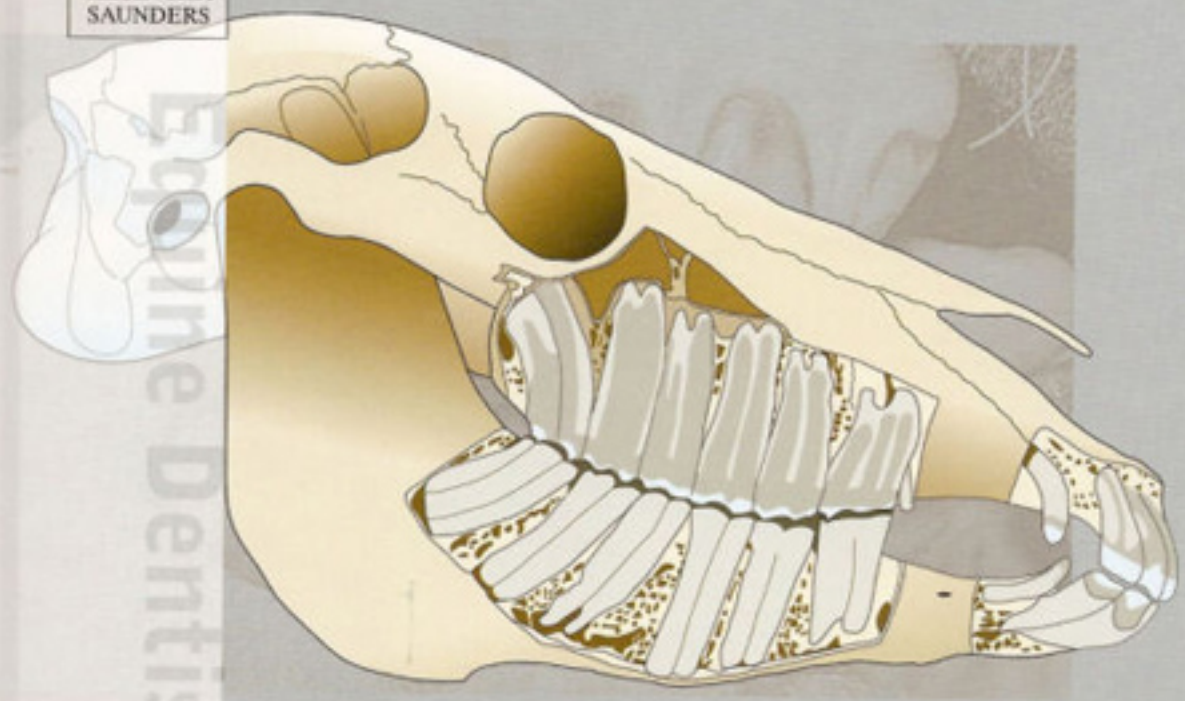
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Equine Dentistry

Second Edition



Edited by
Gordon J. Baker and **Jack Easley**

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Preface and Acknowledgments

First Edition

This textbook could not have been produced without the encouragement and help of many people. We thank our families for their patience and support throughout the writing, editing, and production. We were fortunate to have so many quality colleagues to contribute many chapters in this work. We hope that covering the many topics by worldwide authors has given us a chance to present a thorough documentation of the art and science of Equine Veterinary Dentistry.

We are indebted to the excellence and patience of the editing and production staff of Harcourt Brace (W. B. Sounders, London) and, in particular, to Catriona Byres, Deborah Russell, and to Emily Pillars for their skill in keeping us on–what we hope will prove to be–the right track. We are grateful for the enthusiastic support and work of the staff of the Word Processing and Biomedical Communications Centers of the University of Illinois College of Veterinary Medicine.

Our own interest and enthusiasm for this subject is based on a total of some fifty years of observing, working, "wrestling," and studying the processes of dental structure, function, malfunction, pathology, and the treatment of tooth disorders in the horse. For these experiences, we thank the many colleagues, outside our co-authors, who have been willing to share their ideas with us and to those who have referred cases to us to investigate and treat. We remember with thanks, our equine patients – the creatures who have made it possible to learn, acquire knowledge, demonstrate, and enjoy this exciting profession. It has also been a great pleasure to work with owners and trainers who continually remind us that, just when you think you've seen "it" and understand "it," something else comes along to, in some cases, shed a new or different understanding on a problem or, in other cases, to present a new problem that still awaits a complete investigation.

It also became clear to us, as we worked through the text, that a number of "principles and processes" that have been covered in previous articles and textbook chapters do not hold true under severe scrutiny, i.e., we certainly do not know or understand many things–at best, we only think we know.

The things we know
The things we think we know
The things we don't think we know
The things we wish we knew
The things we hope to know
The things we WILL know

Dr Steve Kneller, University of Illinois, College of Veterinary Medicine, 1996

Consequently, we would like to present this text to our audience, of veterinarians in practice, in research, to veterinary students in training, and to others with an interest in the biology of the lives of horses not as a complete text but, as in all scientific efforts, as a work in progress. It is our sincere hope the information presented in this text will not only benefit the veterinary profession and interest of equine dentistry but more importantly provide the care and consideration our equine friends so rightly deserve. We would encourage readers to commit their views to paper or to cyber space and send us their thoughts, ideas, and suggestions.

A number of the illustrations have been viewed in other media, and we thank the authors, editors, and publishers for permission to use them in this work. This text has four sections: Morphology, Dental Disease and Pathology, Diagnosis of Dental Disorders and Treatment of Dental Disorders, and a total of seventeen chapters. Relevant references follow each chapter and they may be used as a source for further reading and study.

We believe that the use of the modified Triadan numbering system for tooth identification has advantages over traditional nomenclature. It is easier to say or to write 101 than upper right I (incisor) 1. We have used the Triadan system where applicable throughout the text. In some discussions and comments, however, as the reader will see, there is a place for other descriptive terms-the incisors (i.e., all twelve of them in the adult horse), premolars, molars, canine teeth and cheek teeth.

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February 1999.

Preface and Acknowledgments

Second Edition

We wish to thank our colleagues, reviewers, students and everyone interested in the health and welfare of horses for the positive reception of the first edition of Equine Dentistry. This second edition could not have been produced without the encouragement and help of many individuals. Our families were extremely supportive and patient through the writing, editing and production of this second edition. Once again, thanks go to our contributing chapter authors for their knowledge, expertise and hard work in giving us new materials and high-quality illustrations. They performed much of the thankless groundwork that helped make this text complete.

We are indebted to the excellent editing and production staff at Elsevier and in particular, to Joyce Rodenhuis and Zoe Youd for their dedication to this project. Their encouragement, stimulation and of course, patience helped us meet our deadlines. For that aim, we wish to thank Sydney Easley and Christa Petrillo for their essential role in manuscript production and for their skill in ensuring that we, as editors, completed our work.

As was said in introducing the first edition, we have had a combined interest in the clinical aspects of dental diseases in horses of over 50 years. In the second edition several new chapters have been included as a result of our desire to more fully explore the history of equine dentistry and to introduce new materials on the diagnosis, clinical significance, pathology and treatment of dental diseases of the horse.

The field of equine dentistry is ever expanding within the veterinary community. Over the past six years, local, state, national and international veterinary associations and institutions have introduced equine dentistry into their convention programs, continuing education meetings and curricula. We welcome and congratulate them for their efforts to promote this critical aspect of veterinary care. Valuable and constructive ideas and opinions and interesting referrals from our colleagues in equine practice have been appreciated. It is as a result of the continued interest and research both from clinical practice and academic institutions that we feel there is a need and place for this second edition. We would like to present this updated text as a continuation of our understanding of equine dentistry to our audience of veterinarians in practice and research, veterinary students in training and everyone interested in the biology of horses. Dialogue with our readers on this subject is always welcomed and appreciated.

As before, a number of illustrations and novel concepts have been published in journals, texts and proceedings. We thank the authors, editors and publishers for permission to use them in this work.

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1 Equine Dental Evolution: Perspective from the Fossil Record

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Introduction

It is generally believed that horses are native to the Old World and were first brought to North America by the Spanish explorers during the 16th century. While this is correct for historical times, the prehistoric fossil record of horses and their extinct relatives indicates that the Equidae underwent the majority of its evolutionary history in North America from about 55 million years ago (early Eocene) until this family became extinct about 10,000 years ago at the end of the last Ice Age (Pleistocene). The fossil record of horses in North America is a classic and compelling example of long-term (i.e., macro-) evolution.[1,2] Fossil horses were exceedingly widespread and abundant in North America. Their teeth are highly durable and readily fossilize, and therefore figure prominently in our understanding of the evolutionary history of this group. This chapter will review what is known about fossil horse teeth and related morphological adaptations from the rich time sequence in North America to provide the framework within which teeth of modern *Equus* can be understood.

Equid interrelationships and phylogeny

Extant equids (horses, zebras, and asses) and fossil horses are classified in the family Equidae as part of the Order Perissodactyla, or 'odd-toed ungulates.' Other perissodactyl families include tapirs (Tapiridae), rhinoceroses (Rhinocerotidae), and several extinct families. So far as is known, all perissodactyls are united by a suite of unique characters including a concave, saddle-shaped navicular (central tarsal) facet on the astragalus (talus[3]), axis of symmetry through the central metapodial (III), hind-gut fermentation, and particular cheek tooth cusp morphology.[2] Likewise, so far as is known, all perissodactyls living and extinct have been herbivores. With the exception of the extinct clawed chalicotheres, all perissodactlys have a foot terminating with an ungual phalanx that is either padded or hooved.

The eight to ten (i.e., depending upon classification) extant equine species can all be conservatively classified within the single modern genus *Equus*.[4] In contrast to this single genus, about 32 extinct genera and more than 150 species of fossil horses are recognized over the past 55 million years,[2,5] and these also represent a far greater diversity of morphology and adaptations than is seen in modern *Equus*. Fossil horses are first known 55 million years ago during the early Eocene throughout the northern continents (Fig. 1.1). These are represented by *Hyracotherium* (or 'eohippus,' the dawn horse) and a solely Old World group, the palaeotheres (family Palaeotheriidae).[5] Horses persisted in North America after the Eocene, but this family and the horse-like palaeotheres became extinct in the Old World by the early Oligocene, 29 million years ago. During the Oligocene and later times, the major evolutionary diversification of horses occurred in North America. Ancient dispersal events resulted in three-toed (tridactyl) horses immigrating into the Old World during the Miocene 20 million years ago (*Anchitherium*), 15 million years ago (*Sinohippus*), and after 12 million years ago (hipparions; Fig. 1.1). Extinct species of one-toed (monodactyl) *Equus*, which first originated in North America 4.5 million years ago during the Pliocene, subsequently dispersed into the Old World across the Bering Land Bridge 3.5 million years ago.[6] During the Pleistocene after about 2 million years ago, *Equus* species also dispersed into South America after the formation of the Isthmus of Panama. The genus *Equus* subsequently became extinct 10,000 years ago throughout the New World at the end of the last Ice Age (Pleistocene).

Fossil horse dental adaptations

The earliest equid *Hyracotherium* is characterized by the primitive placental mammalian dental formula of three incisors, one canine, four premolars, and three molars (3:1:4:3), both upper and lower. The canine is large and sexually dimorphic.[8] The premolars are primitive in structure, and roughly triangular in shape, whereas the molars are relatively square and have a greater surface area for trituration. During the Eocene and into the Oligocene, fossil horses in North America are characterized by progressive 'molarization' of the premolars (Fig. 1.2), resulting in a functional dental battery consisting of six principal teeth (P2/p2 through M3/m3) for mastication of foodstuffs. The cheek teeth of *Hyracotherium* and other early horses are short-crowned (brachydont). The preorbital cheek region is relatively unexpanded and the mandible is shallow (Fig. 1.3).

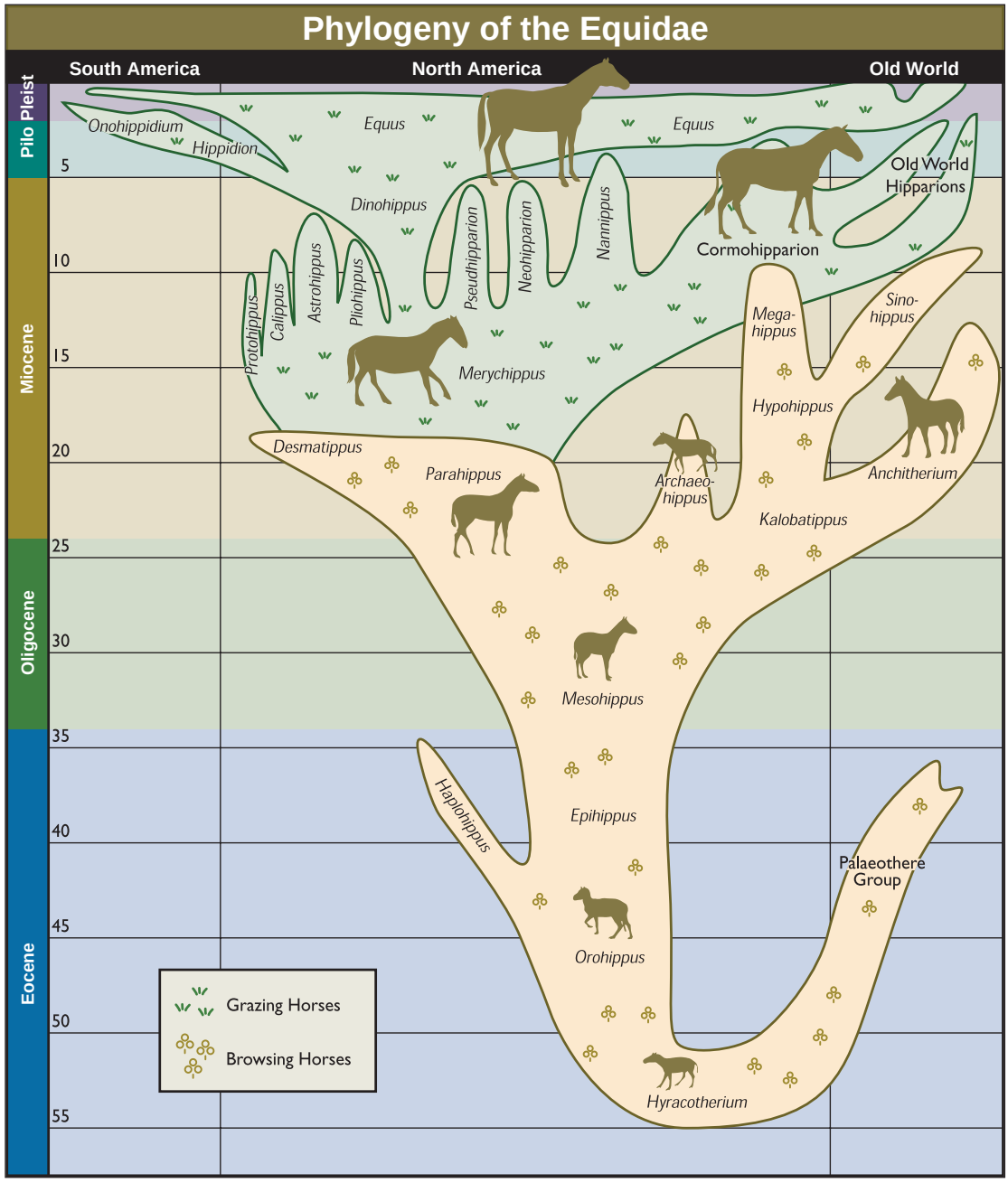


Figure 1.1. Phylogeny of the Equidae. (Modified and updated from refs 2 and 7.)

Studies of dental structure and wear patterns suggest that these early horses were browsers, probably feeding on soft leafy vegetation and groundcover (e.g., including perhaps ferns) in ancient woodlands.[8] This overall dental bauplan and inferred diet continued through the first half of equid evolution from 55 to 20 million years ago. (It also should be noted that grasslands had not yet evolved as principal biome types in North America.[9])

The major morphological evolution of the equid skull and dentition occurred during the middle Miocene, between 20 to 15 million years ago.[10–12] This evolution resulted in a morphology adapted for grazing, including a relatively longer cheek tooth row and deeper skull and jaws accommodating high-crowned (hypsodont) teeth. Miocene and later horses with hypsodont teeth are principally interpreted to have been grazers. Hypsodont teeth are well adapted to increased wear resulting from eating abrasive grasses (in contrast to soft browse), as well as ingesting contaminant grit from plants growing close to the soil substrate. Evidence from the fossil plant record indicates that grasslands became a dominant biome in North America during the middle Cenozoic[9] and horses soon thereafter exploited this newly

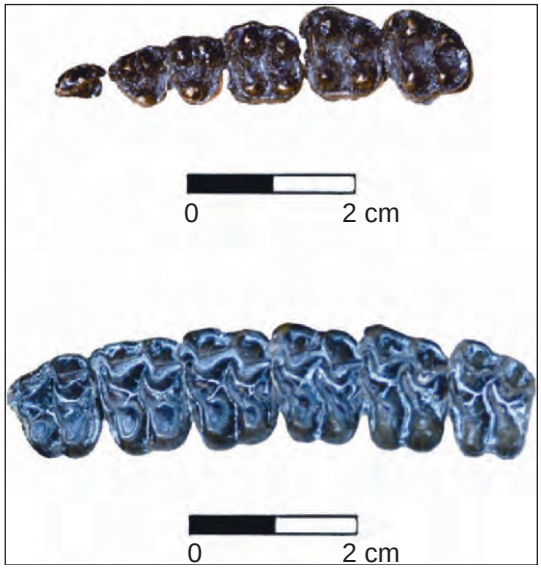


Figure 1.2. Upper cheek tooth dentitions (excluding anterior-most P1) of Eocene *Hyracotherium* (top) compared with Oligocene *Mesohippus* (bottom). Note that relative to the triangular-shaped premolars (P2–P4; i.e., left three teeth in row) in *Hyracotherium*, those of *Mesohippus* are more square, or 'molarized.'

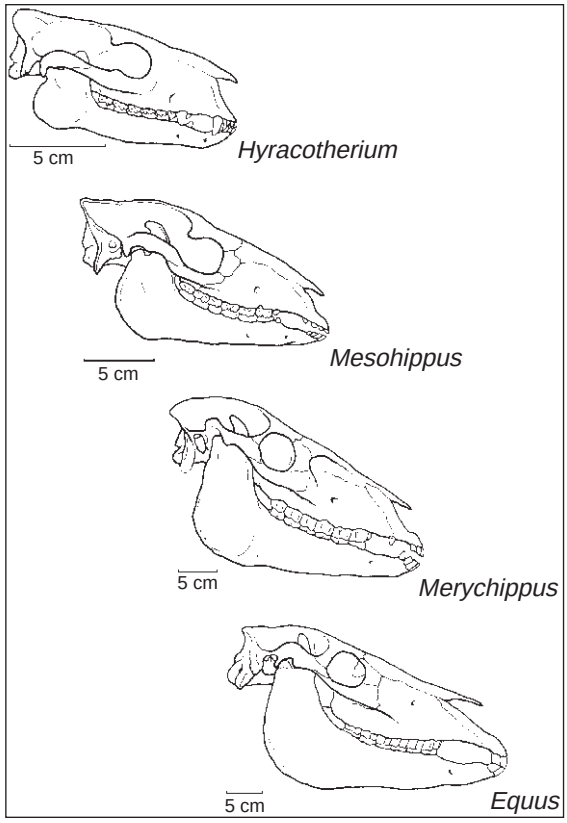


Figure 1.3. Changes in the cranial proportions of the family Equidae as represented in Eocene *Hyracotherium* (top), Oligocene *Mesohippus*, Miocene *Merychippus*, and Pliocene – modern *Equus* (bottom).[10,11] (From ref. 2 and reproduced with permission of Cambridge University Press.)

available food resource as they invaded the 'grazing adaptive zone,'[1] i.e., they became hypergrazers (Fig. 1.4).[13,14] The maximum diversity of horses occurred during the middle Miocene when some dozen genera coexisted at some North American fossil localities.

The direct correlation between high-crowned teeth and grazing in horses is not absolute.[15] Recent studies of the carbon content preserved in fossil hypsodont horse teeth indicate that some coexisting equid species secondarily acquired partial browsing diets.[16] The extant genus *Equus* is first known 4.5 million years ago during the Pliocene from North America. It has a hypsodont dental battery and elongated and deepened skull and jaws, all of which are characters adapted for grazing (Fig. 1.3).

Trends in dental evolution

Number of teeth

Primitive equids from the Eocene have a dental formula of 3 I/i, 1 C/c, 4 P/p, and 3 M/m. The cheek teeth, consisting of the premolars and molars, represent the functional dental battery for post-cropping mastication. During equid evolution the anterior-most cheek teeth, P1/p1, were either reduced to small, relatively functionless teeth, or lost completely. In *Equus* the P1, or wolf tooth, is rudimentary, or often absent. The corresponding p1 is characteristically absent.[3,17] Like most other mammalian families in which there is little evolutionary variation in the dental formula, other than the variable presence of the first premolar, equids are relatively constant in the dental formula throughout their phylogeny.

Histology

The teeth of primitive horses demonstrate three primary dental tissues: pulp, dentin, and enamel. The composition of each of these dental tissues is developmentally very conservative, i.e., there is little variation in mammals, including equids.[18] Composed of collagen, connective tissue, and reticulin fibers, pulp is the relatively soft tissue located in the center of the tooth,[19] but is not normally exposed on the occlusal surface unless the tooth is heavily worn. Enamel and dentin are characterized by an inorganic component consisting of the mineral hydroxyapatite (the primary constituent of vertebrate bone). Enamel is more than 95 per cent hydroxyapatite, whereas dentin is about 80 per cent mineral, the remaining portion consisting of organic compounds, mostly collagen.[20] Minor chemical variations in fossil teeth result primarily from changes in diet, difference in climate, and the source elements available in the animals' environments. Considerable infolding of the enamel occurs in later, hypsodont horses, resulting in a more durable tooth surface. Cementum, the external dental tissue in extant horses, first appeared during the Miocene in advanced species of *Parahippus*, and thereafter it was characteristically developed in hypsodont species (Fig. 1.5). Cementum is seen in numerous



Figure 1.4. Reconstruction of a Miocene savanna grassland in North America showing a diversity of horse species, as they might have existed in a local community. (From ref. 13 and reproduced with permission of the American Museum of Natural History.)

herbivorous mammalian groups and functions to provide an additional occlusal surface for mastication of abrasive foodstuffs, i.e., principally grasses.[21]

Dental ontogeny and wear

Most ungulates, including horses, are characterized by determinant dental growth of two sets of premolars and one molar series. Likewise, the individual teeth are characterized by growth that is completed during the lifetime of the individual when crown enamel mineralization ends and the roots form. Despite the fact that some mammals, e.g., elephants and manatees, have supernumerary tooth sets, and other mammals, e.g., rodents and lagomorphs, possess teeth that are ever-growing, dental ontogeny in the family Equidae is very conservative. A fixed set of premolars and molars and determinant tooth mineralization during an individual's lifetime is pervasive in fossil horses and *Equus*, with one notable exception. One species of tiny three-toed horse, *Pseudhipparion simpsoni*, from the 4.5-million-year-old Pliocene of Florida, had teeth that were partially ever-growing,[22] thus providing an effective dental battery for feeding on abrasive foodstuffs and potentially increasing individual longevity.

Like modern horses, individuals of fossil equid species can be aged by the relative wear on teeth as represented in large quarry samples presumed to be ancient populations. It also can be determined if breeding was synchronized, thus implying a relatively seasonal ancient environment, or occurred year-round as in more equable climates. In seasonal climates, tooth wear was discontinuous within the population



Figure 1.5. Left partial adult mandible of the three-toed hypsodont horse *Cormohipparion plicate* from the late Miocene (~9 million years old) of Florida showing the deposition of cement (above arrow) on the erupted portion of p2 (above alveolus) and p3–p4 (bone removed).

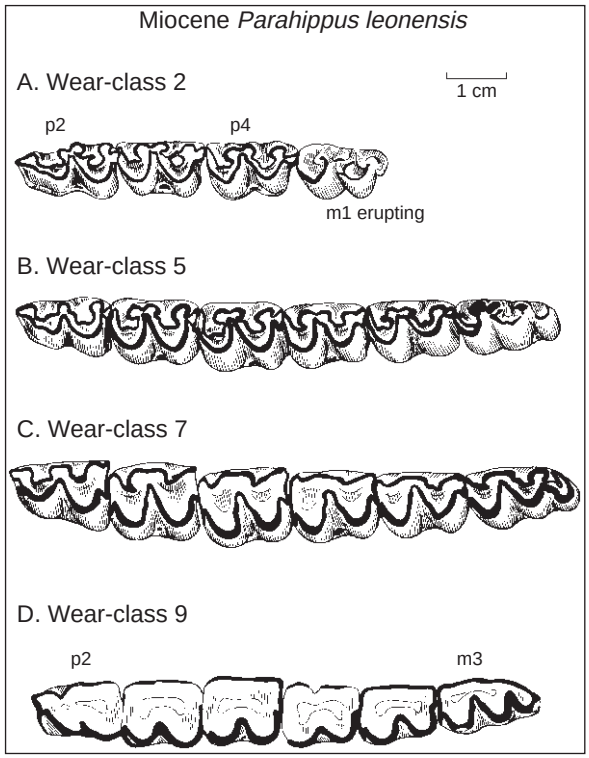


Figure 1.6. Progressive dental wear on the lower cheek teeth of the three-toed horse *Parahippus leonensis* from the 18-million-year-old Thomas Farm locality, Miocene of Florida. The different wear stages shown are interpreted to represent individuals that died at different ages within the same population. The top dentition (A) probably represents an individual about 2 years old, whereas that at the bottom (D) was probably about 9–10 years old when it died. The occlusal enamel pattern is indicated in black. Pulp is exposed in the center of each tooth in Wear-class 9. (Modified from ref. 2 and reproduced with permission of Cambridge University Press.)

because births occurred in annual cohorts, i.e., a group of individuals that all started to wear their teeth about the same time (Fig. 1.6). In contrast, species that lived in equable climates will demonstrate continuous wear because individuals were born at different times during the year.

When horses are aged from fossil sites by the amount of wear on their teeth, we can see that potential individual longevity has evolved since the Eocene (Fig. 1.7). Eocene and Oligocene horses from 55 to 30 million years ago indicate a maximum potential longevity of 4–5 years per individual based on tooth wear and population analysis of *Hyracotherium* and *Mesohippus*. Beginning about 20 million years ago during the Miocene, cohort analyses indicate an increase in potential longevity from 5–15 years depending upon taxon,[2] and thereafter up to 20–25 years per individual during the Pliocene and Pleistocene, as also has been reported for wild populations of *Equus*.[4] As longevity is generally correlated with adult body size in modern mammals,[23] it is not surprising that longevity increased in fossil horses over the past 20 million years because this also was the time of dramatic increases in body size.[24]

Sexual dimorphism

Relative to certain modern mammalian species in which the males can be as much as 30–40 per cent larger than females within a population,[4] the degree and expression of sexual dimorphism as represented in skeletal hard parts is relatively minor in living *Equus*. While male equids are generally larger[23] and have relatively more robust canines, these sexually dimorphic characteristics are much less distinctive than in fossil equids.

