferent: Sending information away from the central nervous system.

effleurage: A French word meaning "to skim" or "to touch lightly on," referring to a series of massage strokes used in Swedish massage to warm up the muscle before deep-tissue work using petrissage.

effusion: The escape of fluid into a body part or tissue.

electrical device: An instrument or modality that applies electrical current to biologic tissue for pain control, tissue healing, or muscle dysfunction; an instrument that records electrical activity from excitable tissues of the body for purposes of neuromuscular diagnosis, education, or relaxation.

electrical stimulation: Treatment through the application of electricity. *Functional:* The application of electrical stimulation to particular peripheral nerves to allow paretic and paralyzed muscles to make functional and purposeful movements.

electrogoniometry: The measurement of the movement of a joint using an electrical potentiometer.

electromyography (EMG): The recording of the electrical activity of a muscle.

electrophysiologic: Concerned with the electrical activity of various body tissues or systems.

electrophysiologic testing: The process of examining the relationships of body functions to electrical phenomena, such as the effects of electrical stimulation on the tissues, the production of electrical currents by organs and tissues, and the therapeutic use of electrical current.

electrotherapeutic modalities: A broad group of therapeutic physical agents (e.g., neuromuscular electrical stimulation, iontophoresis).

endurance: The ability to perform work over time.

environmental, home, and work barriers: The physical impediments that keep individuals from functioning optimally in their surroundings, including safety hazards (such as throw rugs or slippery surfaces), access problems (such as narrow doors or high steps), and home design (such as a multiple-story environment).

evaluation: A dynamic process in which the physical therapist makes clinical judgments based on data gathered during the examination.

evaporation: The change of a liquid into a vapor at a temperature below the boiling point. Evaporation takes place at the surface of a liquid, where molecules with the highest kinetic energy are able to escape. When this happens, the average kinetic energy of the liquid is lowered, and its temperature decreases.

evoked potentials: The electrical signals recorded from a sensory receptor, nerve, muscle, and so forth, of the central nervous system that has been stimulated, most often by electricity (e.g., auditory evoked potentials).

examination: The process of obtaining a patient history, performing relevant systems reviews, and selecting and administering specific tests and measures.

excursion: Movement within the body, with return to the original state implied (e.g., excursion of the diaphragm).

extracapsular suture stabilization: A procedure used to stabilize cranial cruciate deficient stifles using sutures of various materials to span from the caudal aspect of the stifle to the tibial crest.

exudation: The process of expressing material through a wound, usually characterized as oozing.

fibrocartilaginous embolic myelopathy (FCE): A poorly understood phenomenon in which fragments of intervertebral disk material embolize and create regional vascular compromise to the spinal cord.

fibrosis: Fibrosis is the formation of excess fibrous connective tissue in an organ or tissue in a reparative or reactive process.

fibrotic myopathy: Fibrosis and contracture of skeletal muscle secondary to trauma.

field trails: A sporting event in which dogs compete in hunting, retrieving, or pointing competition.

fluidotherapy: "Dry whirlpool"; the application of dry heat through a fluidotherapy machine.

force plate: A plate embedded in the floor used to measure the force that an individual exerts when walking.

fragmented medial coronoid process (FCP): A condition in which the medial coronoid process of the proximal ulna becomes fragmented, possibly because of elbow incongruency or osteochondrosis.

frequency: The number of cycles per second, expressed as hertz (Hz). Ultrasound and neuromuscular electrical stimulation units generally have various frequency settings.

function: The special, normal, or proper action of any part or organ; the action specifically for which an individual or thing is fitted or employed; an act, process, or series of processes that serve a purpose; to perform an activity or to work properly or normally.

functional limitation: A restriction of the ability to perform a physical action, activity, or task in a typically expected, efficient, or competent manner.

gait: The manner in which a dog walks, characterized by rhythm, cadence, step, stride, and speed.

glide joint motion: Shear or sliding motions of opposing articular surfaces.

goal: The long-term statement or statements that define the patient's expected level of performance at the end of the rehabilitation process; the functional outcomes of therapy, indicating the amount of independence, supervision, or assistance required and the equipment or environmental adaptation necessary to ensure adequate performance. Desired outcomes may be stated as long-term or short-term as determined by the needs of the patient and the setting.

goniometry manual: The measurement of the movement of a joint by manual methods. *Electrical:* See *electrogoniometry.*

graded forces: A term used in manual therapy to denote the application by the physical therapist of varying amounts of pressure on the patient's body.