A quarry sample of 24 individuals of *Hyracotherium tapirinum* from a 53-million-year-old (early Eocene) locality from Colorado gives great insight into the sexual dimorphism

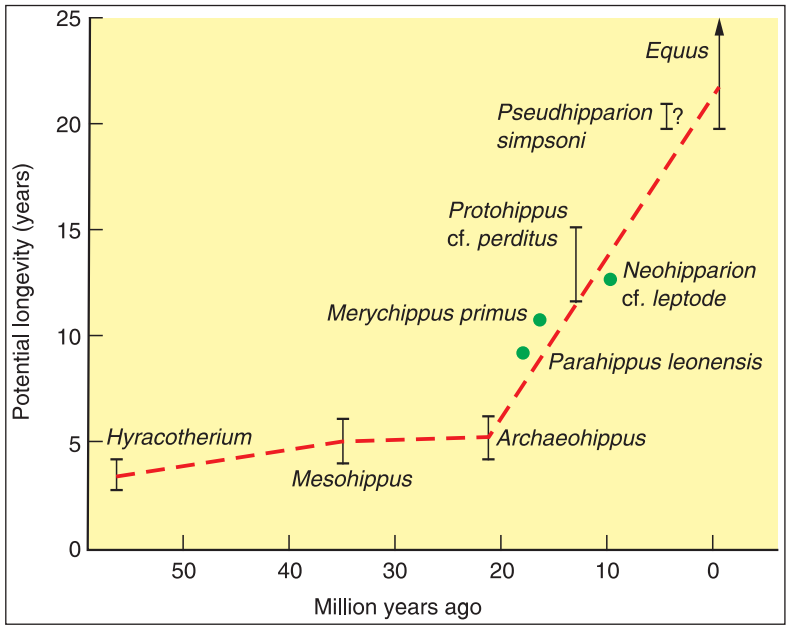


Figure 1.7. Evolution of individual potential longevity in selected species of fossil Equidae based on analysis of the population dynamics of well-preserved quarry samples. (From ref. 2 and reproduced with permission of Cambridge University Press.)

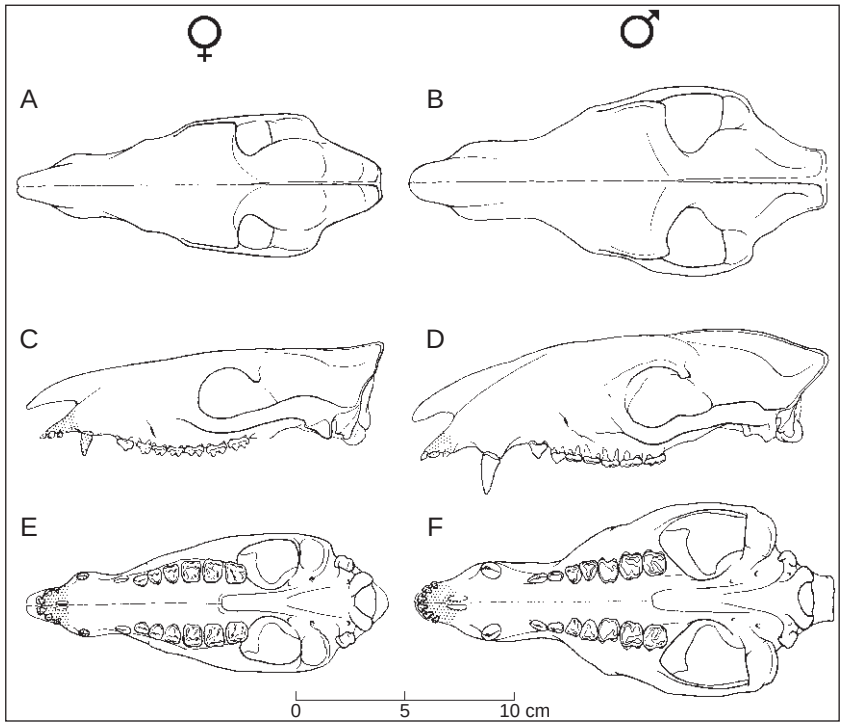


Figure 1.8. Dorsal, left lateral, and ventral views of female (left: A, C, E) and male (right: B, D, F) crania of *Hyracotherium tapirinum* from the 53-million-year old Huerfano Quarry, Eocene of Colorado. These are from the same locality and therefore interpreted to represent individuals within the same ancient population. Note the larger cranium and canine in the male. Shading indicates reconstruction. (Modified from ref. 8 and reproduced with permission of the Paleontological Society.)

in cranial and tooth size in this early horse.[8] The males are on average 15 per cent larger than females, and have markedly robust canines relative to females (Fig. 1.8). Thereafter, during the Eocene through early Miocene, size and canine dimorphism is characteristic of more primitive species for which there are sufficient samples for statistical discrimination. With the evolution of open-country grazing forms during the Miocene, cheek teeth are essentially monomorphic,[25] but sexual discrimination can be seen in the relative canine size (Fig. 1.9). Likewise, in an extraordinary quarry accumulation interpreted to represent an ancient population of *Equus* (*E. simplicidens*), the species close to the origin of the modern genus, from 3.5-million-year-old Pliocene sediments of Idaho,[26] males and females can be distinguished based on relative canine size.

Cranial adaptations

The 55-million-year evolutionary history of the family Equidae is characterized by profound changes in cranial morphology. Primitively, *Hyracotherium* had a skull in which the orbit was centrally located, a postcanine diastema, and a relatively shallow mandible that accommodated short-crowned teeth (Fig. 1.4). In contrast, *Equus* has a preorbital region that is much longer than the postorbital region, a relatively more elongated diastema, and the mandible, which accommodates high-crowned teeth, is very deep. These trends all relate to the fundamental change in diet that occurred from the morphology seen in *Hyracotherium* to that of *Equus*. This evolution, however, was not gradual, and a major morphological reorganization occurred in equid skulls during the Miocene related to the adaptation to grazing.[10,11]

Although not directly related to diet and feeding adaptations, fossil horses show a fundamental evolution in the cheek region over the past 20 million years during the middle Cenozoic. Primitively, *Hyracotherium* has a smooth preorbital cheek region (junction of nasal, maxillary, and lacrimal bones), but during the Miocene there was an

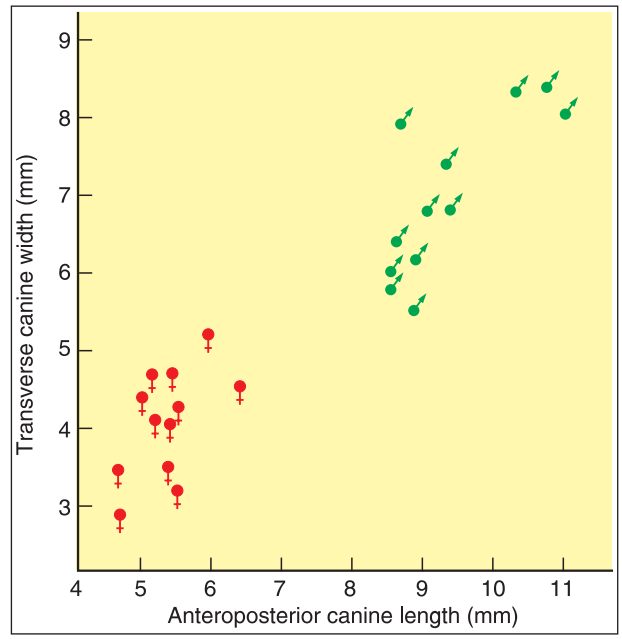


Figure 1.9. Bivariate plot of canine length versus width in a late Miocene quarry sample of the three-toed horse *Hipparion tehonense* from MacAdams Quarry, Texas. The distinctly bimodal populations represent individuals interpreted to represent females (lower left) and males (upper right). (Modified from ref. 27 and reproduced with permission of the American Museum of Natural History.)



Figure 1.10. Adult skull and mandible of 18-million-year-old three-toed short-crowned *Archaeohippus blackbergi* from the Miocene of Thomas Farm, Florida, showing dorsal preorbital fossa (below finger).

adaptive radiation resulting in an elaboration of a pit, or multiple pits, in the facial region. These are collectively termed preorbital fossae, of which the dorsal preorbital fossa is most widespread (Fig. 1.10). Preorbital fossae are absent in living *Equus*, so the function of this structure cannot be based on a modern closely related analog, and has therefore engendered much discussion in the literature. One theory suggests that preorbital fossae housed an organ complex that could have been used for vocalization. The time of maximum morphological diversity of facial fossae is seen at the time of maximum equid diversity during the Miocene. During the Pliocene and Pleistocene, when equid diversity declined, facial fossae became reduced and were ultimately lost in *Equus*.[2]

Summary: modern *Equus*

The cranial and dental adaptations of modern *Equus*, in particular the elongated preorbital region, high-crowned molarized cheek teeth, and deep mandible, represent an integrated character complex related to feeding on abrasive foodstuffs. These morphological adaptations are first seen 20 million years ago during the Miocene when equids exploited the grazing niche during the expansion of grasslands. The 55-million-year fossil record, particularly the ubiquitous and abundant horse teeth, provides fundamental evidence for macroevolution within the family Equidae in North America.

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REFERENCES

1. Simpson GG (1953) *The Major Features of Evolution*. Columbia, New York.
2. MacFadden BJ (1992) *Fossil Horses: Systematics, Paleobiology, and Evolution of the Family Equidae.* Cambridge University Press, New York.
3. Getty R (1975) *Sisson and Grossman's The Anatomy of Domesticated Animals.* WB Saunders, Philadelphia.
4. Walker EP (1975) *Mammals of the World* (3rd edn). Johns Hopkins, Baltimore.
5. McKenna MC and SK Bell (1997) *Classification of Mammals Above the Species Level*. Columbia, New York.
6. Lindsay EH, Opdyke ND and Johnson ND (1980) Pliocene dispersal of the horse *Equus* and late Cenozoic mammalian dispersal events. *Nature*, 287, 135–138.
7. Simpson GG (1951) *Horses: The Study of the Horse Family in the Modern World and through Sixty Million Years of History*. Oxford University Press, Oxford.
8. Gingerich PD (1981) Variation, sexual dimorphism, and social structure in the early Eocene horse *Hyracotherium* (Mammalia, Perissodactyla). *Paleobiology*, 7, 443–455.
9. Jacobs BF, Kingston JD and Jacobs LL (1999) The origin of grass-dominated ecosystems. *Annals Missouri Botanical Garden*, 86, 590–643.
10. Radinsky LB (1983) Allometry and reorganization in horse skull proportions. *Science*, 221, 1189–1191.
11. Radinsky LB (1984) Ontogeny and phylogeny in horse skull evolution. *Evolution*, 38, 1–15.
12. MacFadden BJ and Hulbert RC, Jr (1988) Explosive speciation at the base of the adaptive radiation of Miocene grazing horses. *Nature*, 336, 466–468.
13. MacFadden BJ (1994) The heyday of horses. *Natural History*, 103(4), 63–65.
14. MacFadden BJ (1997) Origin and evolution of the grazing guild in New World terrestrial mammals. *Trends in Ecology and Evolution*, 12, 182–187.
15. Janis CM (1988) An estimation of tooth volume and hypsodonty indices in ungulate mammals, and the correlation of these factors with dietary preference. In*: Teeth Revisited: Proceedings of the VIIth International Symposium on Dental Morphology*, Paris 1986, ed. DE Russell, JP Santoro and D Sigogneau-Russell, *Mémoires Musée National Histoire Naturelle, Paris (série C)*, 53, 367–387.

16. MacFadden BJ, Solounias N and Cerling TE (1999). Ancient diets, ecology, and extinction of 5-million-year-old horses from Florida. *Science*, 283, 824–827.
17. Sach WO and Habel RE (1976) *Rooney's Guide to the Dissection of the Horse*. Veterinary Textbooks, Ithaca, NY.
18. Janis CM and Fortelius M (1988) On the means whereby mammals achieve increased functional durability of their dentitions, with special reference to limiting factors. *Biological Reviews*, 63, 197–230.
19. Dixon PM (1999) Dental anatomy. In: *Equine Dentistry*, eds. GJ Baker and J Easley, WB Saunders, Philadelphia, pp. 3–28.
20. Carlson S (1990) Chapter 21. Vertebrate dental structures. In: *Skeletal Biomineralization: Patterns, Processes and Evolutionary Trends*. Volume 1, ed. JS Carter, New York. Van Nostrand Reinhold, pp. 531–556.
21. White TE (1959) The endocrine glands and evolution, no. 3: Os cementum, hypsodonty, and diet. *Contributions from the Museum of Paleontology, University of Michigan*, 13, 211–265.
22. Webb SD and RC Hulbert, Jr (1986) Systematics and evolution of *Pseudhipparion* (Mammalia, Equidae) from the late Neogene of the Gulf Coastal Plain and the Great Plains. In: *Vertebrates, Phylogeny, and Philosophy,* eds. KM Flanagan and JA Lillegraven, Contributions to Geology, University of Wyoming, Special Paper 3, pp. 237–272.
23. Eisenberg JF (1981) *The Mammalian Radiations: An Analysis of Trends in Evolution, Adaptation, and Behavior.* University of Chicago Press, Chicago.
24. MacFadden BJ (1987) Fossil horses from "Eohippus" (*Hyracotherium*) to *Equus*: scaling, Cope's Law, and the evolution of body size. *Paleobiology*, 12, 355–369.
25. MacFadden BJ (1989) Dental character variation in paleopopulations and morphospecies of fossil horses and extant analogues. In: *The Evolution of Perissodactyls*, eds. DR Prothero and RM Schoch, Clarendon Press, Oxford, pp. 128–141.
26. Gazin CL (1936) A study of the fossil horse remains from the Upper Pliocene of Idaho. *Proceedings US National Museum,* 83, 281–319.
27. MacFadden BJ (1984) Systematics and phylogeny of *Hipparion, Neohipparion, Nannippus*, and *Cormohipparion* (Mammalia, Equidae) from the Miocene and Pliocene of the New World. *Bulletin of the American Museum of Natural History,* 179, 1–196.

2 Bits, Bridles and Accessories

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Introduction

A veterinarian must understand the action and purpose of bridles, bits and accessories (e.g. nosebands and martingales) not only to provide optimal health care to horses' mouths but also to be able to address owners' concerns about their horses' performance. We must be aware of what a horse does for a living, become familiar with what is expected, and provide the kind of dental care required to help horses perform most comfortably and at their best.[1]

Refinements in the way that teeth should be floated depend both upon the job of the horse and the type of bit used. The bitting requirements are different for western performance, English pleasure, polo, jumping, dressage, racing, equitation, driving, etc. For example, the D-ring snaffle is a popular bit for Thoroughbred racing, in which the jockey's hands are above the horse's neck, but this bit is seldom used in Standardbred racing because the angle of pull on the lines (the proper name for the reins of a driving horse) is straight back toward the driver.

The second premolars of a racing Thoroughbred, whose chin must extend to achieve maximum speed, require more rounding than those of a pleasure horse who performs in a nearly vertical head set (compare Fig. 2.1C with Fig. 2.1A and Fig. 2.3C with Fig. 2.3A). A barrel-racing horse in a gag bit requires a deeper bit seat than a cutting horse in a grazer curb bit (compare Fig. 2.7B with Fig. 2.9D).

Proper use of bits and bridles

Bits and bridles are for communication. They are not handles to stabilize the rider in the saddle or instruments for punishing the horse.[2,3] The western horse is ridden with slack in the rein, while the English horse is generally ridden with more contact with the bit, but in either case the accomplished rider uses his seat and legs before his bit to communicate his wishes to his mount. Indeed, the most important factor in having soft, sensitive hands on the reins is developing a good seat.

For the driver of the horse in harness, communication via the seat and legs is not an option. The bridle and lines are the only non-verbal means of communication and thus assume even more importance than they do in the ridden horse.

As with all methods of training and communicating with the horse, the key to the proper use of bits and bridles is the principle of pressure and release. A horse does not intuitively move away from pressure. Rather, he learns to seek a position of comfort to relieve the pressure applied by the bit in his mouth. Consequently, the rein pressure must be released the instant that the horse complies (or even tries to comply) with the request sent to him via the bit. If the pressure is not released, the horse has no way of knowing that his response was correct and becomes confused. When a rider applies rein pressure he is asking the horse for a response, when he releases the pressure he is thanking the horse for complying.[2,4]

Bits, bridles and accessories can exert pressure on a horse's mouth bars (the horseman's term for the lower interdental space), lips, tongue, hard palate, chin, nose and poll. Of these the tongue and the hard palate are the most sensitive and the most responsive to subtle rein pressure. Depending upon the type of headgear used, however, commands sent to the horse via the bars, lips, chin, or nose can be more important than those transmitted via the tongue and palate.

An important concept in bitting is signal, which is defined as the time between when the rider or driver begins to pull on the reins and the time when the bit begins to exert pressure in the horse's mouth. As a horse becomes schooled, he learns to recognize the initial increase in rein pressure and to respond before significant pressure is applied.[3]

Signs of bitting problems

Although cut tongues are the most obvious injuries associated with the improper use of bits, less spectacular injuries to the bars and other tissues are also signs of bitting problems. Tissue trapped by a bit may bunch between the bit and the first lower cheek teeth where it is pinched or cut. The damaged area may then be irritated every time the bit moves.[1] Trauma to the lower interdental space frequently penetrates to the mandible with resulting mandibular periostitis.[5] All types of headgear can press the lips and cheeks against points or premolar caps on the upper cheek teeth.

A horse with a sore mouth or improperly fitting bit will often gape his mouth and pin his ears. He may nod his head excessively or toss his head. He may extend his neck (get ahead of the bit) or tuck his chin against his chest (get

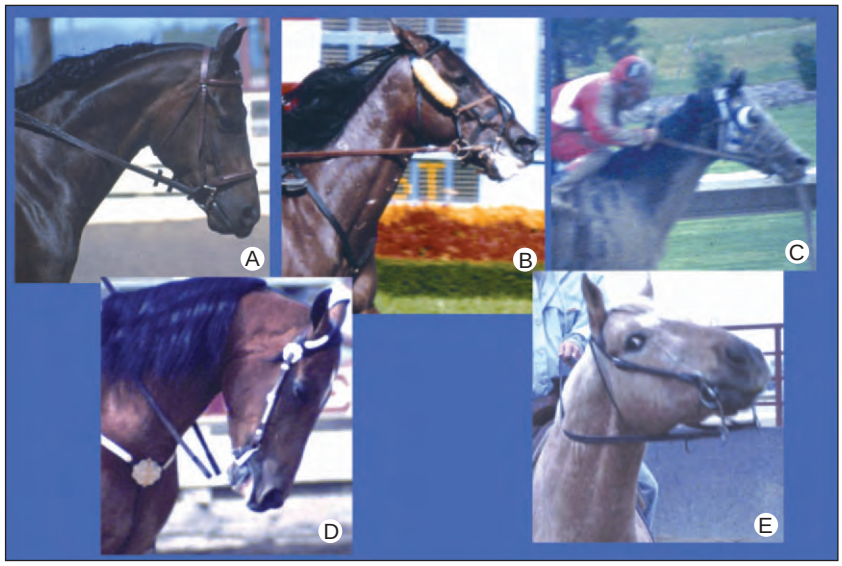


Figure 2.1. The proper head carriage when a horse is 'on the bit' varies depending upon the function of the horse. (A) The pleasure horse with a nearly vertical head set is collected, that is, his weight is shifted to the rear. (B) The racing Standardbred needs to extend his nose to achieve speed but his head position must be controlled to keep him on gait. (C) The racing Thoroughbred, in order to achieve maximum speed, must be able to fully extend his nose and shift his center of gravity forward. (D) This horse is 'behind the bit,' overflexing his chin to his chest to evade bit pressure. (E) This horse is 'ahead of the bit,' overextending his chin to evade bit pressure.

behind the bit) (Fig. 2.1).[4,5] Bitting problems can be mistaken for lameness, as when a horse fails to travel straight.

It is a common misconception that a horse with a painful mouth will be especially sensitive to bit cues. In fact, horses tend to push into pain.[1,4] A horse with bilaterally tender bars may root into the bit. A horse who is sore on one side of his mouth may lean on the bit on the tender side. A vicious cycle can result from attempts to gain such a horse's respect by changing to increasingly severe bits. Oral discomfort causes horses to focus on pain rather than on performance. They may fail to respond to the bit cues, may evade the action of the bit or may ignore the bit completely.[1]

When consulted about a horse that has performance problems, the veterinarian should always inquire about the type of bit used and carefully examine the tongue, lips, bars, palate, chin and nose for subtle signs of injury. It is important to compare the left and right interdental spaces to detect subtle differences.[5,6]

A localized soft and thickened raised area may indicate mandibular periostitis, especially if the horse reacts violently when pressure is applied to it. Techniques such as mental nerve blocks, radiographs, scintigraphy, and computed tomography may be necessary to confirm the presence of this condition. A simple surgical procedure has been described for removing the periostitis and making the horse more comfortable with his bit.[5] Even in the absence of an obvious injury, a change to a gentler bit will often lead to an improvement in a horse's performance.

Mouthpieces

The mouthpiece of a bit may be solid or may have one or more joints. A mouthpiece made up of two or more pieces is referred to as a jointed or broken mouthpiece (Fig. 2.2A). The two halves of a simple jointed mouthpiece are called the

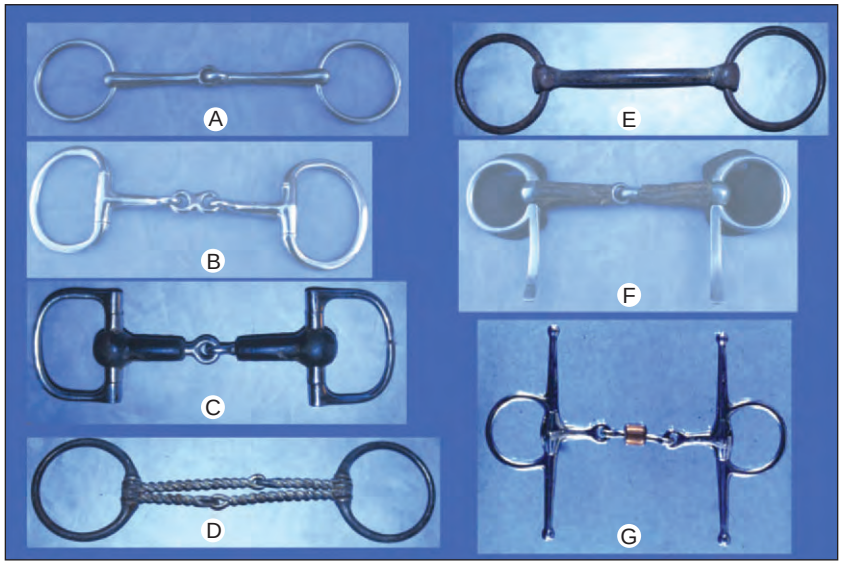


Figure 2.2. Examples of snaffle bits. (A) O-ring with broken mouthpiece. (B) Egg butt with center link in mouthpiece. (C) D-ring with rubber-covered mouthpiece. (D) Fixed ring with double twisted wire mouthpiece. (E) O-ring with solid mullen mouthpiece. (F) Half cheek with leather-covered mouthpiece. (G) Full cheek with cricket in mouthpiece.

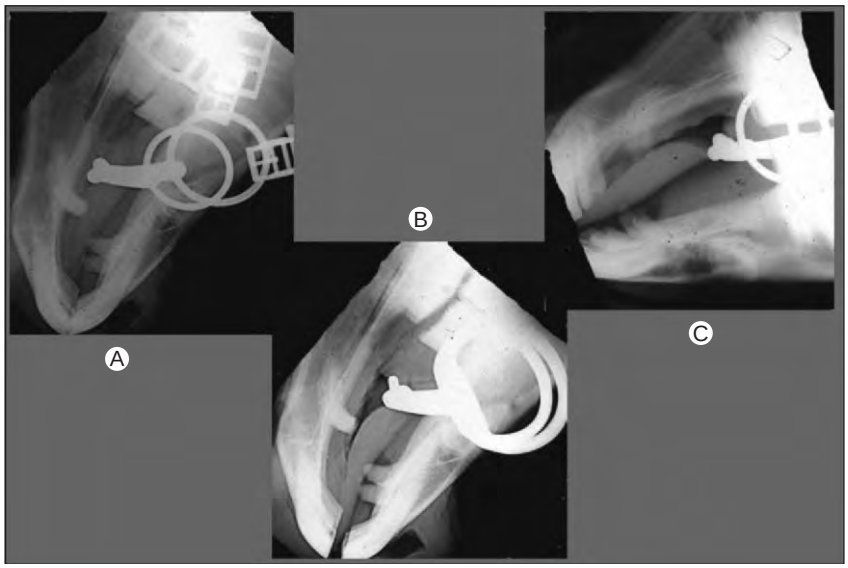


Figure 2.3. Lateral radiographs of snaffle bits under rein pressure. (A) Broken mouthpiece, poll flexed. (B) Center-linked mouthpiece, poll flexed. The extra link transfers pressure from the bars to the tongue. (C) Broken mouthpiece, nose extended. The more a horse's nose is extended, the more likely that his lips will be pinched against his teeth and his tongue will be punished by the bit.

'cannons.' One purpose of the joint is to form a roof over the tongue, which gives the tongue some relief from the pressure of the bit. Another purpose is to change the angle of pull. As the cannons collapse, pressure is transferred from the tongue to the bars and lips. Some jointed mouthpieces (e.g. Dr Bristol and French snaffle) have an extra link between the cannons. The center link creates more room for the tongue, but changes the angle at which the pressure is applied to the tongue, bars and corners of the lips. There is more pressure on the tongue and less leverage on the bars and lips (Fig. 2.3). Of course, the position of the horse's head, which varies depending upon the horse's use, will have a profound effect upon the bit's action (Figs 2.1 and 2.3).

A solid mouthpiece may be straight, curved or ported. One of the most common misconceptions in bitting is that a low port makes a mouthpiece mild and that a high port makes it severe. The error in such a conception becomes evident when we consider that the tongue is the most sensitive part of the horse's mouth and that the purpose of the port is to prevent the bit from applying the majority of its force directly to the tongue (Fig. 2.4). A high port is severe only if it comes into contact with the horse's palate (Fig. 2.7D). In most horses the port must be at least 2–2½ inches high to contact the palate.

A straight, solid mouthpiece can be severe because the tongue takes almost the full force of the pull. The mullen mouthpiece (Figs 2.2E and 2.12A), with its gentle curve from one side to the other, still lies largely on the tongue and gives only a small margin of tongue relief. When using a bit with a straight or mullen mouthpiece, a hard jerk on the reins can easily cut the tongue.

A mouthpiece's severity is inversely related to its diameter. Mouthpiece diameter is measured 1 inch in from the attachment of the bit rings or shanks, because this is the portion

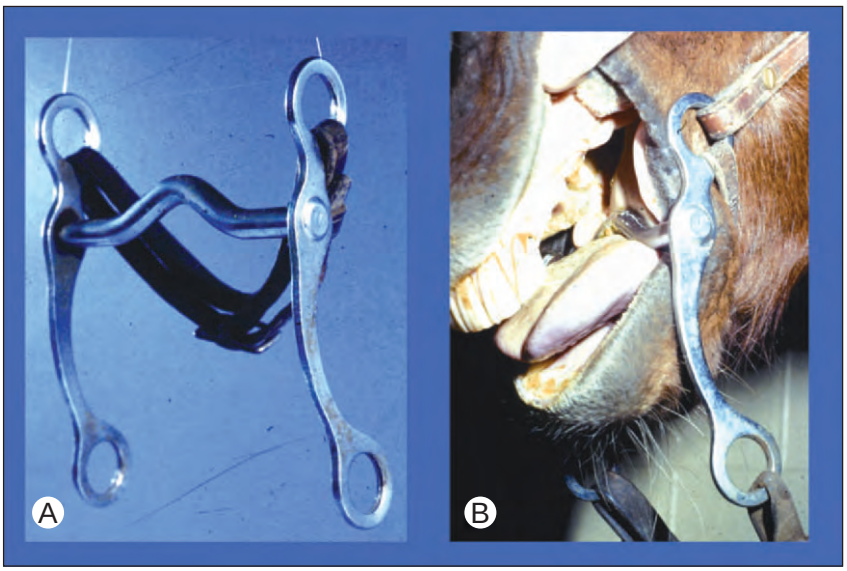


Figure 2.4. (A) Standard curb bit. (B) The lower the port, the greater the chance that the tongue will be damaged by a curb bit.

of the mouthpiece that ordinarily comes into contact with the bars of a horse's mouth. A standard mouthpiece is 3/8 inches in diameter. Most horse show associations prohibit a 1/4-inch (or smaller) mouthpiece because it is considered too severe. Although a 1/2-inch mouthpiece is generally mild, some horses may be uncomfortable carrying so thick a mouthpiece.[6,7] One should always look into a horse's mouth to assure that a mouthpiece fits comfortably.

Mouthpieces are constructed of many different materials and combinations of materials (Figs 2.2, 2.5 and 2.12). In order for a bit to function properly, the horse's mouth must be wet. Copper is frequently incorporated into mouthpieces because it is reputed to promote salivation. Cold-rolled steel, sometimes called 'sweet iron,' is second to copper in stimulating salivation. Sweet iron will rust and, while it may be unattractive, rust seems to taste good to many horses and may further stimulate salivation. Rust-proof stainless steel, however, will also promote salivation to some degree and has the advantages of being hard, staying smooth and cleaning easily. Some bitmakers assert that mouthpieces which combine two different metals are superior for saliva production to mouthpieces made with a single metal. Aluminum, chrome-plated, and rubber- and leather-covered mouthpieces are thought to produce dry mouths.

Of course the metal used in the mouthpiece is not the only factor involved in producing a wet mouth. A dry mouth, usually a result of excessive epinephrine secretion, is a sign of a stressed, unhappy horse. When it comes to generating a wet mouth, the horse's mental state is probably more important than the metal used in the bit. A severe mouthpiece which causes the horse to worry or fret is unlikely to promote a wet mouth regardless of its chemical make-up. Some mouthpieces incorporate rollers, commonly called 'crickets,' or danglers, commonly called 'keys,' to stimulate tongue movement and thus enhance salivation. Such tongue toys also have a pacifying effect on nervous horses.

Some horsemen cover their mouthpieces with latex in the early stages of training or use rubber- or leather-covered mouthpieces on very soft-mouthed horses to protect the bars and tongues.[8] Plastic and synthetic mouthpieces are gradually coming into greater acceptance.[9]

The more complicated the mouthpiece of a bit and the more contact used by the rider, the greater the risk of oral discomfort and/or injuries. Smooth mouthpieces are obviously gentler than those with edges, ridges, teeth or chains.

Snaffle bits (Fig. 2.2)

Regardless of the bit they will ultimately wear, the great majority of today's horses are started in snaffle bits. Snaffle bits are used on 2–5-year-old western performance horses as well as on all classes of English riding for younger horses. Nearly all racehorses, both ridden and driven, spend their entire careers in snaffle bits. A snaffle bit is any bit, whether it has a jointed or solid mouthpiece, in which the cheeks of the bridle and the reins attach to the same or adjacent rings on the bit.[10] There is a direct line of pull from the rider's hands to the horse's mouth with no mechanical advantage. The snaffle's primary contact is with the horse's tongue, bars and lip corners.

Snaffle bits are often identified by the shape of their rings (e.g. O-ring, D-ring, half-cheeked, full-cheeked) and by how their cannons attach to the rings (e.g. loose-ring, fixed ring, egg butt). All ring shapes and attachments have their advantages and disadvantages. A loose ring snaffle, in which O-shaped rings run through holes in the ends of the mouthpiece (Fig. 2.2A), affords the maximum signal. The rings revolve freely and tend to rotate slightly when the reins are picked up but before the bit engages. However, the rotating rings can pinch the corners of a horse's mouth.

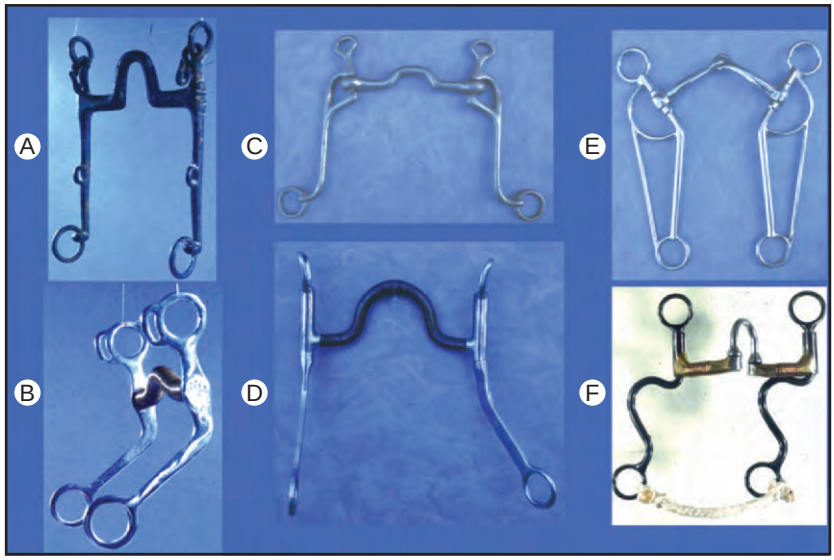


Figure 2.5. Examples of leverage bits. (A) Straight-shanked pleasure horse bit. (B) Grazer bit. (C) Loose cheeks. (D) Myler bit with fixed cheeks and independently rotating shanks. (E) Loose cheeks, broken mouthpiece. (F) Correction bit.

In egg butt and D-ring snaffles (Fig. 2.2B,C) a metal cylinder connects the mouthpiece to the cheek rings and prevents pinching at the corners of the mouth. The well-defined corners of the D-ring snaffle (the straight line of the D) increase the pressure on the horse's cheeks and thus the control over the horse. However, this same pressure increases the chances that the horse's cheeks will be pressed against points on the upper premolars and these fixed-ring bits provide less signal than loose-ringed snaffles.

Some snaffles have prongs or 'cheeks' attached to the rings (Fig. 2.2F,G). 'Full cheek' snaffles have prongs both above and below the mouthpiece, while half-cheek snaffles have prongs below the mouthpiece. Like the D-ring or cylinder type snaffles, the cheeks encourage the horse to turn in the desired direction by increasing the pressure on the corners of the mouth and sides of the face. The cheeks also prevent the bit from being pulled through the mouth.

Leverage bits (Figs 2.4 and 2.5)

Leverage bits, or curb bits provide a mechanical advantage to the rider. There are two sets of bit rings; the upper rings attach to the bridle and the lower rings attach to the reins. The ratio of the length of the shanks of the bit (the portion below the mouthpiece) to the cheeks of the bit determines the amount of leverage. The severity of a bit increases as the ratio increases. For example, in a standard curb bit with 4½-inch shanks and 1½-inch cheeks (a 3:1 ratio), 1 lb of pressure on the reins translates into 3 lb of pressure in the horse's mouth. When using a bit with 8-inch shanks and 2-inch cheeks, 1 lb of pull results in 4 lb of pressure. However, regardless of the ratio, the longer the shanks, the less the force on the reins required to exert a given pressure in the mouth.

Although the severity of a bit increases with the length of the shanks, this severity is partially offset by the fact that the signal provided to the horse increases as well. A long-shanked bit must rotate more than a shorter-shanked bit before it exerts significant pressure in the horse's mouth.

Leverage bits are called curb bits because to exert their leverage they depend upon a curb chain or strap that passes beneath the horse's chin groove and attaches to the rings on the cheeks of the bit. The bit rotates in the horse's mouth until the curb strap stops (curbs) the rotation and the leverage action of the bit takes effect (Fig. 2.6). The leverage bit exerts pressure primarily on the chin groove, the tongue and the bars (Figs 2.4 and 2.7).

The adjustment of the curb strap determines the point at which it snugs up into the chin groove, how quickly and where the bit makes contact with the mouth, and how far the mouthpiece will rotate (Fig. 2.6). The tighter the setting, the less the pull required to activate the bit. The more the bit rotates before the chin strap engages, the more the pressure is transferred to the corners of the lips and to the poll and the less to the tongue, bars and chin groove. Of course, if the bit has a high port or spoon, and the curb strap is loose, the rotation may be halted by contact with the palate, which then must bear part of the pressure.

Typically, the more moving parts within a leverage bit, the more signal it will provide to the horse. For example, a loose-jawed bit, one that attaches to the mouthpiece via hinges or swivels, will provide a certain degree of rotation before the bit engages. Add a loose rein ring to the loose jaw, and the bit will provide even more signal. Install a broken mouthpiece in those shanks and the signal is amplified even more.[4] The downside of a broken mouthpiece in this type of bit is that it increases the potential severity of the bit. In a swivel-ported bit, often called a 'correction' bit, there are joints on each side of the port where it joins the bars (Fig. 2.5F). Such bits are capable of exerting tremendous bar and tongue pressure.

The angle between the shanks and the cheeks affects the speed of communication. The straighter the line, the less

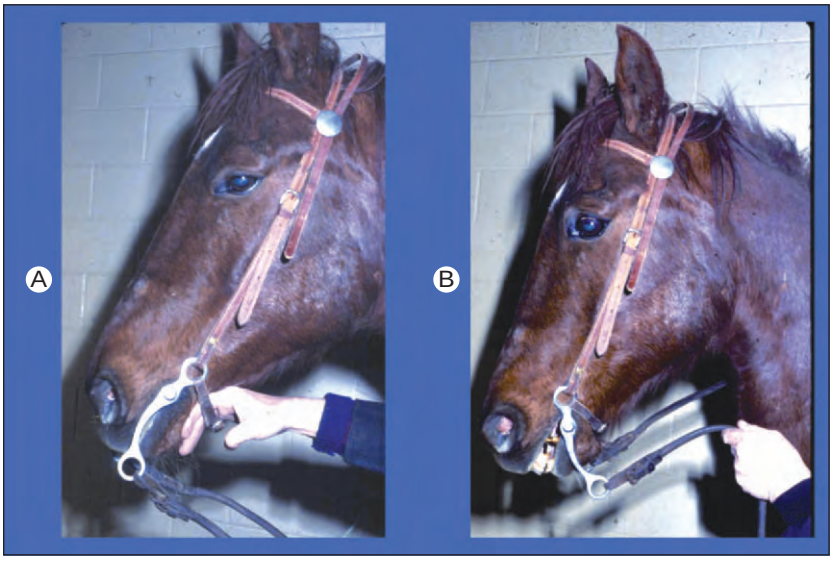


Figure 2.6. (A) A curb strap's adjustment is often based upon the number of fingers that can be slipped under it. (B) A better way is to determine how much rotation of the bit is desired and to set the curb strap accordingly.

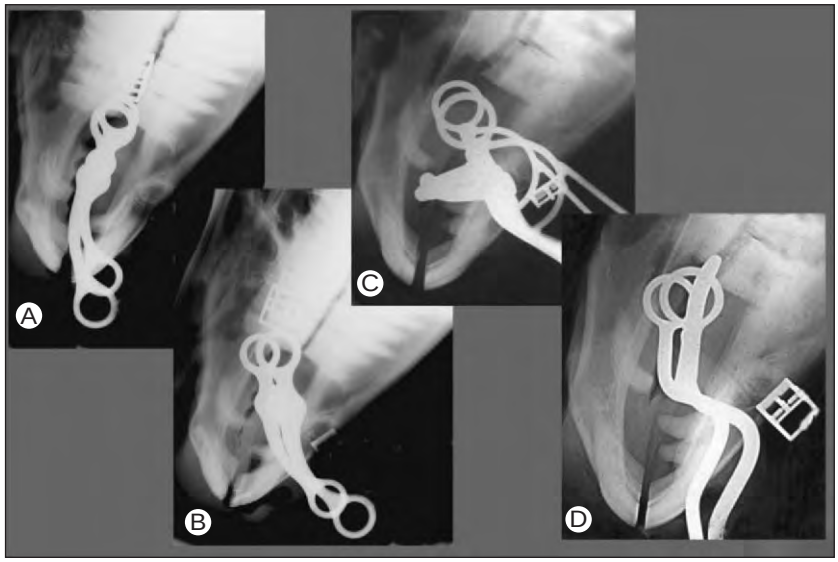


Figure 2.7. Lateral radiographs of curb bits. (A) No rein pressure. (B) Rotation under rein pressure. (C) Rein pressure on a bit with loose cheeks and a broken mouthpiece can force the mouthpiece against the palate. (D) A bit with a high port or spoon can contact the palate and a lateral pull of the reins can force the bit against the cheek teeth.

signal the bit provides. In the so-called grazer bit (Fig. 2.5B) with swept-back shanks, the mouthpiece tends to rotate less than in a bit with straighter shanks (Fig. 2.5A) and provides more signal to the horse. Also, a grazer bit will release its pressure more quickly than a straight-shanked bit when the reins pressure is relaxed. Of course, a tight curb strap will reduce the signal of any leverage bit.

Gag bits (Figs 2.8 and 2.9)

In the basic gag bridle the reins and the cheekpieces of the headstall are one continuous unit. When the reins are pulled, the mouthpiece slides upwards in the horse's mouth and transfers much of the pressure from the tongue and bars to the lips and poll. A gag bit (Fig. 2.8), when used properly, provides a rider more control than a standard snaffle without proportionally providing more punishment to the horse's tongue and bars.

It might be thought that the gag functions to lower the head because tension on the reins places pressure on the poll. But head carriage is more a factor of where the horse finds relief from bit pressure. Since the horse's mouth is much more sensitive to pressure than his poll, if the gag is used with no auxiliary aids, its net effect is to accentuate the basic head-raising action of a snaffle bit. If strong rein pressure is applied to a gag bridle, the bit is pulled relatively far caudally and can severely punish the horse's tongue, lips and cheeks (Fig. 2.9).

Full bridle (Fig. 2.10)

The full bridle, or double bridle (Fig. 2.10), has two sets of cheek pieces and two sets of reins. One set is attached to a

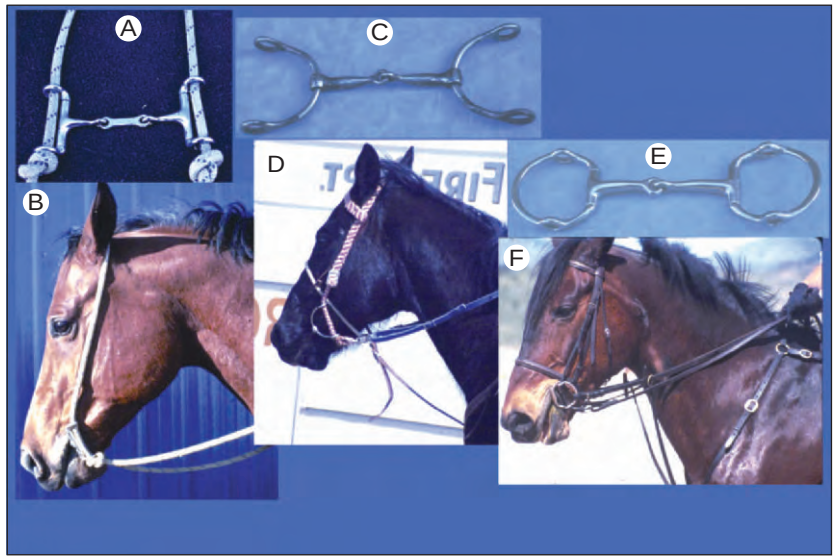


Figure 2.8. Three types of gag bits. (A,B) Basic gag bit with link in mouthpiece. (C,D) Gag snaffle with half-O-rings. (E,F) Gag with full rings for attachment of snaffle rein.

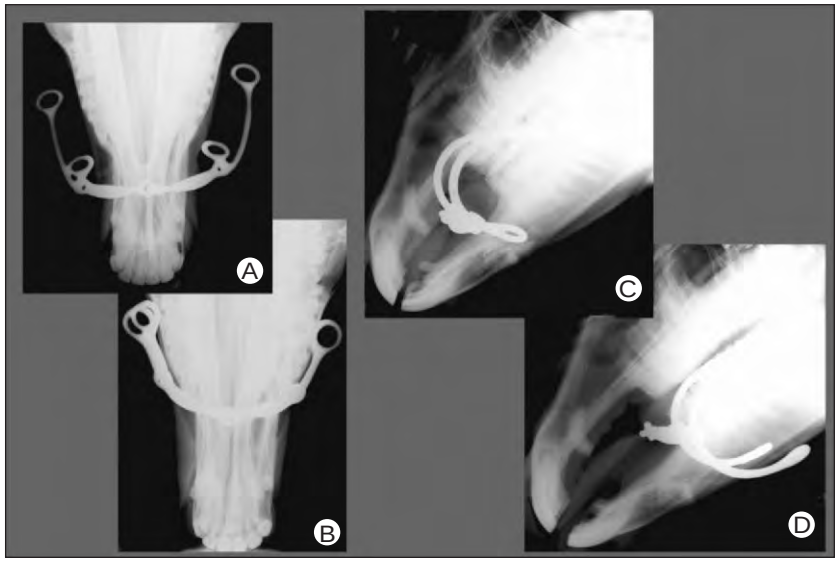


Figure 2.9. Radiographs of gag bits. (A) Ventrodorsal with no rein pressure. (B) Ventrodorsal under rein pressure. (C) Lateral with no rein pressure. (D) Lateral under rein pressure.

curb bit; the other set is attached to a snaffle bit. The snaffle, which is generally relatively small, is called a bridoon or bradoon and is placed above and behind the curb.

The double bridle with its combination of bits, employing a number of forces to achieve its ends, is an extremely sensitive instrument. When used by a skilled rider on a schooled horse, it can place the head with greater finesse than is possible with any other bridle in current use. But the rider needs a considerable amount of skill for this bridle to be effective and humane. It is often stated that with the double bridle the rider uses the snaffle bit to raise the head and turn the horse and the curb bit to lower the head and stop the horse. When the double bridle is used properly, however, nearly all commands for head position, moving and stopping are given via the snaffle. The role of the curb is the basically passive one of promoting poll flexion, collection and balance.[11]

The use of the double bridle when the horse is not sufficiently schooled or the rider is not sufficiently skilled can damage the horse's psyche as well as his mouth. The double bridle puts a lot of hardware in the horse's mouth (Fig. 2.11). The chances of injury are arguably doubled compared with bridles with a single bit. Nearly all the tension should be on the snaffle rein. Excessive tension on the curb rein is the most common cause of problems with full bridles.

Pelhams (Figs 2.12 and 2.13)

A Pelham bit is basically an attempt to gain the advantages of a double bridle with only a single bit in the horse's mouth. The Pelham bit is really just a curb bit with an extra set of rings at the level of the mouthpiece to which an extra set of reins is

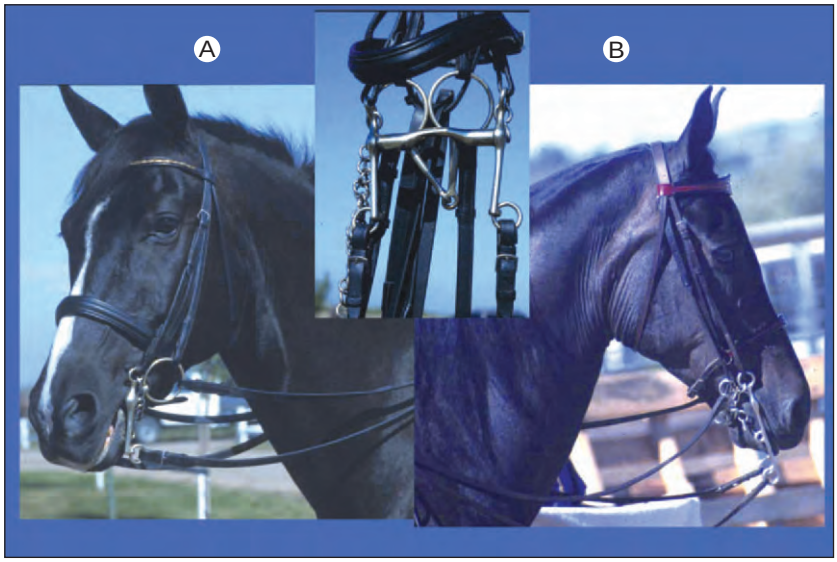


Figure 2.10. (A) Full bridle on dressage horse. (B) Full bridle on English pleasure horse. (Inset) The snaffle and curb bits on a dressage bridle.

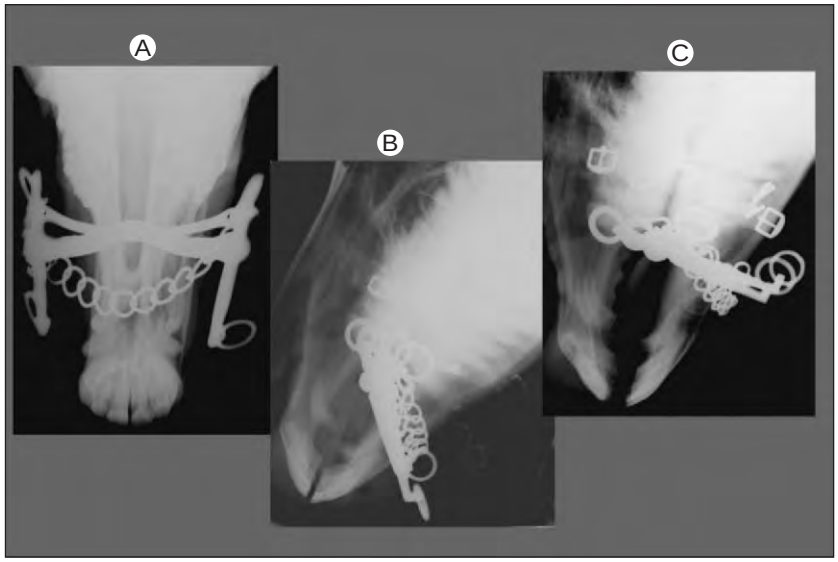


Figure 2.11. Radiographs of bits on full bridles. (A) Ventrodorsal. (B) Lateral without rein pressure. (C) Lateral under rein pressure.

attached. Tension on the lower rein gives the effect of a curb bit and tension on the upper rein gives the effect of a snaffle bit.

Pelham bits come in a wide variety of forms (Fig. 2.12). The mouthpiece may be straight, curved, jointed or ported. The shanks may be long or short, fixed or loose. Some have very short shanks and thick rubber mouthpieces and are very mild. Others have ports and long shanks and are more severe. One type, the Kimberwicke (Figs 2.12C and 2.13B), utilizes only one rein with the hand position, or rein setting, determining whether the bit functions as a snaffle or as a curb.

Critics of Pelhams say that both reins come into play at the same time and confuse a horse. Certainly the Pelham does not work well in a horse with very long narrow jaws or an exceptionally long interdental space. In such a horse it is essentially impossible simultaneously to have the curb chain in the chin groove and the mouthpiece in its proper position against the lip corners. The curb chain, under such circumstances, tends to pull backwards until it is beneath the branches of the mandible, and pressure on these is quite painful to the horse and may result in severe bruising. The use of a lip strap (Figs 2.12D and 2.13C) can help to counteract this disadvantage.

Despite all of the criticisms, some horses perform better in the Pelham bit than in any other. In the horse with short jaws and a relatively small interdental space, the single mouthpiece of the Pelham may fit better than the double mouthpiece of the full bridle.

Driving bits (Fig. 2.14)

In riding horses we have stressed the importance of 'getting off of the horse's mouth.' In other words the rider should cue the horse first with his legs and seat and only secondarily via

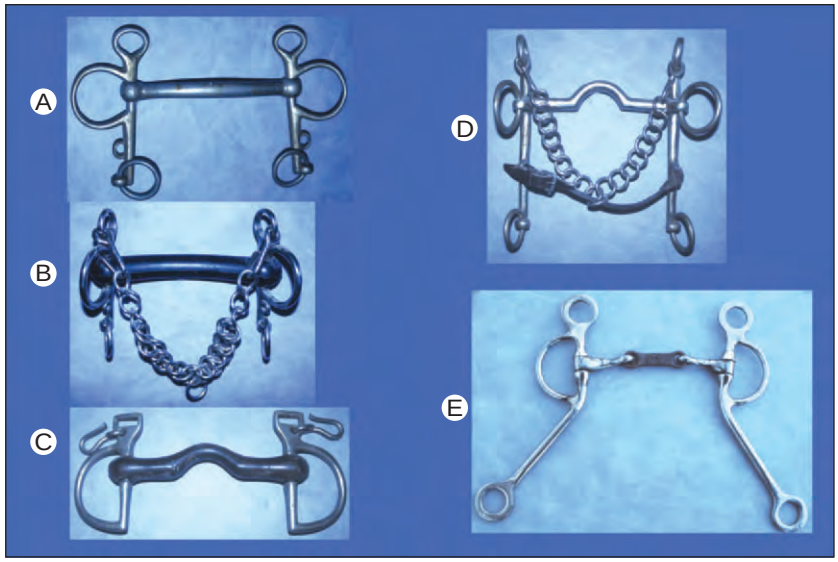


Figure 2.12. Examples of Pelham bits. (A) Mullen mouthpiece with moderate shanks. (B) Rubber-covered mouthpiece with short shanks. (C) Kimberwicke with ported mouthpiece. (D) Long-shanked bit with lip strap. (E) Western Pelham with center link, loose cheeks and long shanks.

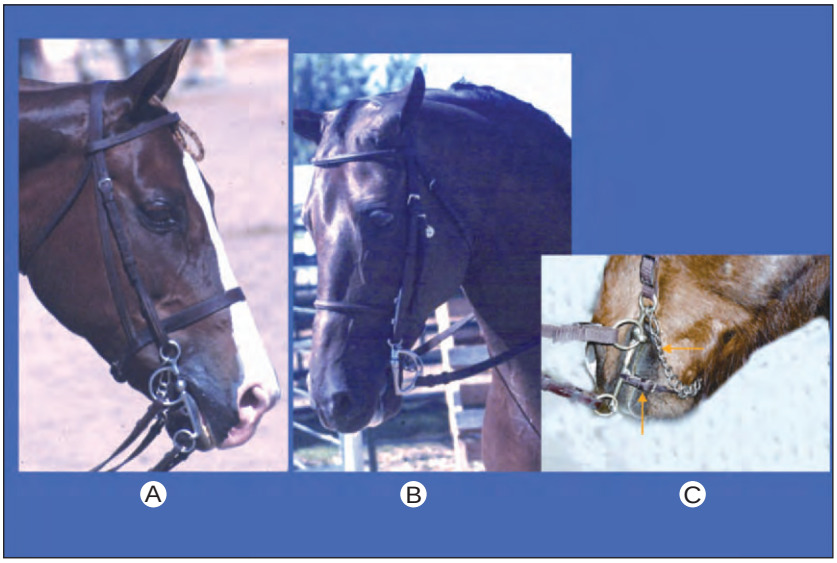


Figure 2.13. (A) Standard Pelham. (B) Kimberwicke with rein set to lower level in Uxeter cheeks. (C) Proper adjustment of curb chain and lip strap (upper arrow points to curb chain, lower arrow points to lip strap).

the bit. But in the driving horse the only direct contact between horse and driver is via the lines and the bit, making this line of communication vitally important. Communication becomes more complicated when horses are driven in teams or multiple hitches.[12]

Driving bits for racing trotters and pacers are essentially always snaffle bits with solid or, more commonly, jointed mouthpieces. Such bits are often used on other types of driving horses as well. Driving snaffles often have half cheeks. The Liverpool, Ashleigh Elbow and Buxton are curb bits commonly used for driving. All three generally have loose cheeks which can be adjusted so that either the corrugated or the smooth side of the straight bar mouthpiece is in contact with the horse's tongue and bars (Fig. 2.14C,D). The reins may be attached to rings at the level of the mouthpiece or to one of two or three slots which are progressively lower in the shanks—the lower the attachment, the more severe the curb action. The shanks of the Liverpool bit are straight, while those of the Elbow bit angle back from the mouthpiece to prevent a horse from seizing them with his lips. The Buxton, with its S-shaped shanks, is a larger and more ornate bit which is used mostly for show.