Hansen's type I disk: An acute explosive intervertebral disk herniation.

Hansen's type II disk: A chronic progressive intervertebral disk herniation.

herniated disk: The protrusion of one of the spinal disks into an opening in the spinal cord, thereby compressing the nerve root.

Hertz (Hz): Cycles per second.

history: An account of past and present health status that includes the identification of complaints and provides the initial source of information about the patient. The history also suggests the patient's ability to benefit from physical therapy services.

home, environmental, or architectural barriers: The physical impediments (such as stairs or slippery surfaces) that restrain or obstruct an individual's ability to function in the usual environment.

Hubbard tank: A shallow tank made of stainless steel, Plexiglas, or tile for administering hydrotherapy.

hydrodynamic: Concerned with the flow of liquids.

hydrotherapy: Treatment with external water.

hygroma: Seroma, hematoma associated with the olecranon.

hypermobility: Abnormally high movement or ability to move (as of a joint).

hypertrophic osteodystrophy: A developmental disease of rapidly growing large- and giant-breed dogs characterized by metaphyseal new bone formation.

hypomobility: Abnormally low movement or ability to move (as of a joint).

impairment: A loss or abnormality of physiologic, psychological, or anatomic structure or function.

infrared heat: A therapy using thermal radiation with a wavelength greater than that of the red end of the visible spectrum.

infraspinatus contracture: Fibrotic contracture of the infraspinatus muscle causing a gait abnormality. Contracture of the infraspinatus muscle limits full extension of the shoulder and causes the affected forelimb to be abducted.

innervation: The supply of nerve fibers to a part of the body, such as an organ.

integrity: The characteristic of being whole or fully functional.

intensity: Refers to the rate of energy delivery per unit area.

intervention: The purposeful and skilled interaction of the physical therapist with the patient, using various methods and techniques to produce changes in the patient's condition.

intervertebral disk disease: A common condition, especially in chondrodystrophic breeds of dogs, in which protrusion or extrusion of an intervertebral disk causes pain, weakness, and varying degrees of neurologic dysfunction.

iontophoresis: Use of continuous direct electrical current to enhance the transdermal administration of medication.

ischemia: Local anemia caused by mechanical obstruction (mainly arterial narrowing) of the blood supply.

jack: Local irritation, seroma, or chronic periostitis associated with mechanical interference between the forelimbs and hindlimbs.

joint integrity: The conformance of the joints to expected anatomic, biomechanic, and kinematic norms.

joint mobility: The ability to move a joint; takes into account the structure and shape of the joint surface as well as characteristics of tissue surrounding the joint.

kinematic: Having to do with the possible motions of a part or all of the human body.

kinesthesia: The awareness of the body's or a body part's movement.

knocked-up toe: Abnormal carriage of the toes caused by avulsion or rupture of the deep digital flexor tendon.

laser: Short for light amplification by stimulated emission of radiation. A device that creates and amplifies electromagnetic radiation of a specific frequency through the process of stimulated emission. The radiation emitted by a laser consists of a coherent beam of photons, all in phase and having the same polarization.

lateral splint: Splint fashioned from casting material applied to the lateral aspect of the leg.

laxity: Looseness, as in laxity of joint.

lipoxygenase (LOX): An enzyme that converts arachidonic acid to leukotrienes.

loading: The force placed on a body part (such as a foot or the feet); used often in describing the employment of an assistive device.

lure coursing: A sporting event in which sight hounds chase lures pulled by a rope or cord.