Overchecks (Figs 2.15–2.17)

In most driving horses an overcheck or check rein is added to the bridle to prevent the horse from lowering his head. The check rein runs from the back pad of the harness up between the horse's ears, passes down the front of the horse's face and divides into two straps which fasten to either side of a separate overcheck bit, which presses upwards in the horse's mouth (Fig. 2.17). (Less commonly the straps attach directly to the driving bit or to a chin strap.)

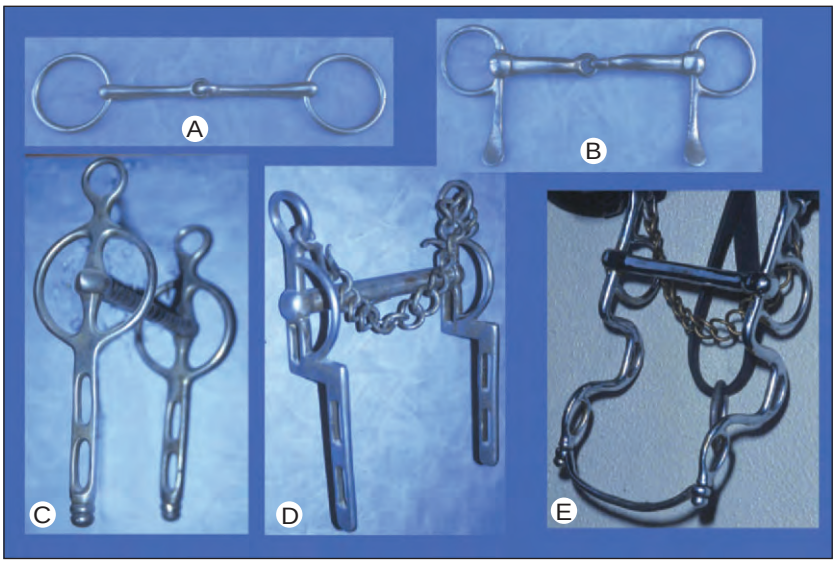


Figure 2.14. Driving bits. (A) O-ring snaffle. (B) Half-cheek snaffle. (C) Liverpool. (D) Ashleigh Elbow. (E) Buxton.

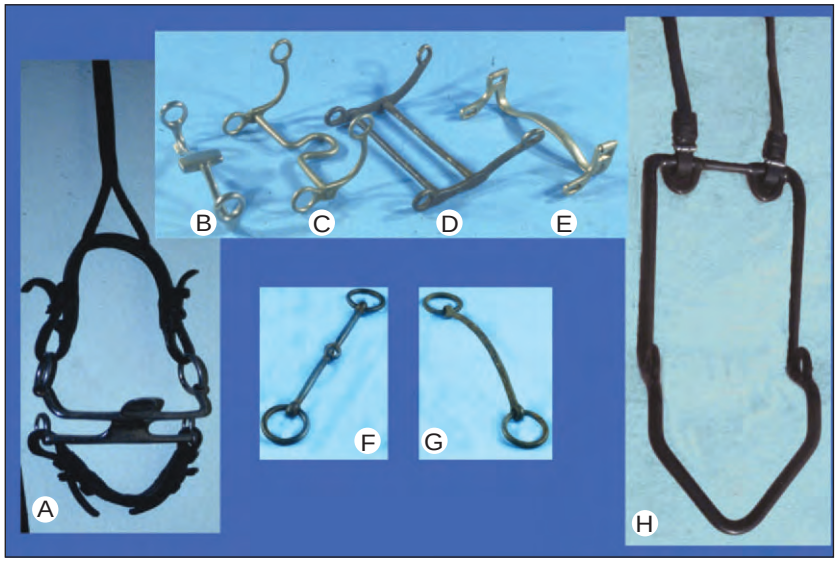


Figure 2.15. Overcheck bits. (A) McKerron complete with nose and chin straps. (B) Burch. (C) Crit Davis. (D) Crabb. (E) Hutton. (F) Plain jointed. (G) Plain solid. (H) O'Mara leverage.

The sidecheck is a variation on the overcheck in which two check reins, rather than joining and running over the top of the horse's head, run through loops on either side of the bridle and back along the sides of his neck to come together at his withers (Fig. 2.16).

Most draft horse bridles are set up with either an overcheck or a sidecheck to prevent the horse from lowering his head to graze or rub and to keep his head in the optimal position for pulling. A check rein is nearly always required for light horses shown in pleasure driving classes or in fine harness classes. Harness racing horses wear overchecks because their heads must be held in an exact position to keep them balanced and on their gait.[8]

The plain overcheck bit is a very small straight bar bit. However, there are many types varying widely in severity (Fig. 2.15). Some racing overchecks like the McKerron (Figs 2.15A and 2.17B), Crit Davis (Figs 2.15C and 2.17C) and Crabb (Fig. 2.15D), listed in increasing order of severity, are used in combination with nose and chin straps to prevent horses from leaning into their check reins.[13] Even more severe is the Burch overcheck (Fig. 2.15B) which is shaped so as to press directly into the hard palate. The cumbersome-appearing, but reasonably humane and effective, Raymond and O'Mara (the so-called leverage overchecks) involve no bit at all (Figs 2.15H and 2.17D). When a horse leans into a leverage overcheck, a strap over his face presses down onto his nose and the U- or V-shaped lower portion of the overcheck lifts up on his chin.[13]

The combination of forces applied by the driving and check reins can place marked stress on a horse's mouth, and one must be aware of the type of overcheck used when caring for a horse's teeth and mouth. For example, the hard palate should be examined carefully for injury in a harness racing horse who performs poorly when checked with a



Figure 2.16. (A) Buxton bit with plain solid overcheck bit attached as sidecheck. (B) Sidecheck attached to O-ring snaffle driving bit.

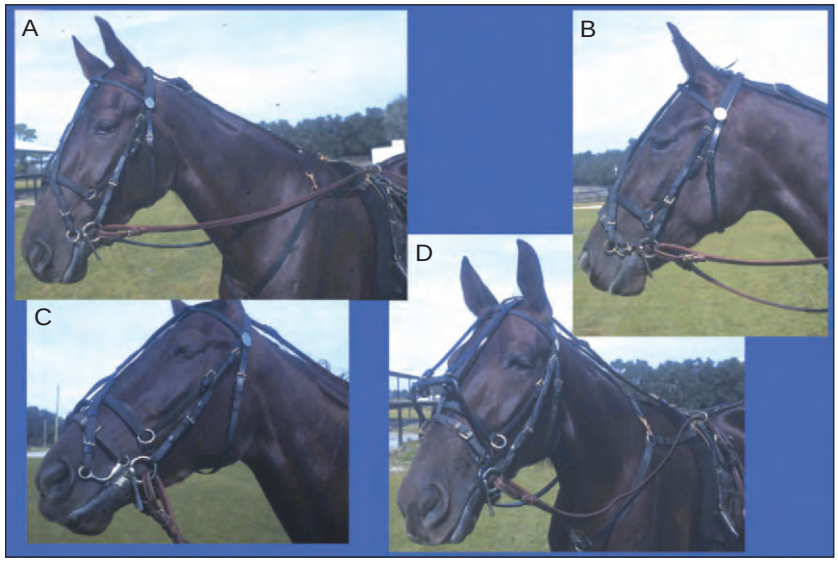


Figure 2.17. Four overcheck systems used on racing Standardbreds. (A) Plain overcheck bit. (B) McKerron overcheck bit. (C) Crit Davis overcheck bit. (D) O'Mara leverage overcheck. All four driving bits are half-cheek snaffles.

Burch, Crit Davis or Crabb bit. If the palate is sore, one should consider recommending a change to a leverage overcheck. Removal of wolf teeth, careful floating and rounding of the upper premolars and removing sharp edges from upper canine teeth are of special importance whenever overchecks are used. The upper canines are placed more caudally than the lower canines, thus providing less space for the overcheck bit than for the driving bit. The overcheck bit may be forced backwards, especially if the horse's head is checked very high, pinching the gums against the teeth. Even leverage overchecks can force a horse's cheeks against upper points or caps.

Fitting the bit

The variation in size, shape and degree of sensitivity of horses' mouths should be considered when selecting and fitting bits and bridles.[4,6] The width of the mouthpiece should accommodate the width of the mouth. If the mouthpiece is too short, it will pinch the corners of the lips against the cheek teeth. Too long, and the bit can shift sideways, sawing on the lips, tongue and bars. An oversized mouthpiece also puts the port or joint out of position and makes the bit ineffective and possibly painful. As a rule, the mouthpiece should not project more than 1/2 inch or less than 1/4 inch beyond the corners of the lips on either side.

The position where the bit fits in the bar space is also important. However, this adjustment will vary from horse to horse and bit to bit. A popular rule-of-thumb for adjusting snaffles has been to adjust the bit so that the commissures of the horse's lips are pulled into one or two wrinkles. The problem with such a fit is that releasing the pressure on the reins gives the horse no relief at the corners of his mouth.[2,4] A better method is to first hang the bit relatively loosely until the horse learns to pick it up and carry it and then adjust the headstall to position the bit where the horse has determined it is most comfortable (Fig. 2.18).

A horse with a short or shallow mouth (from lips to corners) will carry the bit forward in his mouth where his tongue rides highest. A horse with a deep mouth will hold the bit farther back in his mouth where his tongue sits lower in his jaw space and his palate is more concave. Consequently, there is less space between the tongue and hard palate in the shallow-mouthed horse and, everything else being equal, he requires a bit with a thinner mouthpiece and a port providing more tongue relief than the bit required by the deeper-mouthed horse. Some horses, especially Thoroughbred types, have relatively narrow, sharp bars which are easily damaged by pressure.[14] Such horses require thicker and/or softer mouthpieces than do horses with thicker bars.

An older horse may have less space for a bit in his mouth. As a horse ages, his incisors slope further forward, while the cheek teeth wear down, causing the palate to sink closer to the tongue. A bit that was comfortable for a horse when he was five may no longer be comfortable when he is twenty.

One must consider more than the external dimensions of a horse's head and his age in choosing an appropriate bit. Recent research has shown that the size and shape of a horse's oral cavity often correlate poorly with the size and shape of his head, his age or his sex.[6] In selecting and properly fitting a bit there is no substitute for careful manual and digital examination of a horse's mouth. Periodic reexaminations are indicated because wearing of the teeth, or even dentistry, can change the shape of the oral cavity.[6]

Bitless bridles

When choosing bitless headgear, horse owners should consider the same factors that they would when choosing

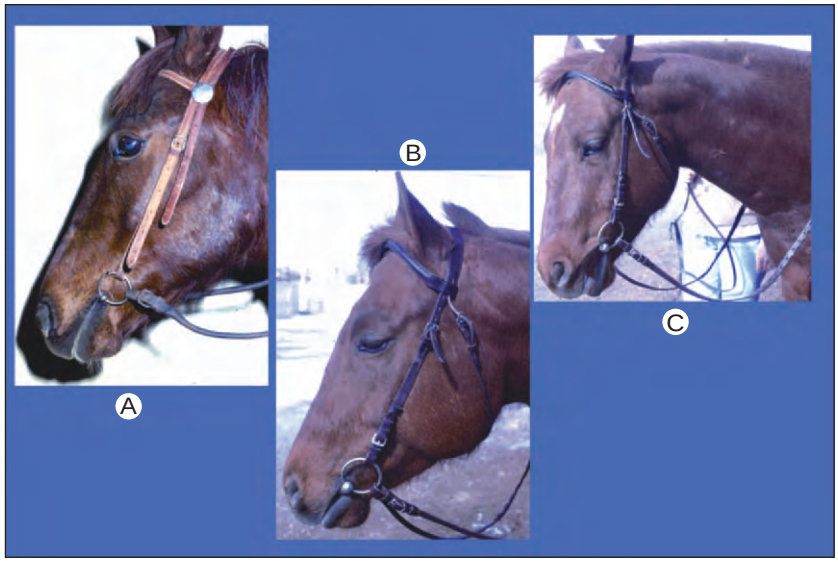


Figure 2.18. (A) Bridles are often adjusted so that bit causes a wrinkle at the commissures of the lips. (B) Bridle adjusted so that bit hangs too low. (C) Bridle adjusted too tight.

any other bridle. Otherwise, they risk dulling the horse's sensitivity and responsiveness to rein signals.[4]

Traditional hackamore (Fig. 2.19A)

The hackamore provides a means of promoting poll flexion, collection and balance along with optimal stopping power and directional control while staying out of the horse's mouth. It is used with a light bumping action, initiated by gently tugging on one rein at a time. Alternating pulls and releases can be used to ask the horse to flex at the poll and stop.[15]

The heart of the hackamore is the bosal, a braided rawhide or leather noseband which is fashioned around a rawhide core. Bosals vary greatly in diameter, with the appropriate size depending upon the horse's sensitivity and stage of training. Generally one moves from thicker, heavier bosals to thinner, lighter ones as the hackamore horse progresses.

The bosal should rest on the bridge of the nose, or just slightly above, where it is supported by the nasal bone. If it is placed too low it will exert excessive pressure on the horse's nasal cartilages and interfere with his breathing.

Obviously a hackamore will not damage a horse's tongue and bars, but the bosal contacts some very sensitive points on his face. Rein pressure presses the bosal into the top of the face and into contact with the cheeks and lower jaw all at the same time. Heavy hands on the reins or an ill-fitting bosal can abrade the horse's nose and jaw and press his cheeks against the upper premolars.

Mechanical hackamore (Fig. 2.19B)

While mechanical hackamores are indeed bitless bridles, they function more like curb bits than like true hackamores.[4] Mechanical hackamores have metal shanks that attach to a

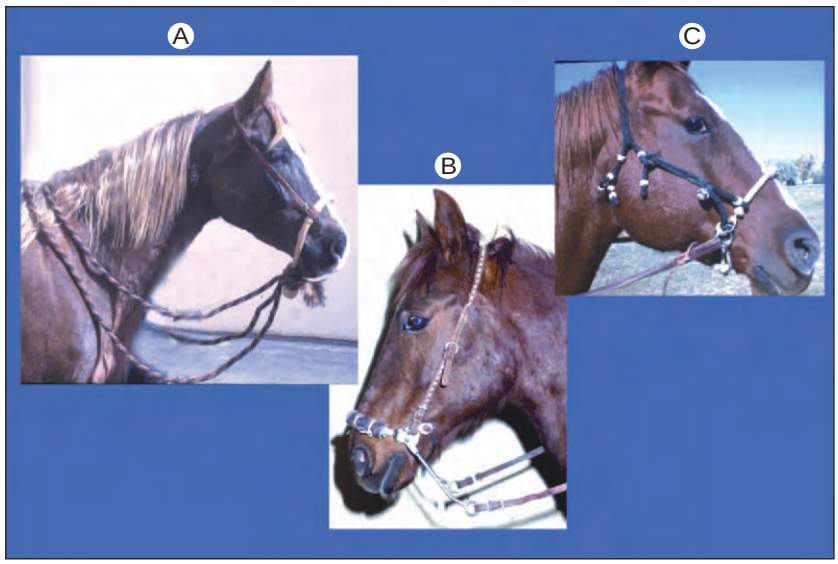


Figure 2.19. Bitless bridles. (A) Traditional bosal hackamore. (B) A severe mechanical hackamore. (C) Side pull.

noseband and curb chain. While there is no mouthpiece, the shanks amplify force to the nose, chin and poll in the same way that a leverage bit works on the mouth, chin and poll. Because of the wide variety of mechanical hackamores, it is possible to vary the severity as required. Some horses who do not respond well to a bit perform well in mechanical bitless bridles.

Other bitless bridles

The side pull (Fig. 2.19C) applies pressure only on the sides of the horse's nose. The Bitless Bridle 2000 ®(Bitless, Bridle, Inc., York, PA) distributes pressure across the poll, behind the ears, down the side of the face, behind the chin and across the nose. These are gentle bridles which minimize the stress on a horse's mouth and work exceptionally well on some horses.

Accessories

We must be familiar with the functions of nosebands and martingales in caring for horses' mouths because these accessories alter the function, or the direction, of pull on the bit.

Nosebands

The simplest noseband, the cavesson, functions merely to stabilize the bridle (Fig. 2.10A,B) or as a point of attachment for a martingale (Fig. 2.21A). Other types of nosebands are used to aid or modify the action of the bit.

Drop, flash and figure-of-eight nosebands (Figs 2.20A–C) are used to hold the bit in the proper position and to keep horses from gaping their mouths. The top of the drop noseband is fitted just at the lower end of the nasal bones, while the lower portion passes below the bit and lies in the chin groove. A drop noseband is fairly restrictive and can cause problems if not properly adjusted.[16] If it is too long on top and too short below, it will hang too close to the nostrils, interfering with breathing, and the bottom will press the bit into the corners of the lips and hold the mouth too tightly closed.

The flash noseband attaches to the center of a simple cavesson above the nose. The lower end passes below the bit and lies in the chin groove. The figure-of-eight, or gackle, noseband has a top strap which fastens above the bit and a lower strap which fastens under the bit and lies in the chin groove. The two straps intersect in the middle of the face at about the level where a cavesson would be located. Both the flash and the figure-of-eight nosebands have actions similar to the drop noseband but are less severe and are not as likely to interfere with breathing.

The so-called 'cheeker' (Fig. 2.20D) is not really a noseband but rather is a rubber strap that runs from the crownpiece of the bridle down the middle of the horse's face where it separates to attach on either side of a snaffle bit. Like the drop, flash and figure-of-eight nosebands, the cheeker holds the bit up in the horse's mouth.

Sheepskin-covered cavessons, or shadow rolls (Fig. 2.20E), are used to prevent a horse from seeing the ground in front of him, and thus to prevent his shying at shadows or other potentially frightening sights. Cheekers and shadow rolls are used mainly on racehorses.

Martingales

There are two basic kinds of martingales: standing (known in western circles as tie-downs) and running (Fig. 2.21). Both types of martingales promote balance and the proper action of a bit by discouraging, or physically preventing, the horse from raising his head too high or extending his nose too far. Both types begin with a strap running from the saddle girth up the front of the horse's chest. The standing martingale, which exerts its pressure on the horse's nose, continues as a single strap that attaches to the bottom of a

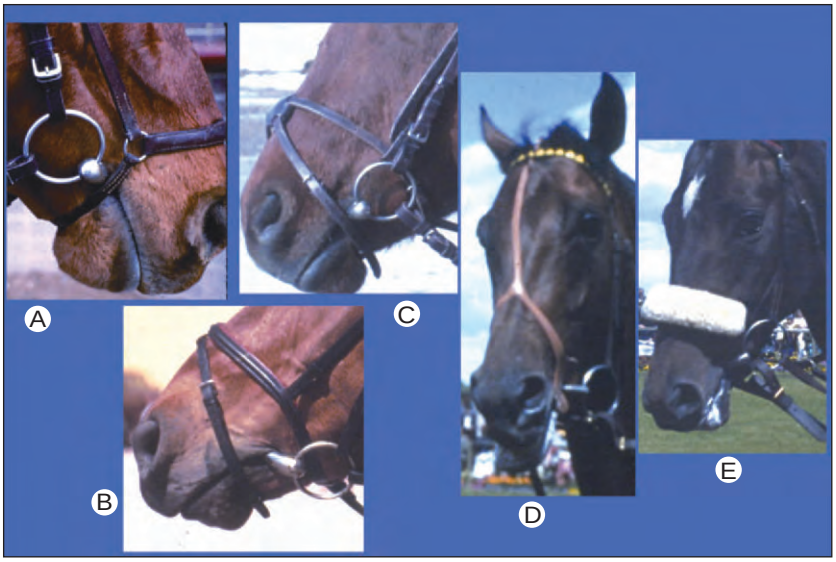


Figure 2.20. Nose bands. (A) Drop. (B) Flash. (C) Figure-of-eight. (D) Cheeker. (E) Shadow roll.

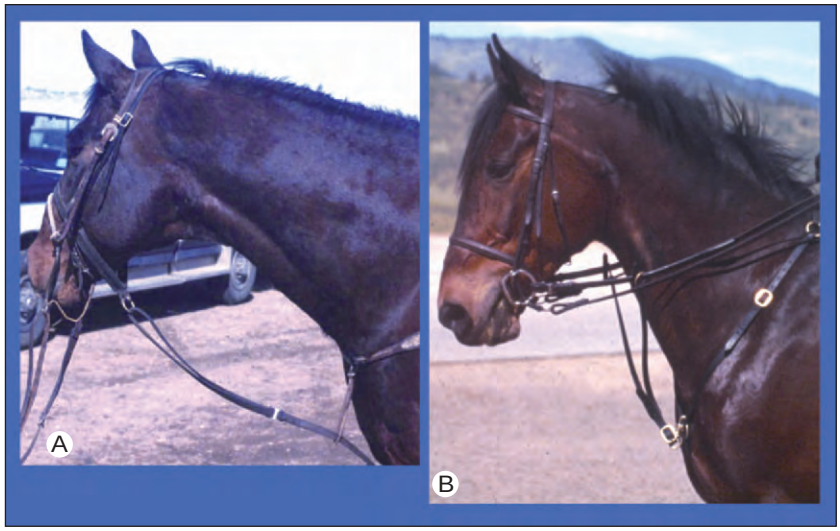


Figure 2.21. (A) Standing martingale. (B) Running martingale.

noseband. The running martingale, which exerts its pressure on the bit, forks into two straps with rings at their upper ends through which the reins run. A martingale should not be adjusted so tightly as to pull the horse's head down into an unnatural or uncomfortable position. The martingale should become active only when the horse raises his head, thus preventing him from evading the bit and becoming unbalanced.

Conclusion

The knowledge of anatomy, physiology, pharmacology and nutrition, even when coupled with high levels of diagnostic, mechanical and surgical skills and the possession of the best equipment available is not always sufficient to provide optimal dental care to horses. Recognizing mouth problems and properly preparing the teeth depends upon much more. One must consider the age, performance discipline, ability and level of competition of the horse, not to mention the level of skill and the experience of his rider or driver. The more the veterinarian knows about bits, bridles and accessories as they relate to the above factors, the better he can fulfill the needs of his clients and the more rewarding his dentistry practice will be.

REFERENCES

1. Scoggins RD (2001) Bits, bitting and dentistry. In: *Proceedings of the 47th Annual Meeting of the American Association of Equine Practitioners*, 47, pp. 138–141.
2. Bennett DG (2001) Bits and bitting: form and function. In: *Proceedings of the 47th Annual Meeting of the American Association of Equine Practitioners,* 47, pp. 130–137.
3. Young JR (1982) *The Schooling of the Horse.* University of Oklahoma Press, Norman, OK, pp. 235–263.
4. Lynch B and Bennett DG (2000) *Bits and Bridles: Power Tools for Thinking Riders.* EquiMedia, Austin, TX.
5. Johnson TJ (2002) Surgical removal of mandibular periostitis (bone spurs) caused by bit damage. In: *Proceedings of the 48th Annual Meeting of the American Association of Equine Practitioners,* 48, pp. 458–462.
6. Engelke E and Gasse H (2003) An anatomical study of the rostral part of the equine oral cavity with respect to position and size of a snaffle bit. *Equine Veterinary Education,* 15, 158–163.
7. Clayton HM and Lee R (1984) A fluoroscopic study of the position and action of the jointed snaffle bit in the horse's mouth. *Journal of Equine Veterinary Science,* 4, 193–196.
8. Riegle G (1996) Training the pacer. In: *The New Care and Training of the Trotter and Pacer,* ed. C Greene, US Trotting Association, Columbus, OH, p. 337.
9. McBane S (1992) *The Illustrated Guide to Horse Tack.* David and Charles, Devon, UK, pp. 49–91.
10. Malm GA (1996) *Bits and Bridles: an Encyclopedia.* Grasshopper, Valley Falls, KS.
11. Crossley A (1988) The double bridle. In: *The Horse and the Bit*, ed. S McBane, Howell, New York, pp. 60–78.
12. Telleen M (1977) *The Draft Horse Primer*. Rodale Press, Emmaus, PA, p. 256.
13. Haughton T (1996) Choosing the right equipment. In: *The New Care and Training of the Trotter and Pacer*, ed. C Greene, US Trotting Association, Columbus, OH, pp. 184–214.
14. Edwards EH (2000) *The Complete Book of Bits and Bitting.* David and Charles, Devon, UK.
15. Connell E (1952) *Hackamore Reinsman.* Lennoche Publishers, Katy, TX.
16. Loriston-Clarke J (1987) *The Complete Guide to Dressage.* Courage Books, Philadelphia, pp. 130–131.

3 Dental Anatomy

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Introduction

Equine dental nomenclature

Adult mammals have four types of teeth, termed incisors, canines, premolars (PM) and molars (M), in a rostrocaudal order.[1] Teeth embedded in the incisive (premaxilla) bone are by definition termed incisors. The most rostral tooth in the maxillary bone is the canine. In horses, the main three premolars have become more complex and morphologically identical to the molars (i.e. molarization of premolars) to facilitate grinding of foodstuffs. Consequently in horses, premolars 2–4 (Triadan 06–08) and the three molars (Triadan 09–11) can be collectively termed cheek teeth. Each type of tooth has certain morphological characteristics and specific functions. Incisor teeth are specialized for the prehension and cutting of food and the canine teeth are for defence and offence (for capture of prey in carnivores). Equine cheek teeth function as grinders for mastication. The occlusal or masticatory surface is the area of tooth in contact with the opposing teeth; the term coronal refers to the crown. The anatomical crown is that part of the tooth covered by enamel and in brachydont (short-crowned) teeth such as in humans, is usually the same as the clinical (erupted) crown, i.e. the erupted aspect of the tooth. However, in equine teeth (hypsodont – long-crowned), especially young teeth, most of the crown is termed unerupted or reserve crown with a smaller proportion (approximately 10–15 per cent in young adult horses) of clinical crown. The term occlusal ('coronal' is less satisfactory for hypsodont teeth) is used when referring to direction toward the occlusal surface. More recently it has been proposed that the reserve crown be divided into alveolar crown (i.e. that part lying in the alveolus) and the gingival crown, i.e. that part which has erupted from the alveolus, but which is still lying subgingivally.[2]

Apical refers to the area of tooth farthest away from the occlusal surface, i.e. the area where the roots later develop and is the opposite of occlusal. Lingual refers to the medial aspect (area closest to the tongue) of all the lower teeth, while palatal refers to the same aspect of the upper cheek teeth. Buccal (aspect closest to cheeks) refers to the lateral aspect of both upper and lower (cheek) teeth, while labial refers to the rostral and rostrolateral aspect of teeth (incisors and canines only in horses) close to lips. The terms interproximal or interdental refer to the area of teeth that face the adjoining teeth (in the same arcade or row). The terms mesial and distal, which refer respectively, to the surfaces of teeth that face toward and away from an imaginary line between the central incisors are satisfactory for equine incisors that form a true arch. However, these terms are unsatisfactory for the equine cheek teeth, because they do not form part of a continuous dental arch as they are separated from the incisors by the inderdental space ('bars of mouth'). The term cheek teeth row is a more appropriate term to describe the rows of six cheek teeth.

Equine dental evolution

The evolution of equine dentition is comprehensively covered in Chapter 1, but some salient anatomical aspects will be discussed here. Following ingestion of their coarse forage diet, the necessary grinding down of this foodstuff to a small particle size (the average length of fibers in equine feces is just 3.7 mm)[3] to allow more efficient endogenous and microbial digestion, causes a high degree of wear on their cheek teeth. However, unlike ruminants, which can later regurgitate their food for further chewing, horses have only one opportunity to effectively grind their foodstuffs.

Brachydont teeth (permanent dentition) fully erupt prior to maturity and are normally long and hard enough to survive for the life of the individual because they are not subjected to the prolonged and high levels of dietary abrasive forces that herbivore teeth must contend with. In contrast, hypsodont teeth erupt over most of a horse's life at a rate of 2–3 mm/year,[4,5] which is similar to the rate of attrition (wear) on the occlusal surface of the tooth, provided that the horse is on a grass diet (or some alternative fibrous diet, e.g. hay or silage) rather than being fed high levels of concentrate food. The latter type of diet will reduce the rate of occlusal wear and also restrict the range of lateral chewing actions;[6] however, the teeth will continue to erupt at the normal rate and thus dental overgrowths can occur. Both brachydont and hypsodont teeth have a limited growth period (although somewhat prolonged in the latter) and thus are termed anelodont teeth. A further progression of the evolutionary development for coping with highly abrasive diets (e.g. in some rodents such as rabbits) is the presence of teeth that continually grow throughout all of the animal's life, termed elodont teeth.

Brachydont teeth have a distinct neck between the crown and root, a feature that could not be present in permanent hypsodont teeth, which have a prolonged eruption period. At eruption, hypsodont teeth have no true roots and in this text the term root specifically refers to the apical area which is enamel free.[7,8] The delayed formation of roots in equine teeth permits further dental growth after these teeth erupt in addition to the very prolonged eruption of these teeth for most of the horse's life. The terms apical or periapical are much more appropriate to describe this area of equine teeth that for example, commonly develop apical infections of the mandibular 07s and 08s (second and third cheek teeth) even prior to the development of any roots. About 25 per cent of equine mandibular cheek teeth still have no root development even 12 months following eruption.[9]

Because of the marked wear on the surface of hypsodont teeth, exposure on the occlusal surface of enamel, and also of dentin and cement (cementum) is inevitable and leads to the presence of alternate layers of these three calcified dental tissues on the occlusal surface. This is in contrast to the sole presence of enamel on the occlusal surface of brachydont teeth. The presence of infolding of the peripheral enamel, and also of enamel cups (infundibula) in the upper cheek teeth and all incisors also increases the amount and irregularity of exposed enamel ridges on the occlusal surface. This feature confers additional advantages to hypsodont teeth, as the different calcified tissues wear at different rates (enamel slowest, dentin and cementum fastest) and therefore a permanently irregular occlusal surface that is advantageous in the grinding of coarse fibrous foodstuffs is created by a self-sharpening mechanism.

Embryology of teeth

Dental development (dentogenesis) involves several sequential processes, including epithelial–mesenchymal interaction, growth, remodeling and calcification of tissues until a tooth is fully developed.[10–12] During dental development, the tooth germ undergoes a series of distinct, consecutive events termed the initiating, morphogenetic and cytodifferentiative phases. These phases occur in all types of mammalian dentition;[13] however, their timing and termination vary, i.e. compared with brachydont teeth, hypsodont teeth have a delayed termination of the morphogenetic and cytodifferentiative stages (at the apical region), while in elodont teeth, these stages continue throughout all of the animal's life.

Tooth formation begins by the development of a horseshoe-shaped, epithelial thickening along the lateral margin of the fetal oral cavity. This epithelial thickening (termed the primary epithelial band) invaginates into the underlying mesenchymal tissue to form two distinct ridges: the vestibular lamina and, caudal to it, the dental lamina. The dental lamina produces a series of epithelial swellings called tooth buds along its buccal margin. This stage is known as the bud stage of tooth development (Fig. 3.1). At this stage, a mesenchymal cell proliferation develops beneath the hollow ectodermal tooth bud and invaginates into the tooth bud, which now develops into an inverted cap-shaped structure called the enamel organ. This is called the cap stage of dental development (Fig. 3.1).

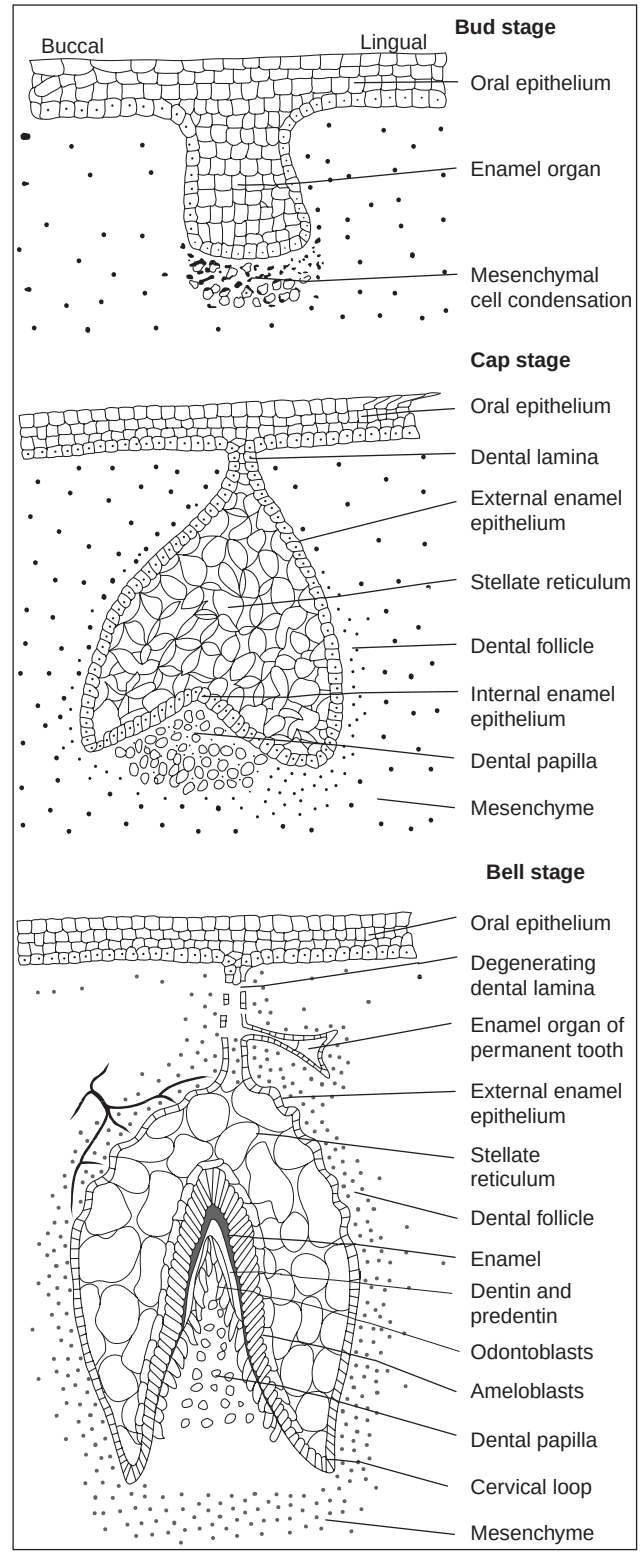


Figure 3.1. Three early stages of development of a brachydont or hypsodont tooth. (From Kilic,[53] with permission.)

All deciduous teeth and the permanent molars develop from the enamel organ of the dental laminae. However, permanent incisors, canines and permanent premolars are formed from separate enamel organs that are derived from lingual (medial) extensions of the dental laminae of the deciduous teeth (Fig. 3.1). Consequently, the deciduous incisors are normally displaced labially (toward the lips) by the erupting permanent incisors.

After formation of the enamel organ, the mesenchymal cells continue to proliferate within the concave aspect of the enamel organ and are now termed the dental papilla, which is later responsible for dentin and pulp formation. These cells now also extend peripherally, as a structure termed the dental sac (follicle), which surrounds and protects the enamel organ and dental papilla until tooth eruption (Fig. 3.2).[1,14] The enamel organ, dental papilla and dental sac are together

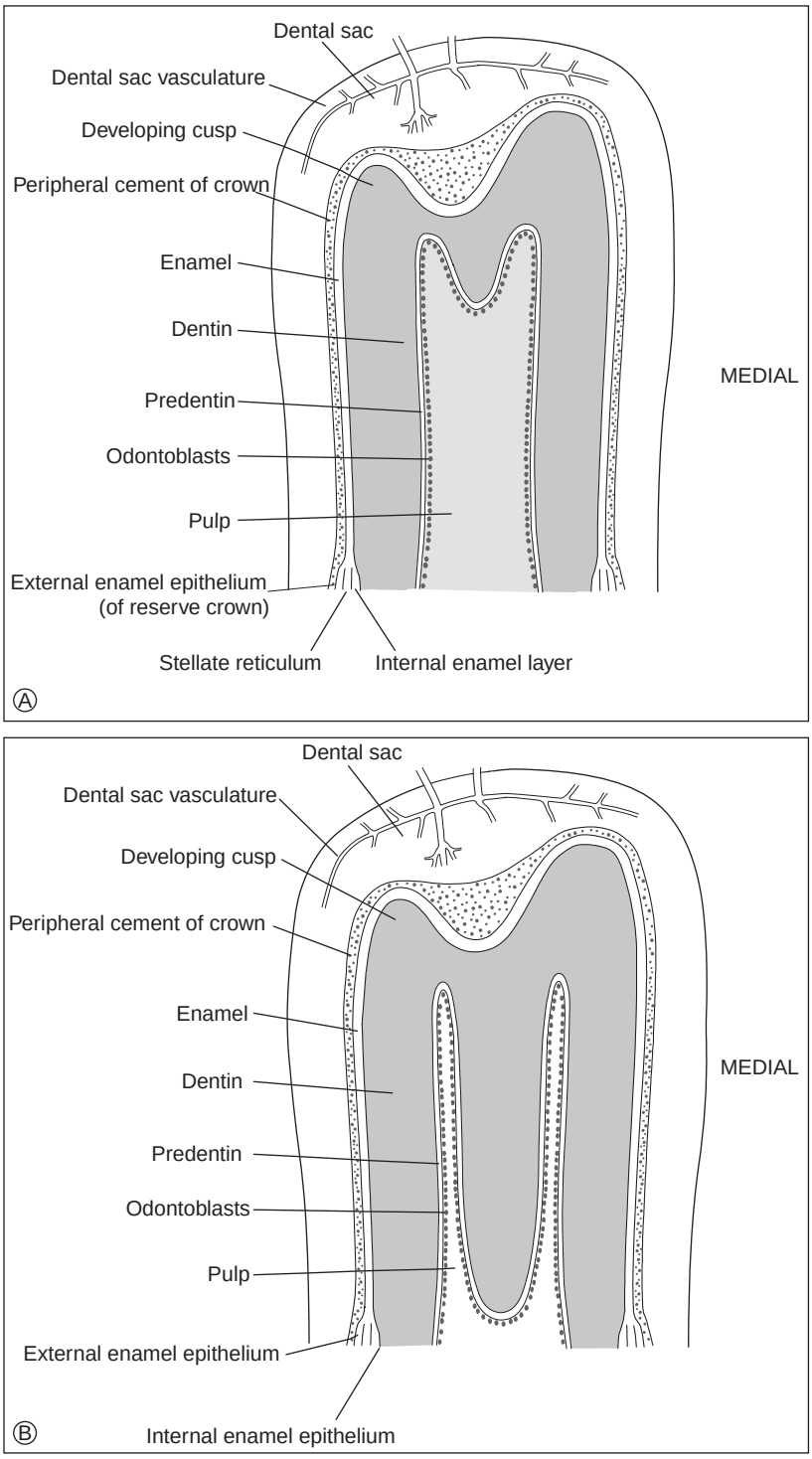


Figure 3.2. Two stages of the development of a multicusped hypsodont tooth without an infundibulum (i.e. a lower cheek tooth), showing the presence of coronal cement and enamel that are covered by the dental sac. The large common pulp chamber (A) later develops two horns (B) due to deposition of dentin by the odontoblasts within the pulp chamber.

termed the tooth germ, with each germ responsible for an individual tooth.

The enamel organ proliferates further and, in brachydont dentition, assumes a bell shape, which is termed the bell stage of dental development. At this stage, the concavity of the enamel organ increases, while the mesenchymal cells of the dental papilla invaginate further into its hollow aspect (Fig. 3.1). Additionally, in some equine teeth, invaginations of enamel epithelium, which will later become infundibula, develop from the convex aspect of the 'bell' into the papilla (one per incisor and two per upper cheek teeth). Equine cheek teeth have multiple cusps (raised occlusal areas) that arise from protrusions on the convex aspect of the bell. The enamel organ in equine incisors and in all brachydont teeth is circular on transverse section; however, the enamel organ of equine cheek teeth develop infoldings[15] that later produce the infolded peripheral enamel.

Most cytodifferentiative events in the tooth germs occur during the transitional period between the cap and bell stages. The cells lining the concave aspect of the enamel organ become the internal enamel epithelium and the cells lining the convex aspect of the enamel organ form the external enamel epithelium.[11] Between them lies a third layer containing star-shaped cells with large intracellular spaces, termed the stellate reticulum (Fig. 3.1), which has nutritive and mechanical functions in enamel development. The cells of the internal dental epithelium develop into tall columnar cells with large, proximally located nuclei. This induces alterations at the molecular level in the underlying dental papilla, whose uppermost cells now rapidly enlarge, becoming odontoblasts. The first dentin layer is now laid down along the basal membrane, which then disintegrates. These changes reciprocally induce the overlying internal enamel epithelial cells to differentiate into ameloblasts, which now begin to produce enamel.[16]

After they initially deposit a structureless enamel layer, the ameloblasts migrate away from the dentinal surface and form a projection termed "Tomes' process" at their distal surface. Secretions from the proximal aspect of Tomes' process form interprismatic enamel and secretions from the surface of Tomes' process form the enamel prisms. The development of enamel and dentin (and later, also of cement) occurs in two consecutive phases, the secretion of extracellular matrix of mucopolysaccharides and organic fibers, which is followed by its mineralization.[17,18]

Odontoblasts, like ameloblasts and cementoblasts (that produce cement) are end cells, meaning that they cannot further differentiate into other cell types. During dentin deposition, the basal aspects of odontoblasts gradually become thinner and form long fine cytoplasmic extensions termed odontoblast processes. They remain in the dental tubule while the odontoblast cell body remains at the surface of the developing dentin (predentin) at the periphery of the pulp cavity.[12]

In multicusped teeth (such as equine cheek teeth) mineralization starts independently at each cusp tip (Figs 3.2–3.4) and then merges, as calcification progresses down toward the amelodentinal (enamel to dentin) junction.[1] As dentin and enamel deposition continues, odontoblasts and ameloblasts move in opposite directions and thus avoid becoming entrapped in their own secretions. Radiography has shown the calcification of equine deciduous cheek teeth buds (three in each quadrant) to be underway by the 120th day of fetal life and to be completed by 240 days.[19] The deciduous 06 (PM2) germs are largest, indicating that they develop first. Calcification of the first permanent tooth bud (09s) begins about 6 months later.[19]

In brachydont teeth, vascularization begins at the periphery of the tooth germs at the early cap stage and blood vessels grow into the dental sac and dental papilla.[12] Until this stage, the enamel epithelium is supplied by small mesenchymal capillaries. Once dentinal and enamel mineralization begins, the connection between the enamel epithelium and the dental papilla is completely lost. The developing enamel is now solely nourished by the vasculature of the surrounding dental sac (Fig. 3.3).

After crown formation is completed in brachydont teeth, the external and internal enamel epithelial cells at the cervical region proliferate down over the dental papilla as a double layer of cells, which (at this site) are termed Hertwig's epithelial root sheath (Fig. 3.2). This epithelium induces the underlying mesenchymal cells to differentiate into odontoblasts, which produce dentin.[12] With the progressive distal disintegration of Hertwig's epithelial root sheath, the dental sac cells come into direct contact with dentin. Interaction between these two tissues now induces cells of the dental sac to convert into cement-forming cells, i.e. cementoblasts, and then to lay down cement.[10,14] In equine teeth this cement deposition occurs over the entire crown, latterly over the future occlusal surface, just prior to eruption[15] (Fig. 3.3). When the equine tooth has reached its full length, the epithelial root sheath disintegrates and no further enamel can be formed.

In the infundibula (two in upper cheek teeth and one in all incisors), cement deposition proceeds by cementoblasts that are believed to be solely nourished by vasculature of the dental sac (Fig. 3.3) because, as previously noted, the underlying infundibular enamel is believed to fully isolate it from the pulp cavity vasculature. Immediately after eruption, the soft tissue of the dental sac is quickly destroyed and consequently infundibular cement no longer has any blood supply (Fig. 3.3) and can now be regarded as an inert or 'dead' tissue. However, there has been some evidence that the apical aspect of some cheek teeth infundibula may contain an opening for a period after dental eruption. This could allow some nourishment of infundibular cement from the pulp, rather than solely from the dental sac via the occlusal surface. Because of the frequent absence of complete cement filling of cheek teeth infundibula, the term 'central infundibular cement hypoplasia' has been advocated for this feature, as discussed further in the cementum section.

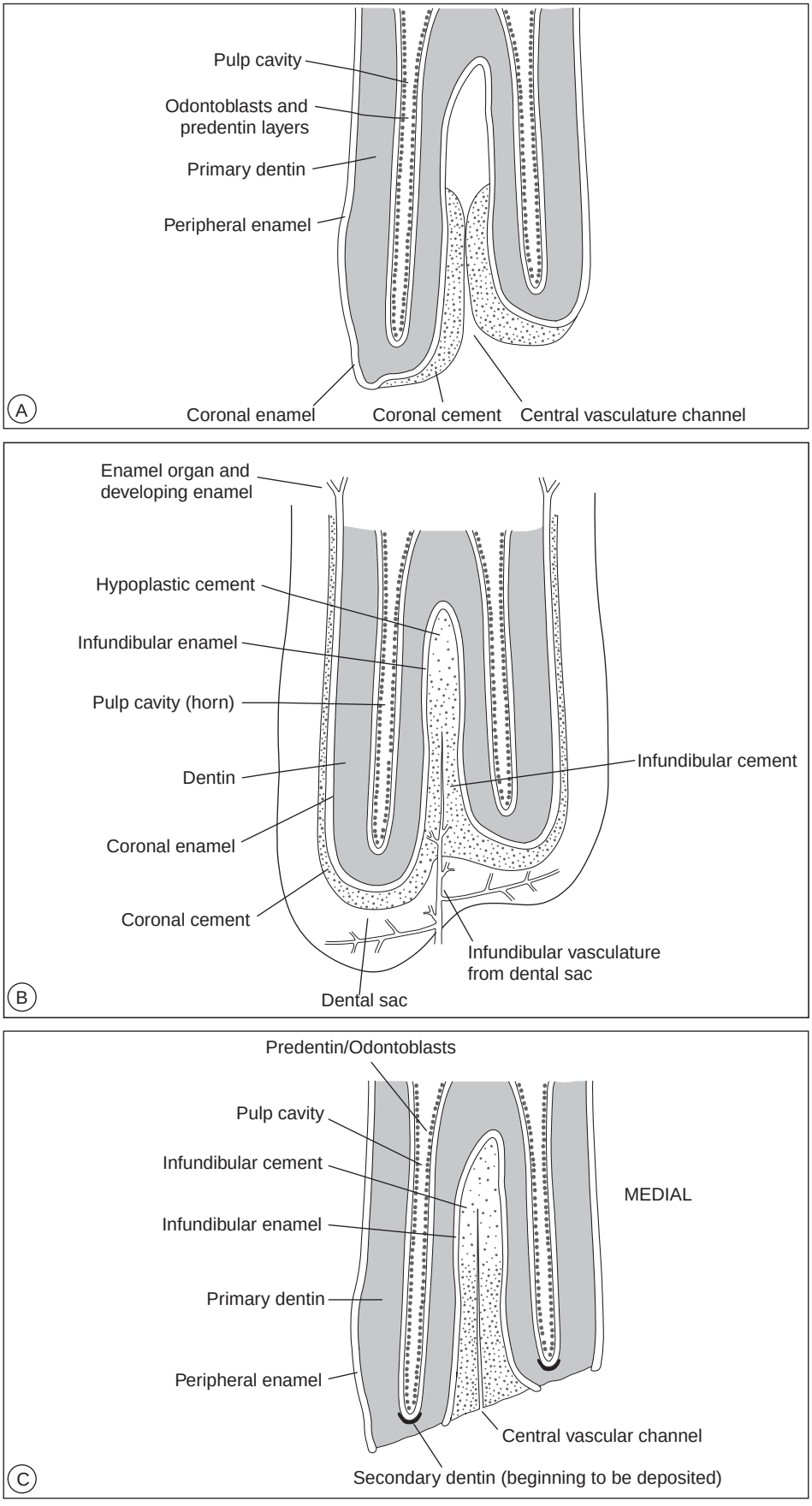


Figure 3.3. The crown and occlusal surface of a multicusped hypsodont tooth with an infundibulum (i.e an upper cheek tooth) (A) immediately prior to eruption, (B) immediately following eruption showing loss of the dental sac over the occlusal surface and (C) following wear of the primary occlusal surface to expose the secondary occlusal surface, which is the permanent occlusal surface in hypsodont teeth.

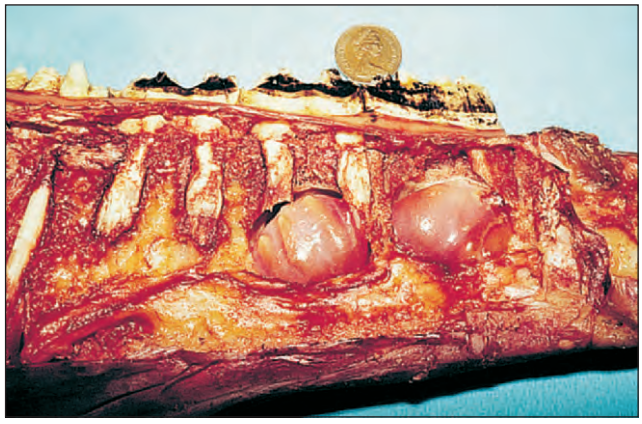


Figure 3.4. Dissected hemimandible of a yearling thoroughbred showing the tooth germs of the first and second mandibular cheek teeth developing beneath, and causing resorption of their temporary counterparts. Through the soft tissue of the surrounding dental sac, it can be seen that the more developed second cheek tooth germ has calcification of the occlusal aspects of the developing enamel cusps.

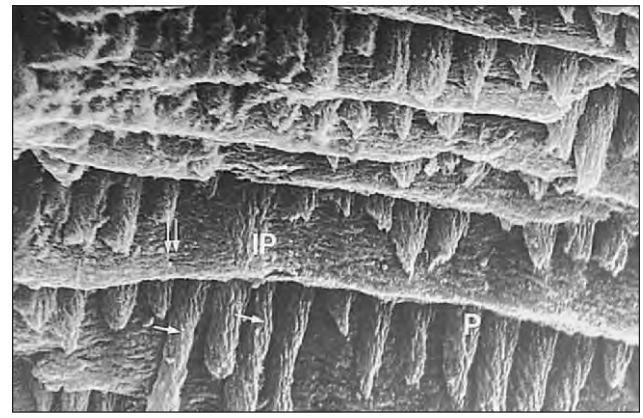


Figure 3.5. Scanning electron micrograph of Equine Type 1 enamel. This shows parallel rows of rounded enamel prisms (P) lying on flat plates of interprismatic enamel (IP). The enamel crystals within the enamel prisms (↓) are oriented parallel to the long axes of the prisms, while the enamel prisms of the interprismatic enamel plates are oriented at right angles to the prisms (↓↓). ×2720. (From Kilic *et al.*,[20] courtesy of the Editor of the *Equine Veterinary Journal*.)

Dental structures

Enamel

Enamel is the hardest and most dense substance in the body. Due to its high mineral (96–98 per cent) content it is almost translucent, but gains its color from that of the underlying dentin. Being ectodermal in origin, much of the limited organic component of enamel is composed of the keratin family of proteins, in contrast to the proteins of dentin and cement, which are largely collagenous (i.e. connective tissue, reflecting their mesodermal origin). In the equine tooth, enamel (except on the occlusal surface) is usually covered by dull, chalk-like peripheral cement. However, at the rostral aspect of the incisors this peripheral cement is usually worn away, thus exposing the shiny underlying enamel. The deciduous incisors often have little overlying cementum and thus appear whiter and shinier than the permanent incisors. Enamel, with its high mineral content and absence of cellular inclusions (unlike dentin or cement) can be regarded as almost an inert or 'dead' tissue. Therefore, as the ameloblasts die off once the tooth is fully formed, enamel has no ability to repair itself. Enamel is almost fully composed of impure hydroxyapatite crystals, which are larger than the equivalent crystals of dentin, cement or bone. These crystals are arranged both into structured prisms which may be contained in a prism sheath and also into less structured interprismatic enamel. Different species, different teeth within a species and even different areas of teeth in an individual can have different-shaped prisms or different arrangements of prismatic and interprismatic enamel, which can form the basis for enamel classification.

Equine enamel is composed of two main types termed Equine Types 1 and 2 enamel, with small amounts of Equine Type 3 enamel.[20] Equine Type 1 enamel is present on the medial aspect of the enamel folds, i.e. at the amelodentinal junction. It is composed of prisms that are rounded or oval on cross-section and lie in parallel rows between flat plates of dense interprismatic enamel (Figs 3.5 and 3.6). Equine Type 2 enamel is present on the periphery of the enamel layer, i.e. at the amelocemental (enamel to cement) junction, and is composed solely of enamel prisms ranging from horseshoe to keyhole in shape (Fig. 3.7) with no interprismatic enamel present. Equine Type 3 enamel is composed of prisms completely surrounded by large quantities of interprismatic enamel in a honeycomb-like structure and is inconsistently present as a thin layer at both the amelodentinal and amelocemental junctions (Fig. 3.7).

The distributions of Equine Type 1 and 2 enamels vary throughout the teeth, with Equine Type 2 enamel increasing in thickness in the peripheral enamel folds (ridges) and decreasing where these folds invaginate toward the center of



Figure 3.6. Scanning electron micrograph of Equine Type 1 enamel showing interprismatic plates (IP) alternating with rows of prisms (P). Note the convergence and branching (↓) of some of the interprismatic enamel plates. ×1450. (From Kilic,[53] with permission.)

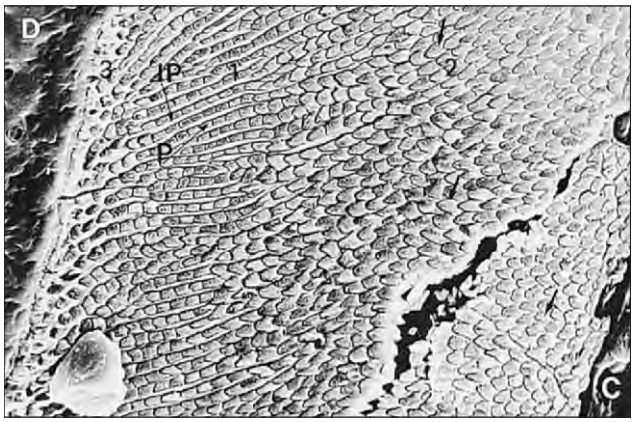


Figure 3.7. Scanning electron micrograph of a section of an equine tooth showing dentin (D) enamel and cement (C). A thin layer of Equine Type 3 enamel is visible on the left (3) at the junction with dentin. Adjacent to this area is a wider layer of Equine Type 1 enamel (1) showing interprismatic enamel (IP) (contiguous with Type 3 enamel and enamel prisms (P)). To the right is a wider layer of Equine Type 2 enamel (2) that in this area has horseshoe-shaped prisms (↓). ×482. (From Kilic *et al.*,[20] courtesy of the Editor of the *Equine Veterinary Journal*.)