lymphatic: Concerned with the lymph nodes and vessels, which form a system for collecting fluid from the tissues and adding it to the venous blood system.

maceration: Softening by the action of a liquid.

magnetic fields energy, pulsed: A therapy using the intermittent application of energy produced by magnetic fields.

manipulation: A therapeutic movement, usually of small amplitude, accomplished at the end of the available range of motion but with the anatomic range at a speed over which the patient has no control.

manual therapy: A broad group of skilled hand movements used by the physical therapist to mobilize soft tissues and joints for the purpose of modulating pain, increasing range of motion, and so forth.

mechanical: Caused by or derived from machinery; habitual, routine, automatic; related to, controlled, or affected by physical forces (such as a traction device). *Biomechanical:* The physical structure, forces, and movements in the human body.

medial buttress: Periarticular fibrosis and swelling of the medial aspect of the distal femur associated with chronic stifle instability and cranial cruciate deficiency.

meniscal release: Involves cutting the medial meniscus just caudal to the medial collateral ligament. Used in conjunction with the tibial plateau leveling osteotomy procedure.

mentation: A mechanism of thought or mental activity.

metabolic: Concerned with metabolism, the sum of all physical and chemical changes that take place within an organism; all energy and material transformations that take place within living cells.

microvolt: One millionth of a volt.

mobilization: A therapeutic movement accomplished within the available range of motion at a speed that the patient cannot control.

modality(ies): Physical agent(s), including, but not limited to, thermal, acoustic, light, mechanical, or electrical energy, applied to produce therapeutic changes in biologic tissue.

monkey muscle: Long head of the triceps.

motor function: The ability to learn or demonstrate the skillful and efficient assumption, maintenance, modification, and control of voluntary postures and movement patterns. *Fine:* Refers to relatively delicate movements. *Gross:* Refers to larger-scale movements.

multisegment motion: Simultaneous movement of several parts of the body.

muscle length: The length of the muscle during various stages of tension (from resting at full extension through the contractile range); in conjunction with joint integrity and connective tissue extensibility, muscle length determines flexibility.

muscle performance: The capacity of a muscle to do work (force × distance).

nerve root compression: A squeezing of one of two bundles of nerve fiber emerging from the spine; frequently caused by a herniated disk.

nervous system: The brain, spinal cord, nerves, and ganglia. *Central nervous system:* The brain and spinal cord. *Peripheral nervous system:* The system of nerves in the extremities.

neural: Having to do with a nerve or nerves.

neurapraxia: Axonal conduction is lost without disruption of the axon. This injury usually results from compression, transient ischemia, or blunt trauma. Loss of conduction may be a result of myelin damage or insufficient energy to maintain axonal resting potential.

neuromotor development: The acquisition and evolution of movement skills throughout the life span.

neurotmesis: The entire structure of the nerve is disrupted. The axon has the ability to regenerate but needs to find a Schwann cell sheath to do so, making it much more difficult.

nonvolitional: Involuntary, not controllable.

nutraceutical: Nondrug substances that are produced in a purified or extracted form and are administered

orally to provide compounds required for normal body structure and function with the intent of improving health and well being.

objective: A measurable behavioral statement of an expected response or outcome; something worked toward or striven for; a statement of direction or desired achievement that guides actions and activities.

open kinetic chain exercises: Exercises with the foot not fixed against a resistance.

orthosis: A device (such as a splint or brace) that supports weak or ineffective joints or muscles.

osteochondrosis: A growth-related disease of the cartilage. The primary problem is failure of endochondral bone formation that allows the articular cartilage to irregularly develop and often tear. A cartilage flap is referred to as *osteochondrosis dissecans.*

osteokinematic joint motion: Primary motion of a joint and the movement of bones as a whole, such as occurs with stifle flexion and extension.

osteoporotic: Pertaining to or characterized by a porous condition of the bones; refers to a reduction in the quantity of bone or atrophying skeletal tissue.

oxygen consumption: The amount of oxygen inspired minus the amount of oxygen exhaled.