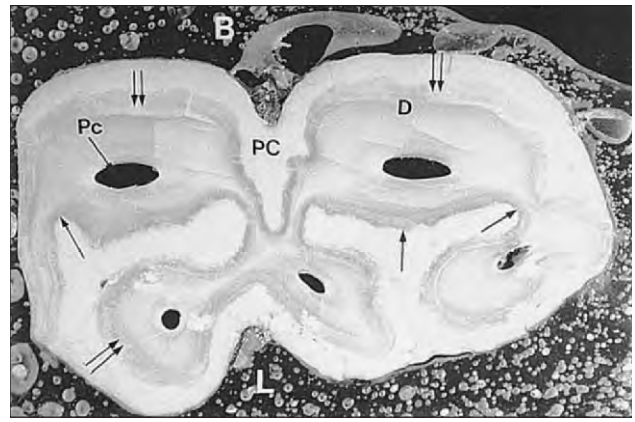


Figure 3.9. Transverse section, 2 cm beneath the occlusal surface of a methyl methacrylate embedded lower fourth cheek tooth of an 8-year-old horse. The enamel (peripheral only) is thickest (↓↓) in regions that are parallel to the long axis of the mandible, and thinnest (↓) in invaginations of enamel. One peripheral infolding is apparent on the buccal (B) aspect, while two deeper infoldings are present on the lingual (L) aspect. PC, peripheral cementum, D, dentin. ×4. (From Kilic,[53] with permission.)

the tooth (Figs 3.8 and 3.9). Almost all enamel folds contain both Types 1 and 2 enamel. However, increased amounts of Equine Type 1 enamel are present in the upper cheek teeth, and almost equal amounts of Equine Type 1 and 2 occur in the lower cheek teeth, whereas incisor enamel is composed almost solely of Equine Type 2 enamel. Equine Type 1 prisms are oriented at angles of approximately 45° to both the amelodentinal junction and the occlusal surface, but bundles of Equine Type 2 enamel prisms are oriented at a wide variety of oblique angles.[20]

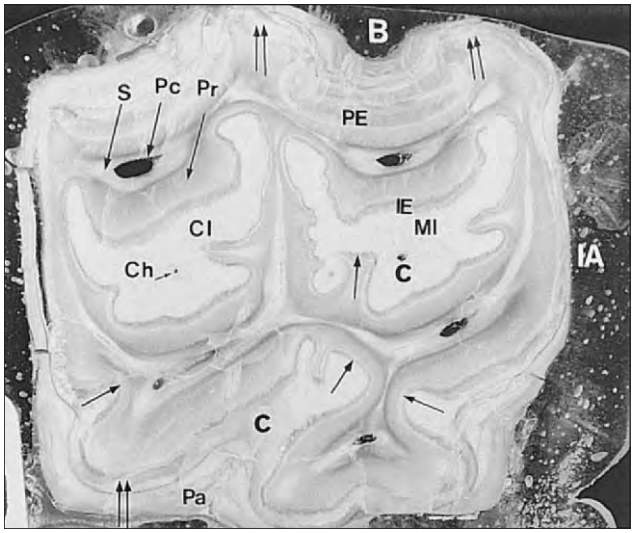


Figure 3.8. Transverse section, just beneath the occlusal surface of a methyl methacrylate embedded upper fourth cheek tooth of an 18-year-old horse. The mesial (rostral) infundibulum (MI) and the caudal (distal) infundibulum (CI) are surrounded by infundibular enamel (IE) and the infundibular cement has a central channel (Ch). Five pulp cavities (Pc) are present and are surrounded by areas of secondary dentin (S) that in turn is surrounded by primary dentin (Pr). Both the peripheral enamel (PE) and infundibular enamel (IE) are thicker at the palatal (Pa) and buccal (B) aspects than at the interdental aspects (IA). Additionally, the enamel is thicker in ridges (↓↓) than in invaginations (↓). ×4. (From Kilic,[53] with permission.)

Although enamel is the hardest substance in the mammalian body, it is brittle. The closely packed prisms of Equine Type 1 enamel form a composite structure with dense interprismatic plates that confer very strong wear resistance. However, these often-parallel rows of enamel prisms and interprismatic enamel are susceptible to cracking along prismatic and interprismatic lines. One adaptive process to prevent such cracks, that is particularly noticeable in Equine Type 2 enamel, is the presence of enamel decussation (interweaving, with changes of direction of bundles of enamel prisms) (Fig. 3.10). In contrast, Equine Type 1 enamel contains no decussation. Equine incisors are smaller and flatter than cheek teeth, have less support from adjacent teeth and yet undergo great mechanical stresses during prehension that could readily cause enamel cracks. Therefore it is not surprising that they are largely composed of Equine Type 2 enamel prisms. Cheek teeth primarily have a grinding function and so the presence of enamel that confers high wear resistance is more essential, and this in turn is fulfilled by the higher amounts of Equine Type 1 enamel in cheek teeth.[20] Close examination of cheek teeth enamel will often show the presence of fine transverse cracks (microfractures) through the peripheral enamel, which does not appear to be clinically significant, as the progression of these cracks through the remaining part of the tooth may be prevented by the adjacent cementum and dentin.

In equine cheek teeth, both peripheral and infundibular enamel are about three times thicker in areas where they are

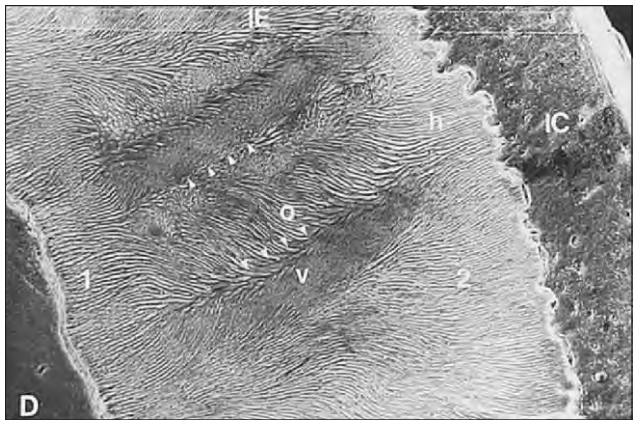


Figure 3.10. Scanning electron micrograph of a section of an equine incisor tooth, showing dentin (D), infundibular enamel (IE) and infundibular cement (IC). A thin layer of Equine Type 1 enamel is present on the left (1). The bulk of the enamel is Equine Type 2 (2) and this is oriented at a wide variety of angles including horizontal (h), obliquely (o) and vertically (v) relative to the occlusal surface. The bands of enamel oriented obliquely and vertically form alternating bands that are oriented perpendicular to the amelodentinal and amelocemental junctions with their junctions demarcated by grooves (Δ Δ Δ Δ). ×131. (From Kilic *et al.*,[20] courtesy of the Editor of the *Equine Veterinary Journal*.)

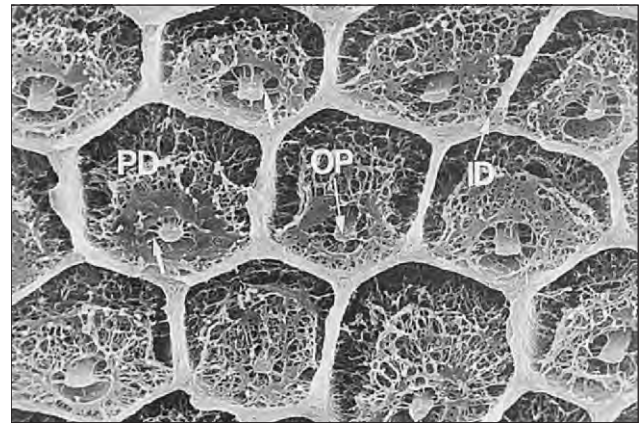


Figure 3.11. Scanning electron micrograph of a partially decalcified dentin. The hexagonal shaped intertubular dentin (ID) has a compact appearance. A network of collagenous fibers is apparent in the fully decalcified peritubular dentin (PD) and these fibrils are attached to the odontoblast processes (OP). ×2020. (From Kilic,[53] with permission.)

parallel to the long axis of the maxilla or mandible than where they are perpendicular to this axis, i.e. are invaginated into the tooth.[20] However, enamel thickness remains constant throughout the length of the teeth. Therefore, as the animal ages, the enamel thickness remains constant at the different sites in the transverse plane. It appears that enamel may have evolved to become thinner in certain regions of the tooth in response to localized reduced masticatory forces.

Dentin

The bulk of the tooth is composed of dentin, a cream-colored, calcified tissue composed of approximately 70 per cent minerals (mainly hydroxyapatite crystals) and 30 per cent organic components (including collagen fibers and mucopolysaccharides) and water. The latter content is obvious in dried equine teeth specimens where the dentin (and also cement) will develop artificial cracks. The mechanical properties, including tensile strength and compressibility of dentin, are highly influenced by the arrangements and relationships of its matrix collagen fibers (Fig. 3.11), other organic components and its calcified components, with the heterogeneity of its structure contributing to its overall strength.[21] High-powered examination of equine dentin shows that it contains both calcified fibers and calcospherites. In equine teeth, the presence of dentin (and also cement) interspersed between the hard but brittle enamel layers forms a laminated structure (like safety glass) and allows the two softer calcified tissues to act as 'crack stoppers' for the enamel[21] as well as creating an irregular occlusal surface, due to the differential wear between the hard enamel and the softer cementum and dentin.

Dentin can be divided into two main types, primary and secondary dentin, and the latter can be further subdivided into regular (physiological) and irregular (pathological, reparative or tertiary) dentin.[22,23] Some debate remains on the classification of irregular secondary dentin and tertiary dentin and this topic is more fully discussed in Chapter 9. Even in a morphological resting phase, odontoblasts remain capable of synthesizing dentin throughout their lives if appropriately stimulated,[12, 24] as do undifferentiated connective tissue cells of the pulp, which can differentiate into odontoblasts when appropriately stimulated. In equine teeth, odontoblasts synthesize *regular* secondary dentin on the periphery of the pulp cavity throughout life that gradually occludes the size of the pulp cavity and thus pulp (Fig. 3.12). This process has great practical significance because the occlusal surface of equine teeth would otherwise develop pulpar exposure due to normal attrition on the occlusal aspect. With insults, such as traumatic injury, infection or excessive attrition, primary dentin can respond by developing sclerosis of the primary dentinal tubules to prevent micro-organisms or their molecular products gaining access to the pulp, a defensive feature that is additional to the deposition of reparative (irregular) secondary dentin or tertiary dentin, as discussed later in Chapter 9.

As noted, the cream color of dentin largely contributes to the color of brachydont teeth. Because equine primary dentin contains very high levels of heavily mineralized peritubular dentin, it too has an almost translucent appearance similar to enamel. In contrast, the less mineralized regular secondary dentin (produced at the site of the former pulp cavity) has a dull opaque appearance. It also absorbs pigments from foods such as grass (but little from grains), which gives it a dark brown color that is obvious in the so-called 'dental star' of incisors or in the brown linear areas of secondary dentin that occur on the occlusal surface of cheek teeth that are in wear (Fig. 3.13).

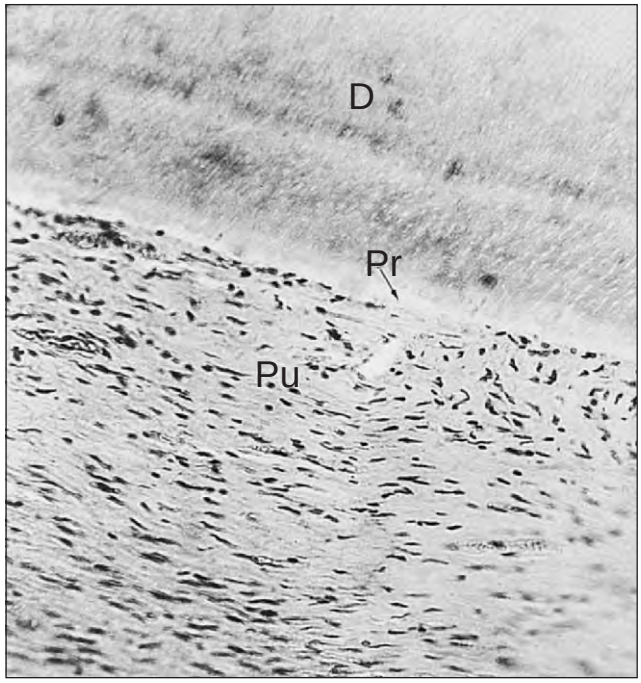


Figure 3.12. Light micrograph of a decalcified equine cheek tooth showing mineralized dentin (D), a thin layer of predentin (Pr) and the pulp (Pu) which contains cells – odontoblasts on the surface of the predentin and fibroblast-like cells within the remaining pulp. ×64. (From Kilic,[53] with permission.)

Dentin is composed of several distinct structures, including dentinal tubules, which are its characteristic histological feature, peritubular dentin (which forms the tubule walls), intertubular dentin (which lies between the tubules) and odontoblast processes. Dentinal tubules extend from the pulp cavity across the width of the tooth to the enamel (i.e. the amelodentinal junction). The odontoblasts reside in the predentin at the periphery of the pulp cavity but their odontoblast processes extend through the dental tubules (Figs. 3.11 and 3.14) as far as the enamel, sometimes subdividing into two or three tubules and displaying a sharp curvature just before reaching the amelodentinal junction. There is a debate on whether the odontoblast processes reach as far as the amelodentinal junction in other species, but in the horse it appears that the odontoblast processes do reach this far.[25] Because there is an intimate association between the pulp and dentin that causes them to act as a single

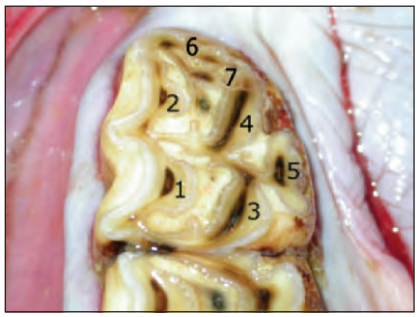


Figure 3.13. Maxillary cheek tooth showing seven pulp horns.

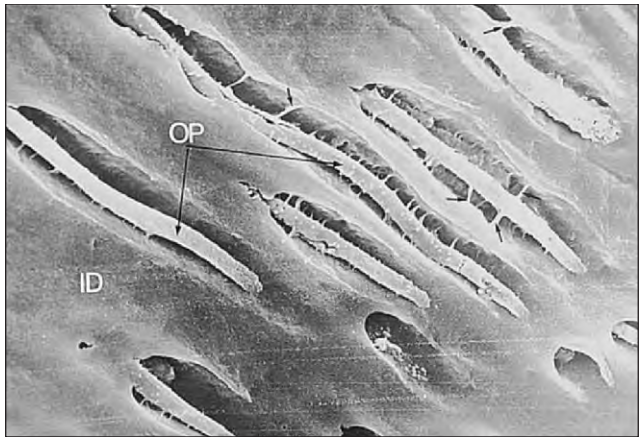


Figure 3.14. Scanning electron micrograph of an untreated dentinal section showing a longitudinal profile of dentinal tubules containing odontoblast processes (OP) that are attached to the intertubular dentin (ID) by calcified fibrils (↓). ×1010. (From Kilic *et al.*,[25] courtesy of the Editor of the *Equine Veterinary Journal*.)

functional unit, the term pulpodentinal complex is appropriately used for these two tissues. Because its tubules contains odontoblast processes, dentin is considered as a sensitive living tissue and thus grinding of dentin, e.g. reducing larger overgrowths that contain dentin, involves interference with odontoblast processes and thus potentially can cause pain.[26]

In brachydont species, odontoblast processes or their surrounding fluid can convey pain signals from insulted (e.g. by excessive heat or cold, trauma, infection) dentin to the pulp, by incompletely understood mechanisms. In horses, where exposed dentin constitutes a major part of the occlusal surface, it is most unlikely that such pain-producing mechanisms exist on the normal occlusal surface. It is interesting that on the occlusal surface of normal equine teeth, apparently intact, odontoblast-like processes are visible protruding from the dentinal tubules of primary and regular secondary dentin (Fig. 3.15), even though this area is constantly

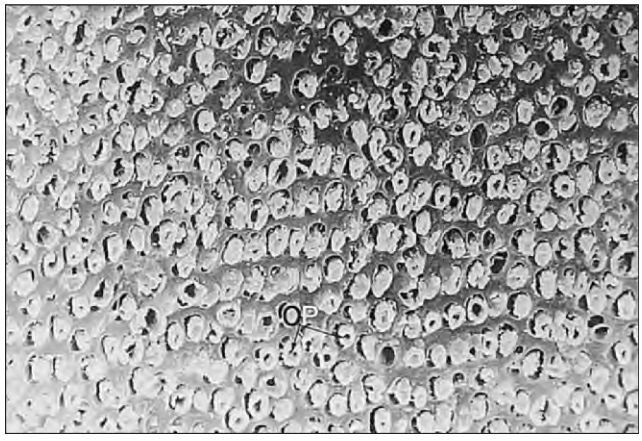


Figure 3.15. Scanning electron micrograph of the occlusal surface of an equine cheek tooth showing regular secondary dentin. Almost all of the dentinal tubules contain protruding odontoblast processes (OP) which are believed to be calcified, and many which are hollow. ×1010. (From Kilic,[53] with permission.)

exposed to oral microbial and biochemical insults.[27] A possible explanation for their apparently undamaged morphology is that they have become calcified. However, even if microorganisms could enter patent dentinal tubules on the occlusal surface, they may not reach the pulp cavity because the dentinal tubules are sealed by a smear layer of ground dental tissue and additionally, retrograde flow of fluid from dentinal tubules[28] may also prevent descent of microorganisms down these tubules. Irregular (reparative) secondary dentin is less organized than primary and contains no odontoblast processes as its dentinal tubules are fully obliterated and so can fully seal off the pulp.

Peritubular dentin (Fig. 3.11) has a higher mineral content than intertubular dentin and therefore has a higher resistance to wear. A transitional region exists between equine primary and secondary dentin where peritubular dentin is absent. Similarly, regular secondary dentin, which contains no (dense) peritubular dentin, is also more susceptible to attrition than primary dentin. Likewise, the dentin near the amelodentinal junction contains the lowest amounts of peritubular dentin and would theoretically be expected to wear faster, however it is protected from excessive wear by the adjacent enamel.

Pulp

The histology of equine teeth pulp has not yet been fully evaluated and most information is derived from studies on brachydont teeth pulp. Pulp is a soft tissue within the dental pulp cavities that contains a connective tissue skeleton, including fibroblasts, thick collagen and a network of fine reticulin fibers, connective tissue cells (that, as noted, can differentiate into odontoblasts if appropriately stimulated), many blood vessels (to allow active continuous secondary dentin deposition) and nerves (sensory and vasoregulatory). In mature teeth, pulp is contiguous with the periodontal connective tissue at the apical foramen. Peripherally, a thin layer of predentin (that becomes thinner in older brachydont teeth) lies between the dentin and pulp (Fig. 3.12), which as noted contains odontoblast cell bodies whose cytoplasmic processes extend into the dentinal tubules.

At eruption, equine permanent teeth possess a large common pulp that is contiguous with the primordial pulp that surrounds the developing apices (Figs 3.16 and 3.17). At the apex of these young teeth, only a thin layer of enamel surrounds this pulp. Later, following deposition of apical dentin and cement, root formation is complete in all equine cheek teeth by approximately 2 years after eruption, but the separate pulp canals may not develop until 5–6 years following mandibular cheek tooth eruption.[9] The above features have significant implications for endodontic therapy. The 07s to the 10s all contain five pulp cavities but the 06s and 11s usually contain six, the 11s occasionally having seven pulp horns (Fig. 3.13).[29]

Unlike brachydont teeth, hypsodont teeth need to continue to lay down secondary dentin over a prolonged period in order to prevent occlusal pulp exposure. Consequently,

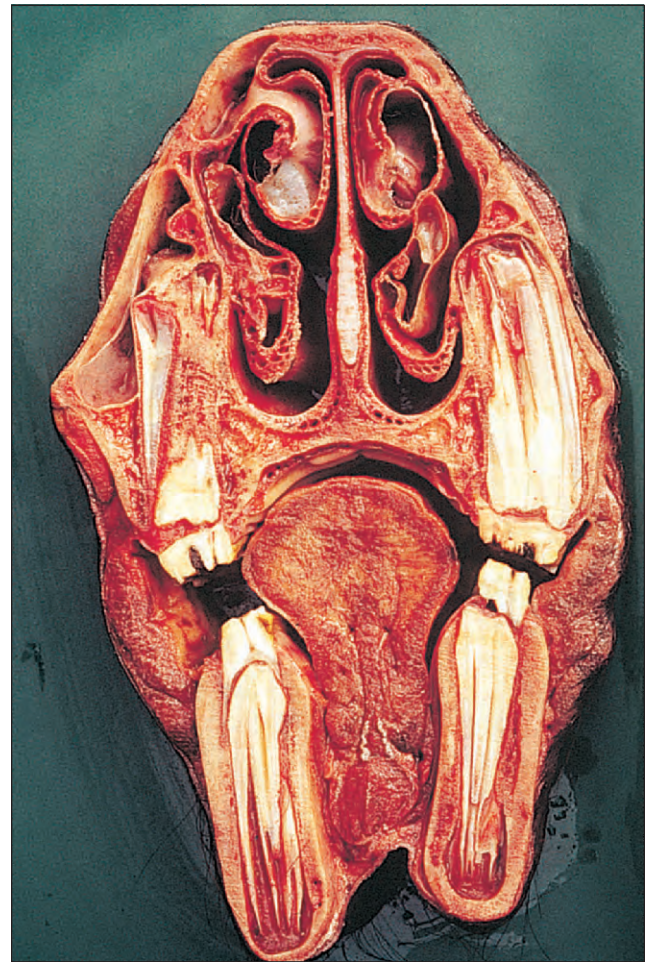


Figure 3.16. Transverse section of the skull of a 3.5-year-old horse at the level of the second maxillary cheek tooth on right and between the second and third cheek teeth on the left, with part of the third in the rostral maxillary sinus. Note the remnants of the deciduous teeth ('caps'), the angulation of the occlusal surfaces and the anisognathia. There are wide common pulp chambers of the apices of the permanent teeth and absence of roots. The infundibula of the temporary maxillary teeth have cemental hypoplasia.

in order to supply the metabolically active odontoblasts, the apical foramina, through which the tooth vasculature passes into the pulp, must remain relatively dilated ('open') for a prolonged period, although progressive reduction in foramen size does occur with age.[30] The apical foramina also becomes displaced more coronally by continued cement deposition at the apical aspect with age. Kirkland *et al.* found constricted ('closed') apical foramina in equine mandibular cheek teeth at 5–8 years after their eruption, with development of two apical foramina in the rostral (mesial) root.[9] This is in contrast to the apical foramina of brachydont teeth, which become more rapidly and extensively constricted ('closed') by deposition of secondary dentin within the pulp canal[1] and also by cement deposition externally.

A practical result of these features is that pulpar exposure in mature brachydont teeth causes pulpitis, which will compress and constrict the limited pulpar vasculature, usually

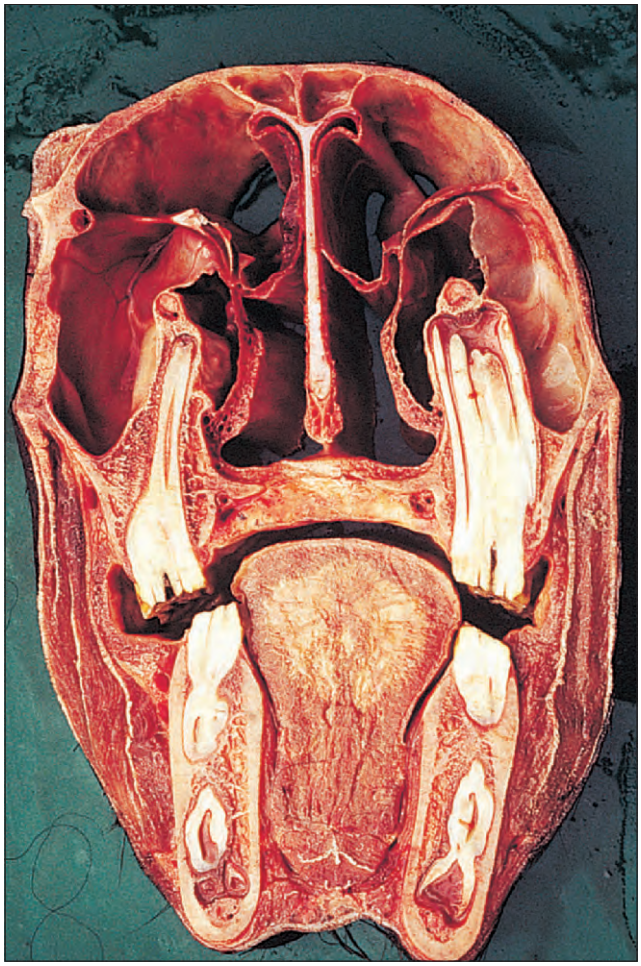


Figure 3.17. Transverse section of the skull of a 3.5-year-old horse at the level of the fifth maxillary cheek teeth that lie at the borders of the rostral and caudal maxillary sinuses. Due to their curvature, parts of the fifth and sixth mandibular cheek teeth are shown. The mandibular canal lies on the medial aspect of the mandible. The wide common pulp cavity of the left maxillary cheek tooth (with infra-orbital canal above) has pulp horns that extend to within 1 cm of the occlusal surface.

leading to pulpar ischemia and necrosis with death of the tooth. However, in hypsodont teeth – especially when young – the dilated apices and good blood supply often allows the pulp to withstand such inflammation by maintaining its blood supply. Local macrophages within the pulp, along with extravasated white blood cells and their molecules, can then control such pulpar infections. Additionally, the odontoblasts laying down secondary dentin can also quickly lay down reparative dentin in response to infection of the overlying dentin or following traumatic pulp exposure. In the absence of sufficient local odontoblasts, adjacent undifferentiated connective tissue cells or fibroblasts in the pulp can transform into odontoblasts and lay down reparative dentin.

As well as the occlusal aspect of the equine pulp cavity being progressively (fully) occluded with secondary dentin, the continued but slower similar deposition over all of the pulp cavity walls causes the overall pulp size to reduce with age, as the surrounding dentin becomes thicker. A practical consequence of this is that the cheek teeth in younger (e.g. <7–8 year old) horses contain a high proportion of hard but brittle enamel, and thus are somewhat shell-like. These teeth are readily rasped but may fracture if cut with shears (whose use is no longer advocated, as mechanical burrs are much safer). In contrast, the teeth of older horses contain large amounts of secondary dentin, which makes them more solid and less likely to shatter when cut, but more difficult to rasp (float) than young teeth. With age, the pulp of brachydont teeth loses much of its vasculature, fibroblasts and odontoblasts while its collagen content increases. This process may be delayed in equine teeth due to the prolonged higher metabolic activity of their pulp in laying down secondary dentin.

Cement

Cement (cementum) is a white or cream-colored calcified dental tissue with mechanical characteristics and a histological appearance similar to bone. It contains approximately 65 per cent inorganic (again mainly impure, hydroxyapatite crystals) and 35 per cent organic and water components. Similar to dentin, its high organic and its water content give it flexibility. The organic component of cement is composed mainly of extensive collagen fibers that include small intrinsic fibrils (produced by cementoblasts) and larger extrinsic fibers (produced by fibroblasts of the periodontal membrane), some of which form tight bundles termed Sharpey's fibers (median 2.5 microns in diameter in horses) that cross the periodontal space to become anchored in the alveolar bone[3] (Fig. 3.18), thus indirectly attaching the cement and alveolar bone. Cementum may be classified as cellular or acellular; peripheral or infundibular; coronal or root.

Under polarized light undecalcified (ground) transverse sections of equine cheek teeth show two distinct regions. Adjacent to the peripheral amelo-cemental junction, the

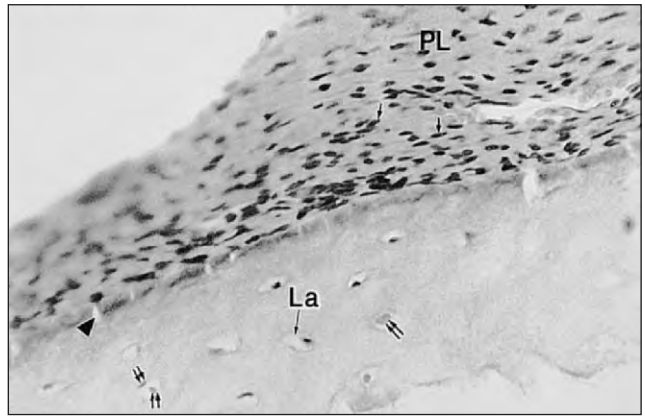


Figure 3.18. Light microscopy of the periphery of an upper cheek tooth showing the periodontal ligament (PL) containing fibroblast-like cells (↓). The adjacent peripheral cement contains lacunae (La) of the cementoblasts (↓↓). Projections of the periodontal ligament into the cementum (▲) probably represent Sharpey's fibers. ×1000. (From Kilic *et al.*,[31] courtesy of the Editor of the *Equine Veterinary Journal*.)

crystalloid nature of the cementum is observed to be irregular in hydroxyapatite crystal orientation. This is similar to maxillary cheek teeth infundibular cement. Beyond this, its nature changes to become regular, with crystals having a similar concentric orientation. It is in this zone that 'peripheral lines' may be observed in decalcified transverse sections. These two zones of regular and irregular peripheral cementum are more developed in sections of older teeth near the occlusal surface.[31]

Like dentin, cement (of subgingival area only, i.e. of reserve crown and roots) is a living tissue with its cells (cementoblasts) nourished by the vasculature of the periodontal ligament. Cement and its periodontal membrane can be considered as a single functional unit[29] as are dentin and pulp. After eruption onto the clinical crown, cementoblasts lose their blood supply from the periodontium and therefore cement on the clinical (erupted) crown can be regarded as an inert tissue. However, recent work has shown active vasculature extending from the gingival margin beneath the surface of cementum on the clinical crown.[2] Cement is the most adaptable of the calcified dental tissues and can be quickly deposited (within the alveolus or subgingivally) in response to insults such as infection or trauma[32] as commonly observed in teeth with more chronic apical infections (see Chapter 9). In hypsodont teeth, cement covers all of the crown (including the occlusal surface transiently after eruption) (Fig. 3.13) and fills the infundibula (often incompletely).

In hypsodont teeth, cement deposition continues throughout the life of the tooth, both around the roots (root cement) and also on the reserve crown (coronal cement) (Fig. 3.19). The latter allows new Sharpey's fibers (Fig. 3.20) to be laid down, a process necessary to allow both the prolonged eruption of hypsodont teeth and additional cement deposition to contribute to the clinical crown. However, further cement cannot be deposited in an infundibulum that has lost its blood supply following tooth eruption, or likewise, on the cement of the clinical crown once it moves away from the gingival vasculature. The main functions of cement are to provide anchorage for fibers of the periodontal ligament that support (with some flexibility) the tooth in the alveolus and to protect the underlying dentin at the dental apex. These two features of cement are present in both brachydont and hypsodont teeth. However, in hypsodont teeth, cement has major additional roles by contributing significantly to the bulk of the clinical crown (especially in the lower cheek teeth), protecting the coronal enamel from cracking and helping to form the protruding enamel ridges on the occlusal surface.

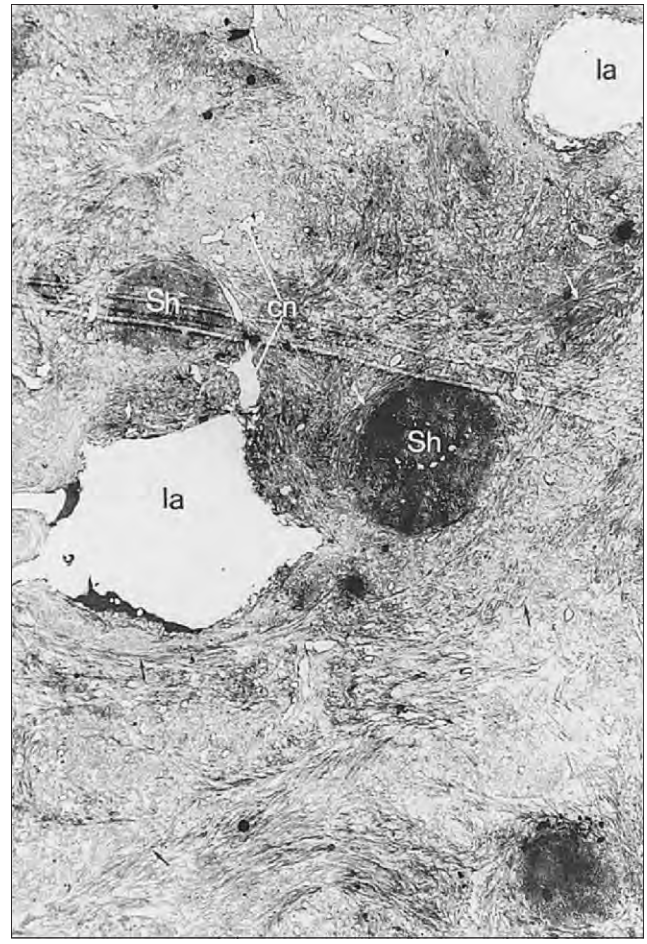


Figure 3.20. Transmission electron micrograph of peripheral cement of a cheek tooth. This shows irregularly shaped lacunae (la) and their canaliculae (cn) but the cementoblasts have been lost during sample preparation. The dense Sharpey's fibers (Sh) have been transversely sectioned. The intrinsic fibrils of the cement (↓) are also apparent. ×2150. (From Kilic *et al.*,[31] courtesy of the Editor of the *Equine Veterinary Journal*.)

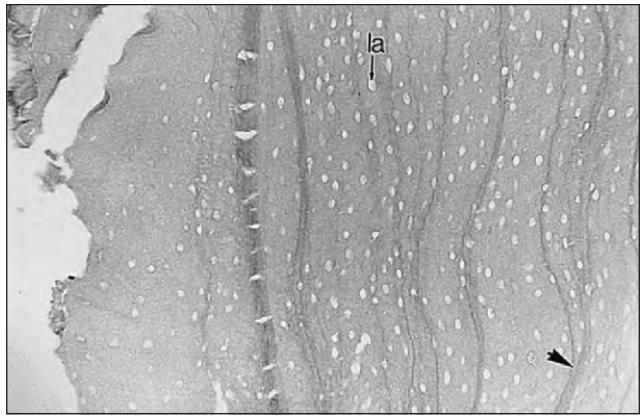


Figure 3.19. Light micrograph of the peripheral cement of the deep reserve crown (adjacent to the apex) of a recently erupted cheek tooth. This contains wavy incremental lines (➤) between successive depositions of cement that have occurred even at this early stage of tooth growth. Cementoblast lacunae (la) are present at all levels of the cement. ×44. (From Kilic,[53] with permission.)

To provide additional cement on the clinical crown, there is a large increase in cement deposition once the tooth exits from the spatial restrictions of the alveolus[2] (Fig. 3.21). In older hypsodont teeth, cement significantly contributes to the size and strength of the remaining tooth to compensate for crown wear and also to protect the enamel from cracking.[2,30] In some aged horses, the dental remnants exposed at

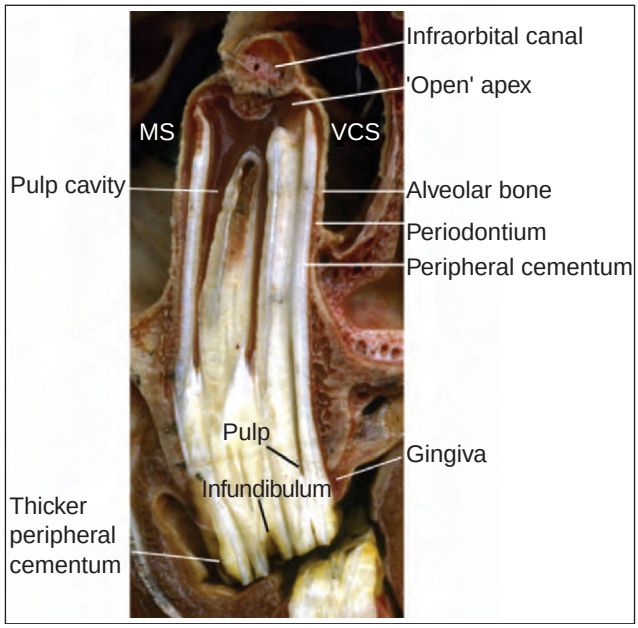


Figure 3.21. Longitudinal section of a young maxillary cheek tooth lying in the maxillary sinus (MS) and ventral conchal sinus (VCS). Note the very extensive pulp chambers and limited amount of (secondary) dentin present, which is characteristic of young equine teeth. Localized (clinically insignificant) central cemental caries is present in the transected infundibulum. The alveolar bone and periodontal membrane can be identified adjacent to the cement at the periphery of the tooth. Note the increase in thickness of the peripheral cement (of the 'gingival reserve crown') immediately following eruption of the tooth from the alveolus.



Figure 3.22. Occlusal view of a maxillary cheek teeth row of an aged horse. Just the roots (rostral roots separate) that have heavy peripheral cement deposits remain of the 109 and 111. These remnants contain little enamel ('smooth mouth') and consequently will soon fully wear out. The infundibula of most of the remaining teeth have fully worn out and diastema is present between the more rostral teeth.

the occlusal surface may eventually be composed only of roots (dentin and cement) with surrounding heavy cemental deposits. As this dental remnant contains no enamel it becomes smooth on its occlusal surface ('smooth mouth') and wears away quickly (Fig. 3.22).

There is little peripheral cement in incisors and canines, but much greater amounts in cheek teeth, where its thickness varies greatly, largely depending on the degree of infolding of peripheral enamel. It is thickest in deeply infolded areas, especially in the two folds on the medial aspect of the lower cheek teeth (Fig. 3.9). At these sites, especially toward the tooth apex, this thick peripheral cement can be fully enclosed by these deep enamel folds, and these areas of cement can resemble infundibula.

As noted, the infundibula (in all incisors and the upper cheek teeth) are usually incompletely filled by (infundibular) cement. Kilic *et al.* found that in addition to the 24 per cent of (upper) cheek teeth that had gross caries (mineralized dental tissue dissolution) of their infundibular cement (Fig. 3.23), a further 65 per cent of horses had one or more small central vascular channels in this cement.[27,31] These channels extended from the occlusal surface to a variable depth, and contained smaller lateral channels extending as far as the infundibular enamel. This type of cement hypoplasia was termed 'central infundibular cemental hypoplasia.' In addition, some infundibula had linear areas of cement hypoplasia at the enamel junction termed 'junctional cemental hypoplasia.'[31] As this latter cemental hypoplasia was commonly found in incisor infundibula that show little evidence of caries (albeit they are much shallower infundibula than those of cheek teeth), consequently junctional cemental hypoplasia is not believed to be clinically significant.[31]

The occlusal surface

At eruption, the crowns of equine teeth, including the occlusal surface, are fully covered by coronal cement, which in turn covers a thin layer of coronal enamel. With normal occlusal wear, the coronal cement and coronal enamel are

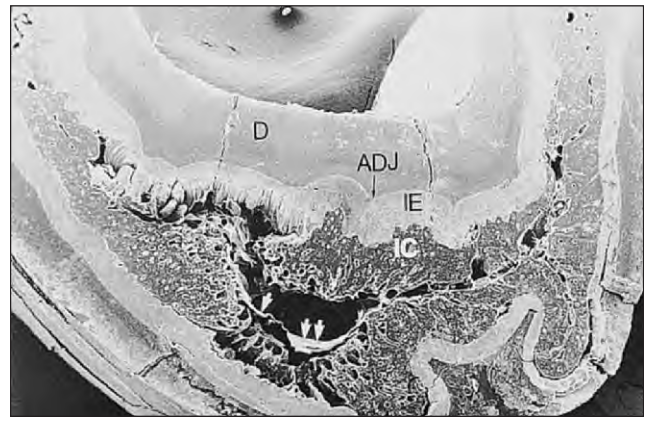


Figure 3.23. Scanning electron micrograph of the deep infundibular area of an upper cheek tooth of a 14-year-old horse. This section contains dentin (D), an amelodentinal junction (ADJ), infundibular enamel (IE) and infundibular cement (IC). The infundibular cement shows extensive hypoplasia, with the large central defect partially lined by shrunken organic tissue (▼) and also exposure of the infundibular enamel in several areas. ×10.6. (From Kilic *et al.*,[31] courtesy of the Editor of the *Equine Veterinary Journal*.)

very soon worn away, thus exposing the secondary occlusal surface of these teeth, which in fact is the permanent occlusal surface of hypsodont teeth (Fig. 3.3). The wear process on the occlusal surface is a complex phenomenon depending on many factors including the type of diet, e.g. in the winter outdoor horses may be forced to graze lower and thus ingest more soil-covered roots and leaves, or even eat the roots of plants such as nettles (M Booth 1996, personal communications), thus greatly increasing the amount of silicates that are ingested. When grazing is scarce, horses may also eat coarser food including bushes, such as gorse. The duration of eating also varies according to the season from up to 13 hours/day in summer to 16.5 hours/day in winter in outdoor horses, in some environments (M Booth 1996, personal communications). While eating hay, horses and ponies have 58–66 chewing movements per minute, with 4200 chews/kg of dry matter,[33] while at grass they have 100–105 chews per minute.[34] Dental attrition also depends on the force and the direction of the chewing action, the sizes, shapes and angles of the opposing occlusal surfaces and the relationship of opposing cusps and crest patterns to the occlusal motion. Consequently, painful oral disorders can cause changes in the direction and forces of mastication and thus will affect the wear patterns of cheek teeth.

The occlusal surfaces of equine teeth contain an organic pellicle[27] containing micro-organisms and small food particles and a smear layer of finely ground dental particles formed by masticatory action. The underlying enamel contains differing wear patterns, including polished areas, small local fractures, pit striations and depressions. Most large striations are at right angles to the long axis of the dental arcades (the buccolingual plane) and appear to be caused by the normal side-to-side chewing motion of the cheek teeth during the grinding down of small ingested phytoliths (calcified plant particles). In these deep grooves, scanning electron microscopy shows that prismatic enamel is more deeply worn than interprismatic enamel, confirming that the former is softer. Additionally, some shorter striations are present on the occlusal surface of equine teeth parallel to the buccolingual plane (at right angles to the normal chewing direction) and it is suggested that these striations occur due to ingested phytoliths during the crushing phase of chewing.[27]

This softer dentin on the occlusal surface wears quicker than the surrounding enamel and therefore the dentinal surface becomes depressed. The depth of these depressions is directly related to the area of the dentin, with larger exposed areas more deeply recessed. In contrast, smaller exposed areas, being better protected by the surrounding enamel folds, have less wear. Therefore, the orientation and invaginations of the enamel folds (peripheral and infundibular) play an important role in dividing the occlusal surface of dentin into smaller areas and thus protecting it from excessive attrition. There may be less infolding of peripheral enamel folds more apically in teeth and thus some older teeth may show excessive dentinal wear at such unprotected areas (Fig. 3.24). Similarly, if infundibula are absent or are short and wear

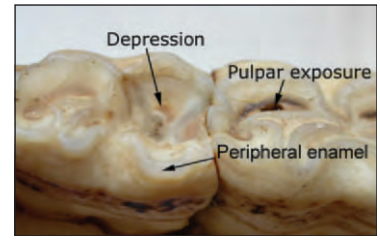


Figure 3.24. Two mandibular cheek teeth, with the tooth on the left showing limited peripheral cement infolding of its caudal aspect - with consequent excessive wear of the adjacent dentin causing a depression in this area of the occlusal surface ('cupping'). The adjacent cheek tooth has normal enamel infolding, but has occlusal exposure of one of its pulps.

away prematurely (in upper cheek teeth), excessive local dentinal wear will also occur around their site (Fig. 3.25). In this respect, the lower cheek teeth have three very deep infoldings of enamel, two on the medial (lingual) aspect and one on the buccal aspect. The upper cheek teeth have less peripheral enamel infolding; however, they contain two enamel infundibula, which further subdivide and compartmentalize their occlusal dentin, thus protecting it also.[27]

Gross anatomy of equine teeth

Incisors

The deciduous 01s (central), 02s (middle-intermediate) and 03s (corner) incisors erupt within a few days of birth, 4–6 weeks and 6–9 months of age, respectively.[35] Deciduous

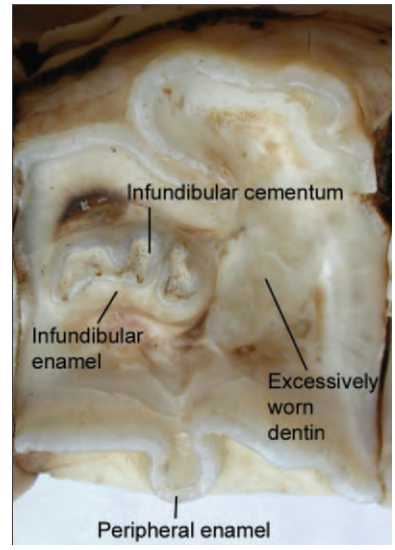


Figure 3.25. Occlusal surface of a maxillary cheek tooth which is missing one of its infundibula, with resultant excessive wear causing a deep depression ('cupping') in the occlusal surface at this site. This 'cupping' may predispose to dental fracture. Unusually, the remaining infundibulum appears to consist of two separate, smaller infundibula.

incisors are whiter and contain wider and shallower infundibula than their permanent successors, which erupt on their lingual aspects. As noted, the eruption of both deciduous and permanent teeth can be used to estimate the age of horses up to 5 years old with a reasonable degree of accuracy[36,37] (see Chapter 5).

The dental formulas of deciduous and permanent teeth in horses are:

- Deciduous teeth: 2 (Di 3/3, Dc 0/0, Dm 3/3) = 24 teeth.
- Permanent teeth: 2 (I 3/3, C 1/1 or 0/0, PM 3/3 or 4/4, M 3/3) = 36 to 44 teeth, depending on the presence and number of canine teeth or first premolar (wolf teeth).[38]

The Triadan system of dental nomenclature utilizes three digits to identify each tooth; the first digit refers to the quadrant, with 1 for upper right, 2 for upper left, 3 for lower left and 4 for lower right[39] (Fig. 3.26). The deciduous teeth are similarly identified using the prefix 5–8 for the four quadrants.

Adult horses also have 12 incisors in total, six in each arcade. The upper incisors are embedded in the premaxillary (incisive) bone and the lower incisors in the rostral mandible with the reserve crowns and apices of incisors converging toward each other. Incisor teeth are curved convexly on their labial aspect (concavely on their lingual aspect) and taper in uniformly from the occlusal surface toward the apex (unlike equine deciduous and all brachydont incisors, which have a distinct neck). Therefore with age, spaces will eventually develop between equine permanent incisors, but this is delayed by the medial (mesial) pressure of the 03s on the remaining incisors. The fully developed incisor arcade in a young adult horse has an almost semicircular appearance, which gradually becomes shallower with age, due to alteration of teeth shape caused by progressive wear.[40] The occlusal angle of incisors also changes from almost vertical apposition in the young horse (Fig. 3.27) to an increasing angle of incidence with age.

Equine incisor teeth also develop certain wear-related macroscopic features that have also been traditionally (if not very accurately) utilized for estimating age[36–38] as discussed in detail in Chapter 5. The infundibulum present in all incisors is termed the incisal 'cup.' This funnel-like enamel structure is almost circular in shape and approximately 10 mm deep when the tooth first erupts. However, variations in its depth may cause the infundibulum to wear away quicker or slower than 'normal' and thus make aging difficult. The infundibulum is usually incompletely filled with cement and later, becomes filled with food material and appears dark.[36] When the infundibular cavity is worn away, it leaves behind a small ring of the remaining infundibular enamel, located on the lingual aspect of the tooth, which is called the enamel spot (enamel ring or mark).[36] Due to the slower wear of enamel compared with dentin, the enamel spot becomes elevated above the occlusal surface. The dental star represents exposure of secondary (regular and irregular) dentin that has been deposited within the former pulp cavity on the occlusal surface of incisor teeth. It appears sequentially in the 01s, 02s and 03s (see Chapter 5). It initially appears as a dark yellow, transverse line on the labial aspect of the infundibulum. With further tooth wear, it gradually becomes oval in shape and moves toward the center of occlusal surface.

Galvayne's groove is a longitudinal groove that appears on the labial aspect of the upper 03s (corner incisors). A 'hook' (a colloquial term for a localized dental overgrowth) is often recognized at the caudolabial aspect of the occlusal surface of 103 and 203 after approximately 6 years of age, due to incomplete occlusal contact between the upper and lower 03s. It is often termed a '7-year notch or hook' because it was traditionally (but erroneously) believed to always appear at 7 years of age.[7,40] Variations in incisor teeth appearance can also be due to individual and breed variation, differences in diets, environmental conditions, eruption times, mineralization rates, depth of enamel infundibulum, amount of infundibular cement and the presence of certain stereotypic behaviors such as crib-biting and wind-sucking.[41,42] The occlusal surface of individual incisors is elliptical in recently erupted incisors, but with wear, they successively become round, triangular and then oval in shape. These changes are more apparent in the lower 01s (centrals) and 02s (intermediates) than in the lower 03s (corner incisors).[37,38]

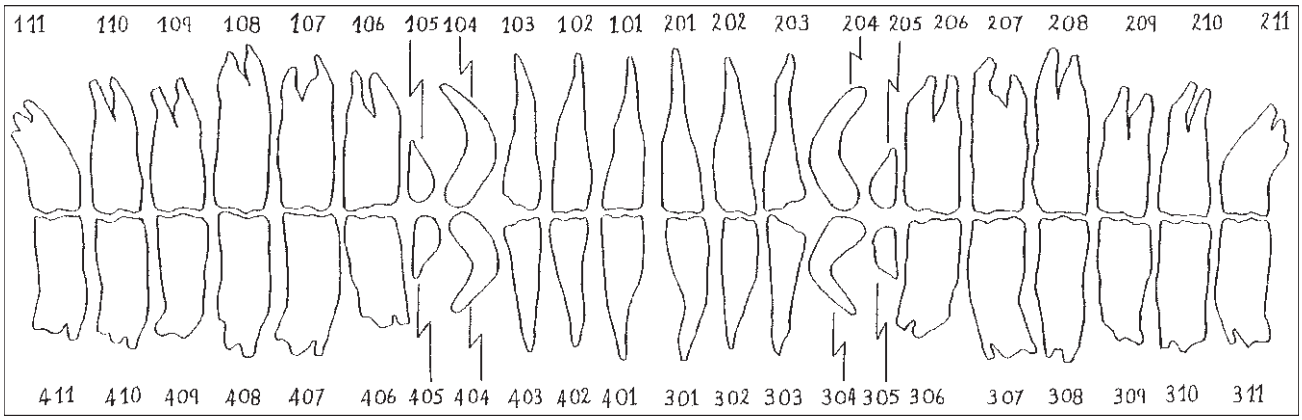


Figure 3.26. The Triadan classification of equine teeth.

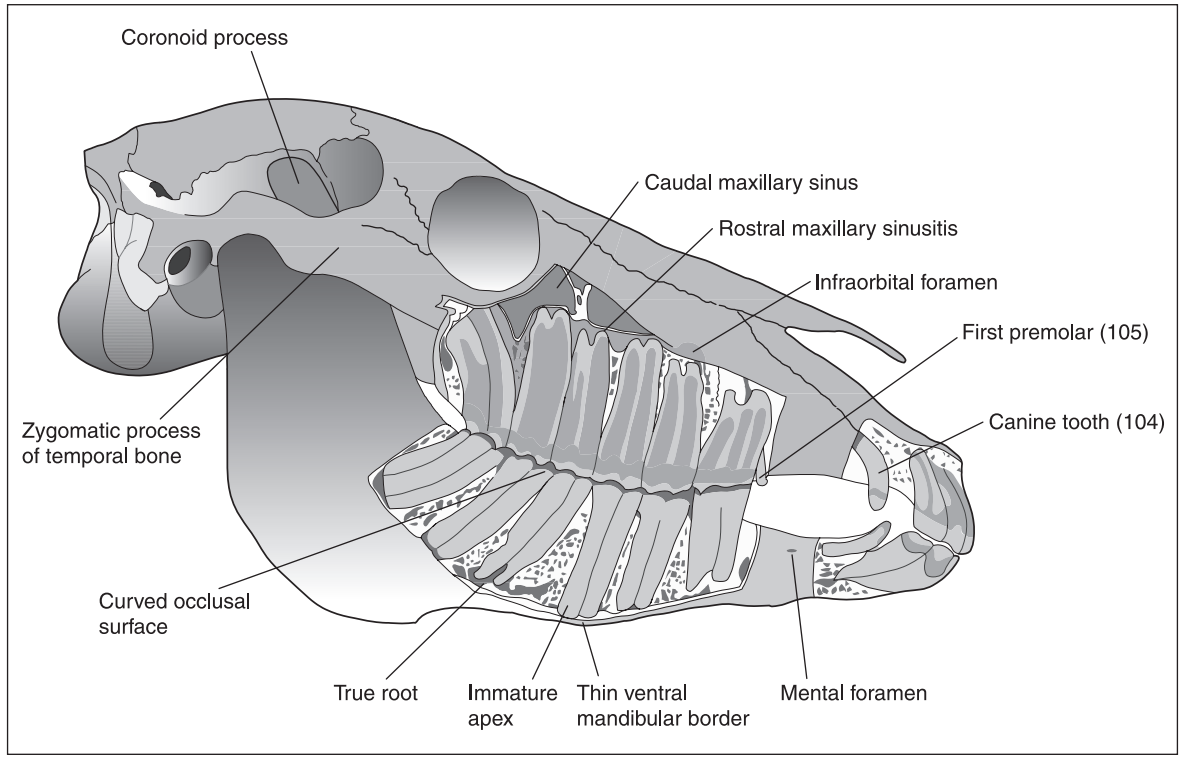


Figure 3.27. Diagram of a skull of a 5-year-old horse demonstrating ventral deviation of the mandible associated with eruption of 408. Note the shape and apposition of the incisors of this young horse. The angulation of the rostral and caudal cheek teeth and the curvature of the sixth teeth maintain tight apposition of all six cheek teeth at the occlusal surface. The temporomandibular joint is high (approximately 15 cm in an adult thoroughbred) above the level of the occlusal surface. Note the small coronoid process (CP) and the large area of mandibular muscle attachment.