oxygen saturation: The degree to which oxygen is present in a particular substance.

outcome: The result of physical therapy management expressed in five areas: prevention and management of symptom manifestation, consequences of disease (impairment, disability, or role limitation), cost-benefit analysis, health-related quality of life, and client satisfaction. A successful outcome includes improved or maintained physical function when possible, slows functional decline when the status quo cannot be maintained, or is considered meaningful by the client.

outcomes analysis: A systematic examination of patient outcomes in relation to selected patient variables (e.g., age, sex, diagnosis, interventions performed); outcomes analysis may be used in quality assessment, economic analysis of practice, and so forth.

pain: A disturbed sensation causing suffering or distress.

palpation: Examination using the hands (such as palpation of the spleen).

panosteitis: A growth-related problem of immature rapidly growing dogs. It is characterized by intramedullary deposition of bone and a shifting leg lameness.

paraffin bath: A superficial heat treatment using paraffin wax and mineral oil.

passive range of motion: Motion across a joint with no muscle contraction.

pathomechanical: Describing a disturbance in function not resulting from disease.

pathophysiologic: Describing the functional changes that accompany a particular disease or syndrome.

pathway: A conduction route for nerve impulses. *Sensory pathway:* A conduction route for nerve impulses from the sense organs.

patient: One who is being treated for an illness or injury; an individual receiving health care.

percussion (mechanical): A diagnostic procedure in which the clinician taps a body part with a finger or rubber-headed hammer to estimate its density.

performance battery: A set of tests designed to measure a patient's ability to function in a particular area(s).

peripheral circulation: The movement of blood through the extremities.

peripheral vascular: Concerned with the blood vessels of the extremities.

petrissage: Massage movements with applied pressure which are deep and compress the underlying muscles. Kneading, wringing, skin rolling, and pick-up-and-squeeze are the petrissage movements

phonophoresis: The use of ultrasound to enhance the delivery of topically applied medications through the skin.

photosensitivity: Sensitivity of the skin to light, usually due to the action of certain drugs (or plants, or other substances).

phototherapy: Treatment using the application of light. *Ultraviolet:* Light therapy using rays with wavelengths beyond the violet end of the visible spectrum.

physical agent: A form of mechanical, radiant, thermal, acoustic, or electrical energy that is applied to biologic tissues in a systematic manner to achieve a therapeutic effect; a therapeutic modality used to treat physical problems.

physical function: The measurement of physiologic, biomechanical, social, and psychological performance in practical or goal-oriented terms.

physical therapist: A licensed health professional who offers services designed to preserve, develop, and restore maximum physical function.

physical therapist assistant: An educated health care provider who performs physical therapy procedures and related tasks that have been selected and delegated by the supervising physical therapist.

physical therapy aide: A nonlicensed worker, trained under the direction of a physical therapist, who performs designated routine physical therapy tasks.

physiologic joint motion: See *osteokinematic motion.*

planes (midline and segmental): Imaginary flat surfaces drawn through the body; the midline plane bisects the body vertically, whereas segmental planes are drawn at various angles.

posterior: Relating to the back or dorsal side.

postural drainage: Placing the body in a position that causes fluid to drain from the lungs.

postural reactions: The adjustments of the body to gravity required for normal performance; the ability to alter the position of the head, trunk, and extremities to balance one's body with gravity.

posture: The alignment and positioning of the body in relation to gravity, center of mass, and basis of support.

posturography: Procedures to test standing posture, balance, and equilibrium sense.

power: Work produced per unit of time.

presenting problem: The specific dysfunction that causes a client's owner to seek attention or intervention (that is, the chief complaint).

proactive: Seizing the initiative; responding actively rather than passively; performing an action with the idea of influencing events.

prognosis: The determination of the level of maximal improvement that might be attained by the patient and the amount of time needed to reach that level.

proprioception: The reception of stimuli from within the body (such as from muscles or tendons); includes position sense (the awareness of the joints at rest) and kinesthesia (the awareness of movement).

prosthesis: An artificial device, often mechanical or electrical, used to replace a missing part of the body.

pulses: The dilations of arteries (occasionally veins or vascular organs) that correspond to the beating of the heart.

quadriceps tiedown: A disability characterized by an inability to fully flex or extend the stifle following a fracture of the femur.

radiation: Streams of photons, electrons, small nuclei, or other particles. Radiation is given off by a wide variety of processes, such as thermal activity, nuclear reactions (as in fission), and by radioactive decay.

range of motion: Describes the space, distance, or angle through which a patient can move a joint or series of joints.

recurvatum: An angular limb deformity in which the affected joint is excessively hyperextended. The most common form is genu recurvatum, in which the stifle joint is abnormally hyperextended.

reepithelialization: Skin growth to replace skin loss caused by a wound or other injury.

referral: A recommendation that a patient seek service from another health care provider or resource.

reflex: A stereotyped reaction to a variety of sensory stimuli.

reflex inhibition: A situation in which sensory stimuli decrease reflex activity or the voluntary activation of muscle.

rehabilitative: Concerned with restoration of a patient to full or at least improved function.

remediation: The act or process of providing some degree of relief for a patient's clinical problems.

respiration: A term that refers primarily to the exchange of oxygen and carbon dioxide across a membrane into and out of both the lungs and cells.

respiratory quotient: The ratio of the carbon dioxide that the body tissues give off to the amount of oxygen that they absorb.

righting: Adjusting or restoring the body to a desired position.

Robert Jones bandage: A soft, padded bandage to limit tissue swelling and joint motion.

Robinson sling: A bandage used to allow only toe touching of the rear leg. Sometimes called a *Humane Society bandage.*

roll joint motion: Involves one bone rolling on another, such as the femoral condyles rolling on the tibial plateau.

Schroeder-Thomas splint: A splint consisting of a metal hoop and stirrups used to immobilize fractures.

screening: Determining the need for further examination or consultation by a physical therapist or for referral to another health professional.

sensory: Having to do with sensations or the senses; includes peripheral sensory processing (such as sensitivity to touch) and cortical sensory processing (such as two-point and sharp/dull discrimination).

sensory integration: The ability to integrate information from the environment to produce normal movement outputs.

sequential casting: A process in which the patient is casted several times, with each cast less restrictive than the previous one.

sequelae: Aftereffects of a disease or injury.

serosanguineous: Containing both serum and blood.

serous: Like serum, watery.

somatosensory: Having to do with the sensations received in the skin and deep tissues.

somatosensory deficit: A shortfall in the reception of sensations in the skin and deep tissues.

spatial average intensity: In reference to therapeutic ultrasound, the total output (in watts) of the transducer divided by the transducer surface area (in cm^2).

spatial peak intensity: The greatest intensity anywhere within the therapeutic ultrasound beam.

spin joint motion: Joint surface motions that result in continual contact of a single area of articular cartilage on adjacent articular cartilage within a joint.

spinal curve: An abnormal curvature of the spinal column (e.g., S curve, kyphosis).

splinting, dynamic: Functional splinting that aids in the movements initiated by the patient and/or controls the plane and range of motion.

spoon splint: A plastic or metal splint shaped like a spoon used to immobilize or support the limb.

sprain: A joint injury without dislocation or fracture involving possible ligament or tendon rupture.

stopper bone: Accessory carpal bone.

standing table: See *tilt table.*

strain: Injury from overuse or improper use.

strengthening, active assistive: A form of strength-building exercise in which the physical therapist applies resistance through the range of motion of the patient's active movement.

strengthening, resistive: Any form of active exercise in which a dynamic or static muscular contraction is resisted by an outside force. The external force may be applied manually or mechanically.

sympathetic disturbance: A malfunction in the sympathetic part of the autonomic nervous system, which governs smooth muscle and the contraction of blood vessels.

symptom magnification scale: An examination tool used to elicit descriptions of levels of pain.

syndrome: The aggregate of signs and symptoms associated with any morbid process that together constitute the picture of a known disease.

synergy: The capability of properly grouping movements in order to perform acts that require special adjustments.

systems review: A brief or limited examination that provides additional information about the patient's general health to help the physical therapist formulate a diagnosis and select an intervention program.

telemetry: The science of measuring a quantity, transmitting the results to a distant station, and then interpreting, indicating, and recording the results.

TENS: See transcutaneous electrical nerve stimulation.

tests and measures: General methods and techniques used to conduct an examination.

therapeutic exercise: A wide range of activities designed to increase strength, improve cardiovascular fitness, increase flexibility, enlarge range of motion, or otherwise increase the body's functional capacity.

thermal: Using heat, as in a thermal agent, for its therapeutic effects.

thermistor: A device for determining temperature; may be extremely small and may also be used to establish and maintain temperature.

thermography: A process of measuring temperature by means of a registering thermometer, one form of which records every temperature variation and registers its rise and fall on a circular temperature chart turned by clockwork.

thermotherapy: The therapeutic application of heat.

tibial plateau leveling osteotomy (TPLO): A corrective osteotomy technique used in the treatment of cranial cruciate ligament disease.

tilt table or standing table: Two kinds of tables used to bring patients from a supine to a vertical position in a deliberate manner.

tissue: Collection of similar cells and the intercellular substances that surround them.

topical agent: An ointment, medication, or similar material applied to the skin for its therapeutic effect.

torque: A force that produces rotation or twisting of a part on its axis.

traction: The therapeutic use of tension created by a pulling force. *Mechanical:* The use of tractive forces to produce a combination of distraction and gliding to relieve discomfort and increase tissue flexibility; also called *passive mobilization.*

transcutaneous electrical nerve stimulation (TENS): The use of electrical current to stimulate cutaneous and peripheral nerves via electrodes on the skin's surface.

treatment: One or more interventions used to cure or ameliorate a disease or pathologic condition or otherwise produce changes in the patient's health status; the sum of the therapies offered to a patient during a complete episode of care.

triage: An initial review of a patient or prospective patient to determine the need for further treatment.

triple pelvic osteotomy (TPO): A surgical procedure involving osteotomies of the ilium, ischium, and pubis and rotation of the acetabulum to increase dorsal coverage of the head of the femur for the treatment of hip dysplasia.

turgor: Fullness, swelling.

ulcer: A break in the skin surface or in a mucous membrane with loss of tissue, usually accompanied by inflammation. *Decubitus ulcer:* Bedsore. *Vascular insufficiency ulcers:* Lesions caused by occlusion of a blood vessel or other vascular disorder.

ultrasound: A diagnostic or therapeutic technique using high-frequency sound waves. Used therapeutically, ultrasound produces heat. *Pulsed ultrasound:* The application of therapeutic ultrasound at frequent predetermined levels.

vasopneumatic compression device: A device intended to decrease swelling by "milking" fluid away from an area, such as an inflatable sleeve strapped around a patient's swollen extremity.

Velpeau sling: A bandage used to secure the forelimb to the thorax, used for immobilization of the shoulder.

ventilation: The movement of a volume of gas into and out of the lungs.

ventral: In animals, a directional term meaning "toward the belly."

vestibular: Describing the sense of balance located in the inner ear.

vital signs: Heart rate, blood pressure, temperature, and respiration rate.

volitional: Intentional, as in controlled movement.

volumetric displacement: The amount of a fluid that leaves a container (of any size) following the introduction of a part or all of the body.

wellness: A concept that embraces a proactive, positive approach to good health. Wellness advocates seek to increase a person's level of health as a preventative measure to guard against future disease.

Wobbler syndrome: See *cervical spondylomyelopathy.*

work conditioning: An intensive, goal-oriented treatment program designed specifically to restore an individual's systemic neuromusculoskeletal functions (strength, endurance, movement, flexibility, and motor control) and cardiopulmonary functions.

zoonosis: The transmission of a disease from an animal or nonhuman species to humans.