Canine teeth

The deciduous canine teeth are vestigial spicule-like structures, 0.5–1.0 cm long, that do not erupt above gum level. The lower deciduous canine is situated caudal to the corner incisor.[35] Male horses normally have four permanent canine teeth (two maxillary and two mandibular) that erupt at 4–6 years of age in the interdental space.[38] They are simple teeth (i.e. contain no coronal cement or enamel folding) and are pointed. Canine teeth are convex on their buccal borders, and slightly concave on their medial (lingual and buccal) aspect, with a caudal facing curve. The lower canines are more rostrally positioned than the upper and thus there is no occlusal contact between them. This is alleged to be a reason why canine teeth (especially the lowers) are prone to develop calculus. Canine teeth are usually absent or rudimentary in female horses with a reported incidence of 28 per cent.[41] Canines do not continually erupt like cheek teeth and thus long reserve crowns can be present in older horses. In the young adult thoroughbred, they are 5–7 cm long with most as unerupted crowns. In some horses just 10–20 per cent of the crown is erupted and consequently, due to the great length and size of the reserve crown and roots, extraction of these teeth is a major undertaking. Canine teeth have a wide pulp cavity that in young adult horses may extend to within 5 mm of the occlusal surface, consequently excessive grinding down of canines in horses risks causing pulpar exposure.

First premolar ('wolf tooth')

One or both of the upper 05s (first premolar) and less commonly, the lower 05s can be present as the small, vestigial 'wolf teeth.' These should normally lie immediately in front of the 06s. They are simple brachydont teeth whose clinical crown can vary from 1–25 cm in length. Their roots can vary from being non-existent (with loose attachments to the gingiva), to being up to 30 mm in length. These teeth are sometimes rostrally or rostrolaterally displaced and also may be angulated (i.e. not vertical in relation to the hard palate). The permanent 05s usually erupt at 6–12 months of age and they do not have a deciduous precursor. They have a reported incidence of 24.4 per cent in females and 14.9 per cent in males,[43] and of 13 per cent[19] to 31.9 per cent[44] in horses of both sexes. This wide range of incidence may be due to loss of some 05s at *ca* 2.5 years of age, when the adjacent deciduous 06s are shed (J Easley, personal communications).

Cheek teeth

The 12 temporary premolars are erupted at birth or do so within a week after birth. These deciduous teeth are replaced

by the larger, permanent premolars at approximately 2.5, 3 and 4 years of age for the 06s to the 08s, respectively. In contrast to brachydont teeth and to equine incisors (where the deciduous teeth are much smaller than the permanent teeth) the transverse (cross-sectional) area of equine deciduous cheek teeth can be somewhat similar to those of adult teeth[30] and thus a retained remnant of a deciduous cheek tooth ('cap') can be difficult to identify from the underlying permanent tooth. The three deciduous cheek teeth in each row have a distinct neck between the crown and roots, unlike their permanent successors.[45] Latterly these deciduous cheek teeth erupt into the oral cavity due to traction by their periodontal ligaments and pressure from the underlying permanent tooth. They are simultaneously resorbed at their apices by immunologically mediated mechanisms until eventually just a thin 'cap' of the temporary tooth remains on the occlusal aspect of the permanent cheek tooth (Fig. 3.16).

An adult equine mouth normally contains 24 cheek teeth (06s–11s, i.e. premolars 2–4, and molars 1–3; the latter erupt at approximately 1, 2 and 3.5 years of age, respectively), forming four rows of six teeth that are accommodated in the maxillary and mandibular bones. On transverse section, equine cheek teeth are rectangular, except the first and last, i.e. PM2 and M3, which are somewhat triangular (Figs 3.13 and 3.28). The maxillary cheek teeth are wider and squarer in comparison with the mandibular cheek teeth, which are narrower and more rectangular in outline. The long axis of all cheek teeth is relatively straight, except the sixth and to a lesser and variable extent the fifth, which have a caudal curvature of their reserve crowns (later, more so of their roots) (Fig. 3.27). The buccal aspect of the upper cheek teeth have two prominent vertical (longitudinal) ridges (cingula, styles) rostrally and less prominent caudal ridges with two deep grooves between them, except the first, which can have three or four small grooves and ridges. These ridges can vary in size, but are often most prominent on the (upper) 10s and 11s. Dental overgrowths can often be prominent on the occlusal aspects of these ridges (especially on larger ridges such as 10s and 11s) even in horses on a permanent forage diet. The palatal aspect of the upper, and both lingual and buccal aspects of the mandibular cheek teeth contain much less distinct vertical grooves and ridges (Figs 3.8 and 3.9).

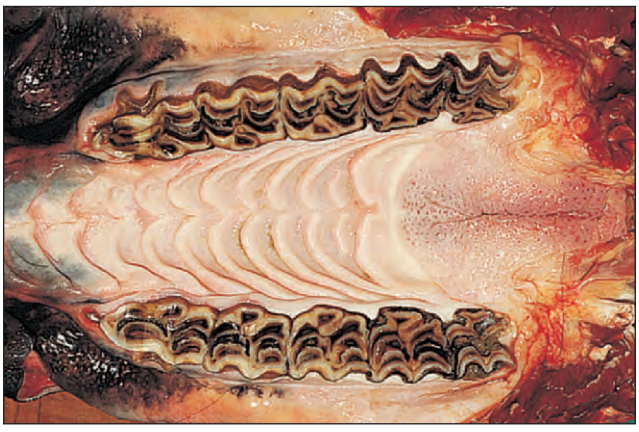


Figure 3.28. Occlusal view of the five maxillary cheek teeth of a 3-year-old horse. Note the absence of spaces between these teeth, the more pronounced ridges on the buccal aspects of these teeth and the triangular shape of the first tooth.

In younger horses, the permanent cheek teeth possess long crowns, most of which is unerupted reserve crown that is embedded in the deep alveoli (Figs 3.16 and 3.17). In Thoroughbreds, the 06 is the shortest (approximately 5 cm maximum length), with the remaining cheek teeth up to 6–8 cm long at eruption. Dental eruption proceeds throughout the life of equine teeth and normally the eruption rate corresponds with tooth wear (attrition) and has been calculated as 2–3 mm per year.[4,5] Therefore, since all equine permanent cheek teeth come into wear by 5 years of age, a 75 mm-long tooth should be fully worn by 30 years of age.[4]

Once fully developed, the upper cheek teeth usually have three roots (two small lateral and a larger medial), but occasionally have four.[46] The lower cheek teeth (except the 11s which have three) have two equally sized roots, one rostral and one caudal, that tend to become longer than those of their maxillary counterparts.

The alveoli of the first two upper cheek teeth (06s and 07s) and often the rostral aspect of the 08, and usually all of this tooth in older horses[45] are embedded in the maxillary bone (Fig. 3.16). The caudal aspects of the 08s and the 09s lie in the rostral maxillary sinus and the 10s and 11s lie in caudal maxillary sinus (Fig. 3.17). However, there can be much variation in this finding, with the rostral aspect of the rostral maxillary sinus varying from overlying the middle of the 07s to the middle of the 09s, and the shell-like, bony transverse maxillary septum (separating the rostral and caudal maxillary sinuses) varying in position from the caudal aspect of the 08s to the caudal aspect of the 09s.[47,48] In young horses, the large reserve crowns occupy much of these maxillary sinuses, but with age and subsequent eruption of their reserve crowns and retraction of their alveoli, the residual sinus cavities increase in volume, as the floor of the sinuses lowers with teeth eruption. In younger horses, the infra-orbital canal lies directly over the apices and is often curved dorsally at this site, whereas in the older horse a thin plate of bone (dividing the sinus saggitally) connects the alveoli to the infra-orbital canal.

Additionally, the head and sinuses lengthen with age and also, the teeth migrate rostrally in the sinuses as they erupt.[5] For example, the apex of the curved upper 11s (sixth maxillary cheek tooth) drifts rostrally from its site beneath the orbit in the young adult, to become sited rostral to the orbit in the aged horse. The intimate relation between the caudal cheek teeth and sinuses can allow periapical infections of the caudal cheek teeth to cause maxillary sinus empyema, as discussed in Chapter 11. The rostral maxillary teeth are embedded in maxillary bones, in young Thoroughbreds, the apices of the 08s often lying 2–3 cm rostral to the rostral aspect of the facial crest, the 07s lying 2–3 cm rostral to this site and the apices of the 06s lying a

further 2–3 cm rostrally. In many young horses eruption cysts occur at these sites during dental eruption, but due to the presence of the overlying levator nasolabialis and levator labii superioris muscles, they may not be detected. The pencil-like muscle body of the latter muscle overlies the infra-orbital foramen and must be pushed dorsally to perform a nerve block at this site.

The six maxillary and mandibular cheek teeth form slightly curved rows, with their concavity toward the buccal and lingual aspects respectively.[36] This curvature can, however, be marked in the maxillary cheek teeth of some horses and render dental rasping impossible unless a selection of angulated rasps is available. A common feature of all ungulates (mammals with hooves) including horses is the presence of an interdental space ('bars of mouth') between the incisors and the premolars, that may just be a side-effect of the evolutionary increase in face length (dolicephalic) to allow these long-legged animals to more comfortably graze off the ground.[49] Its presence, however, necessitates the rostral cheek teeth to face caudally (distally) to help compress each row of cheek teeth (Fig. 3.27). In contrast, the complete arch of teeth of omnivores and many carnivores needs to be compressed in just a single direction with the rostrally (mesially) facing caudal teeth to promote this compression of teeth.[49]

The reserve crowns (and the roots, once developed) of the 06s, both upper and lower, are angled slightly rostrally and therefore their clinical crowns are angled caudally. The 07s–09s are roughly perpendicular. The 10s and 11s have caudally facing reserve crowns with the 11s and to a lesser and variable extent the 10s, also having caudal curvatures of their reserve crowns and roots. Consequently, their clinical crowns are angled rostrally. Pressure between the rostral and caudal cheek teeth tightly compresses the six cheek teeth together at their occlusal surfaces. This causes each row of six cheek teeth to act as a single functional unit (Figs 3.27 and 3.28). Continued eruption of the angulated cheek teeth usually maintains this tight occlusal contact until very late in life in normal horses, despite the fact that equine teeth slightly taper in toward their apex and, with age, would otherwise develop a space or spaces between the teeth (interdentally-interproximally), which is termed a diastemata (Fig. 3.22).[50,51] Many very old horses (>20 years) do develop diastemata between their incisors, which is usually of little consequence in these teeth, unlike the situation with cheek teeth, where diastema can cause food to accumulate between the teeth and in the adjacent periodontal space, possibly leading to severe dental disease.[46]

The occlusal surfaces of the rows of cheek teeth are not level in the longitudinal plane as occurs in some other species, but instead, the surface of the caudal two or three cheek teeth curves upwards in the caudal direction, which is termed the 'curve of Spee'[6] (Fig. 3.27). This curvature is most marked in Arab type breeds that often have a similar curvature on their (dished) facial bones, but can also be marked in other breeds of horses, even in larger horses with convex faces (i.e. 'rams head' or 'Roman nose'). Some horses have an upward curvature of the rostral aspect of their cheek teeth rows, with the lower 06's becoming quite tall (dominant) and little clinical crown present on the upper 06s. If both a curve of Spee and a rostral curvature are present, this will give the lower occlusal surface a concave appearance in the rostrocaudal plane, i.e. raised at the 06s and the 11s.

In normal horses, the distance between the maxillary cheek teeth rows is wider (approximately 23 per cent) than that between the mandibular rows,[52] which is termed anisognathia. This is in comparison with, e.g. human upper and lower dental arcades, which are equally spaced (isognathic). As noted, the maxillary cheek teeth are also wider than their lower counterparts. Consequently, when the mouth is closed, approximately one-third of the occlusal surface of the upper cheek teeth is in contact with about half of the lower cheek teeth's occlusal surface. Additionally the occlusal surfaces of the cheek teeth are not level in the transverse (buccolingual) plane as is usually the case in brachydonts, but are angled at 10°–15° (angled from dorsal on their lingual (buccal) aspect to ventral on their buccal aspect) (Figs 3.16 and 3.17) with the degree of angulation present being largely determined by the direction and forces of mastication. For example, on a normal forage diet where horses have a wide range of lateral masticatory movement, the angle will be shallow (10°–15°). In contrast, on a diet high in concentrates, e.g. processed grains, or with painful dental disorder that causes pain on vigorous mastication, the masticatory angle will be more vertical and will lead to a higher degree of occlusal surface angulation, which is termed 'shear mouth' if severe.

The terminology concerning the irregularities present on the occlusal surface of the cheek teeth can be confusing. A cusp is a pronounced elevation on the occlusal surface of a cheek tooth and is an area with thicker enamel. A ridge (or style) is a linear elevation on the surface (peripheral or occlusal) of a tooth and on the occlusal surface may be formed by connections between cusps. Horses usually have about 12 such ridges running transversely across the occlusal surface of their cheek teeth that are commonly termed transverse ridges, two on the occlusal surface of each tooth, except the first and last which can contain one to three ridges. These can be quite tall, especially over the caudal cheek teeth in younger horses of certain breeds. Diet and age may also influence their size. These normal anatomical structures should not be confused with narrower acquired transverse overgrowths (usually just a lesser number) also termed 'transverse ridges,' due to, e.g. being opposite a wide diastema or some other area of reduced growth of the occlusal counterpart.

Because equine cusps contain sharp ridges of exposed occlusal enamel adjacent to hollows (craters) of dentin (and cementum at some sites) they are classified as lophs and thus the cusp pattern of equine teeth is termed lophodont. A fossa is a rounded depression and a fissure is a linear depression

between cusps or ridges.[1,53] The opposing ridges and fissures of the upper and lower equine cheek teeth interdigitate when the mouth is shut. Other variations in cusp number, size and distribution are used for palentological research and for taxonomic classification of different species.[53]

Nerve supply of teeth

Because of its great importance in human dentistry, the innervation of teeth has been well studied in brachydont teeth. Pulpar nerves enter through the apical foramen and include sensory nerves derived from the trigeminal (fifth cranial) nerve that are most extensive in the coronal region of the pulp where they form the plexus of Raschkow,[12] and sympathetic fibers from the cervical ganglion that supply the vascular smooth muscles to regulate blood flow in the pulp.[54,55] The latter are also believed to control the differentiation and function of odontoblasts, including their circadian rhythm of activity.[23]

The type and duration of pain caused by stimulation of dentin are different from those of pulp. In brachydont teeth, dentin responds to various stimuli including excessive heat and cold and to therapeutic procedures such as drilling, with a sharp pain which stops when these stimuli cease. In contrast, stimulation of the pulpal nerves produces dull pain, which continues for some time after the stimulus is removed.[23] Nerves are present in the pulp of hypsodont teeth, although the role of sensory nerves is unclear, as these teeth have dentin constantly exposed on the occlusal surface, a situation that in some circumstances would cause marked pain in brachydont teeth. Following significant dental overgrowth reductions, some horses will not masticate properly for days to weeks and this is empirically attributed to temporomandibular joint pain from prolonged opening of the mouth with a speculum during the procedure. However, the recent work showing exposed viable dentinal processes following dental rasping[26] makes it more likely that in fact pain from damaged dentin, or in some cases from pulp exposure, is the cause of such post-treatment pain.[56]

Blood supply of teeth

In brachydont teeth, the blood vessels enter pulp through the apical foramen and form an extensive capillary network, particularly in the coronal region of the pulp.[23] These capillaries drain into an extensive venous network which has a more tortuous course than the arterioles and also exits via the apical foramen.[23] Due to difficulties in microscopically distinguishing them from vascular capillaries, it remains unclear if lymph vessels are actually present in pulp.[23] However, other authors believe that pulp tissues, like all other connective tissues, contain lymph vessels that, in humans, drain into the submandibular and deep cervical lymph nodes. As previously noted, the good blood supply and wide apical foramina of even adult equine teeth can allow them to retain a blood supply following pulpar exposure and then allow them to seal off the exposed pulp. Although not directly involved in dental blood supply, the greater palatine artery can be iatrogenically damaged during dental procedures and awareness of its site and size is useful. It runs around the periphery of the hard palate and is not an end-artery as it adjoins its counterpart rostrally and thus receives a blood supply from both internal maxillary arteries. It can be damaged if a dental elevator slips medially when extracting wolf teeth, especially if they are medially displaced. It can also be damaged when orally extracting maxillary cheek teeth–which usually have short erupted crowns on their medial (buccal) aspect–if the extraction forceps are placed too low on the teeth.

Supporting bones and muscles of prehension and mastication

Alveolar bone

Alveolar bone is very flexible and constantly remodels to accommodate the changing shape and size of the dental structures it contains. Alveolar bone can be divided into two main parts; a thin layer of compact (radiodense) bone that lines the alveolus proper, and in which Sharpey's fibers insert, that is termed the lamina dura (lamina dura denta). This area is radiographically detectable as a thin radiodense line in brachydont teeth (Fig. 3.29) but due to irregularities of the periphery of some normal equine cheek teeth, this feature is not always obvious on lateral radiographs. The second part of alveolar bone surrounding the lamina cannot be morphologically differentiated from the remaining bone of the mandible or maxilla in adult brachydont teeth.[1] However, recent studies have shown that in horses, the alveolar bone beneath the lamina dura remains spongy and porous throughout life – similar to the alveolar bone of developing children's teeth – probably a reflection of its constant remodeling as the equine teeth constantly erupt.[2]

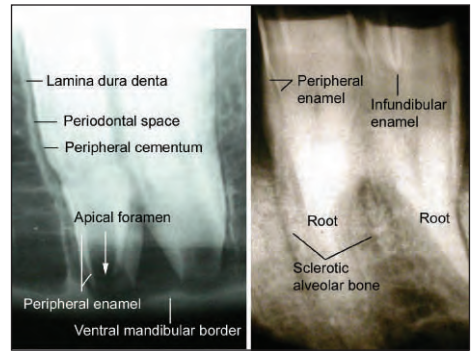


Figure 3.29. X-rays of the apical aspects of a young mandibular (left) and an aged maxillary cheek tooth (right). Note the wide periodontal space of the younger tooth that merges with soft tissue of the apical area and into the large apical foramina (of such younger teeth) and on into wide pulp cavities. The peripheral enamel folds reach to the apex of this tooth, i.e. it has no enamel-free apical area or true roots. In contrast, the older tooth has long true roots composed of cementum, with no radiographically obvious apical foramen or pulp cavities. The peripheral (and infundibular enamel) folds are positioned high up on this old tooth due to prolonged root cementum deposition, following cessation of enamel formation.

This presents an area of anatomical weakness, which may explain why sequestration of the alveolar lining occurs in some cases following oral extraction of cheek teeth. The most prominent aspect of the alveolar bone beneath the gingival margin is termed the alveolar crest (Figs 3.3 and 3.29).

Mandible

The mandible, the largest bone of the equine face, is composed of two components (hemimandibles) that fuse in the foal at 2–3 months of age.[35] It articulates with the squamous temporal bone at the temporomandibular joint. This bone contains the alveoli of the mandibular incisors and lower cheek teeth (and canines and PM1 – if present). The ventral border of the horizontal ramus is wide and rounded in the young horse because of the deep reserve crowns it contains (Figs 3.16 and 3.17) and, conversely, becomes thinner and sharper in older horses – a feature used to age horses in some Eastern countries. Some breeds, especially those that are descendants of the Arab horse (which in turn are descendants of *Equus cracoviensis* – Type IV horse) have shallow mandibles and maxillae and commensurately short reserve crowns, whereas most other breeds, e.g. derived from *E. muniensis* (Type I or mountain pony) or *E. mosbachensis* (Type III – forest or marshland horse) such as the North European draught and native British pony types (e.g. Exmoors) have deep alveoli and long reserve crowns[57] (AN Copeland, 1990, unpublished observations).

It has been proposed that crosses between these two types of horses can develop pronounced ventral swellings under the developing apices of the second and third cheek teeth,[57] due to an imbalance between mandibular depth and tooth length. These mandibular eruption cysts ('osseous tubercles') (Fig. 3.27) usually occur at 3–5 years of age and unless they fistulate, they regress over the following 1–2 years.[30] Other authors suggest that some breeds of horses are predisposed to retention of deciduous cheek teeth remnants ('caps'), which causes these mandibular swellings,[58] but clinical studies have not verified this.[59]

The mental nerve (from fifth cranial) enters the mandibular foramen on the medial aspect of the vertical ramus, level with the occlusal surface of the cheek teeth, and then continues rostroventrally along the mandibular canal. The mental nerve can be locally anesthetized at the mandibular foramen to facilitate painful dental procedures (e.g. oral extraction of a mandibular tooth) in the standing horse. The mandibular canal then passes to the ventral aspect of the mandible, below the apices of the teeth. However, in recently erupted teeth whose apices reach the ventral border of the mandible, the nerve usually lies on the medial aspect of the developing tooth (Fig. 3.16). The main part of the mental nerve emerges through the mental foramen on the rostrolateral aspect of the horizontal ramus, approximately halfway between the lower 06 and the incisors, while a smaller branch continues rostrally in a smaller canal along with the vasculature of the lower incisors. The nerve supply to the lower incisors can be anesthetized at this site.

Caudal to the alveoli of the sixth cheek teeth, the mandible becomes a very thin sheet of bone. This flattened area progressively increases in size with eruption of this tooth and contraction of its alveolus. At the angle of the jaw, this thin plate of bone expands medially and laterally into two wide lips that are roughened to allow muscle attachment (Fig. 3.27). These lips reduce in size toward the dorsal border of the vertical ramus. These normal roughened mandibular areas may be radiologically confused with pathological mandibular changes.

The temporomandibular joint and muscles of mastication

In contrast to carnivores, which have a vertical power stroke, horses have a transverse power stroke in a lingual (medial) direction that is termed a lingual power stroke. Consequently, the masseter and medial pterygoideus muscles are their most highly developed masticatory muscles and are innervated (like almost all of the muscles of mastication) by the mandibular branch of the fifth cranial nerve. The facial (seventh) nerve just innervates the superficial facial muscles (i.e. muscles of expression). The powerful masseter muscle originates along the full length of the facial crest and zygomatic arch and has wide insertions along the caudal lateral aspect of the mandible, with its deeper fibers running ventrocaudally and its more superficial fibers running almost vertically. Its elevated cranial border is caudal to the site where the facial artery, facial vein and parotid duct cross the ventral border of the mandible and ascend vertically. In horses, the temporomandibular joint is approximately 15 cm above the level of the occlusal surface, and thus the movement arm of the masseter is longer. The powerful pterygoideus (medialis and lateralis) muscles lie on the medial aspect of the mandible, and have similar attachments and orientation to the masseter and can move the jaw sideways almost continually with a strong power stroke.[5] In some horses the pterygoideus muscles are larger than the more obvious masseters. The relatively small digastricus muscle, which attaches from the occipital bone to the caudal aspect of the mandible, functions to open the mouth, a process that takes little mechanical effort, hence its small size.

The articular extremity of the mandible is composed of the condyle caudally and the coronoid process rostrally. The latter is poorly developed in the horse (Fig. 3.27) because it has smaller temporalis muscles (which close the jaw) in contrast to carnivores, where the power stroke of the jaws is vertical (to catch and crush prey) and consequently, both the temporalis muscle and coronoid process are larger. Between the articular surfaces of the mandible and the squamous temporal bone lies an articular disc that divides the joint cavity in two. The joint capsule is tight and reinforced by an indistinct lateral and an elastic posterior ligament.[35]

Although it allows just limited opening of the jaws, the equine temporomandibular joint has a wide range of lateral movements to permit the cheek teeth to effectively grind

food, utilizing a side-to-side movement that is combined with a slight rostrocaudal movement of the temporomandibular joint, with one side gliding rostrally and the other caudally. This rostrocaudal movement can vary greatly between horses and can be demonstrated in some sedated horses by gently pushing and pulling the mandible rostrocaudally. More clinically significantly, it can be demonstrated by closing the mouth and then elevating the head, which causes caudal movement of the mandible relative to the maxilla. Lowering the head causes the opposite (rostral) mandibular movement. This maneuver can cause a horse with mild overjet to have normal occlusion on dropping the head. Horses with large focal dental overgrowths may have restriction of their rostrocaudal mandibular movement – but due to individual variation between horses this parameter is difficult to quantify. Further details of the temporomandibular joint are presented in Chapter 4.

Maxillary bones

The upper jaws are largely formed by the maxillary bones that contain the alveoli of the upper cheek teeth (and PM1 and canines if present) and the distribution of these teeth in the maxillary bone and maxillary sinuses has been discussed earlier. In younger horses, this rostral maxillary area may become focally swollen because of the presence of the underlying eruption cysts of the 06s–08s. The overlying bone may even become thin and distended, with a temporary and focal loss of bone over the developing apices, but as noted, this may be masked by the presence of the overlying muscles. Some 3–4 year-old animals (mainly ponies) develop marked bilateral firm swellings of the rostral maxillary bones during eruption of these teeth, giving their face a box-like appearance. These are the equivalent of the mandibular eruption cysts ('osseous tubercles,' 3- and 4-year-old bumps) of this same age group.

The facial crest is a lateral protrusion of the maxilla that continues caudally as the zygomatic process and then joins the zygomatic parts of the malar and temporal bones to form the zygomatic arch (Fig. 3.27). After giving off a small branch that runs rostrally to innervate the maxillary incisor teeth, the infra-orbital nerve (sensory, fifth) emerges through the infraorbital foramen, approximately 5 cm dorsal to the rostral aspect of the facial crest. Its point of exit is covered by the pencil-like levator labii superiorus muscle, which can be dorsally displaced to allow local anesthesia of this nerve within the canal to anesthetize the upper 06 (possibly the 07) and the upper incisors.

The dorsal and caudal borders of the maxillary bone are attached to the nasal and lacrimal bones respectively, while rostrally the maxillary is attached to the incisive bone. The thicker ventral border of each maxillary bone contains the alveoli. Each cheek tooth alveolus is fully separated by transverse interalveolar bony septa. As noted, the equine maxillary sinuses are uniquely divided into rostral and caudal compartments by a thin, transversely angulated bony septum that can vary greatly in position. The medial aspect of each maxillary bone forms a horizontal shelf (the palatine process) that join midline with their opposite counterpart to form the supporting bone of most of the hard palate, the remainder of which is formed caudally by the palatine and rostrally by the incisive bones.

Incisive bone

The paired incisive (premaxillary) bones form the rostral aspect of the upper jaw. Their thick rostral aspects contain the alveoli of the incisors, while their thinner caudal aspects form the rostral aspect of the hard palate. The almost transverse suture line between the incisive bones and the maxillae is an anatomically weak area and a common site for fractures. The canine teeth (if present) are on the maxillary side of this suture.

Oral mucosa

The mucosa of the gingiva and hard palate is a specialized masticatory mucosa. It can be keratinized, orthokeratinized or parakeratinized and has deep interdigitating rete pegs into the underlying vascular subcutaneous connective tissue that limits its mobility. Most of the gingiva is attached to the supporting bone, with a more mobile (usually non-keratinized) area termed the free (marginal) gingiva close to tooth. Between the free gingiva and the tooth lies a depression termed the gingival sulcus that is lined by non-keratinized epithelium. In the deepest area of the gingival sulcus lies the junctional epithelium, which is attached to the peripheral cement of the tooth, with the periodontal ligament lying directly below this layer. In the horse with its prolonged dental eruption, this area is constantly remodeling and reforming new periodontal ligaments and new gingival–dental attachments. In other species interdental papillae of gingiva are present between teeth to prevent food trapping and subsequent periodontal disease, but as noted, most equine teeth are tightly compressed at the occlusual surface and have no interproximal spaces.

The salivary glands

The (paired) main equine salivary glands are the parotid, mandibular and sublingual glands whose ducts drain directly into the mouth. Minor salivary glandular tissue is also present in the lips, tongue, palate and buccal regions. The largest salivary gland is the parotid, which is approximately 20–25 cm long, 2–3 cm thick and weighs approximately 200 g in Thoroughbreds and can produce up to 50 ml/min of saliva. This gland lies behind the horizontal ramus of the mandible, ventral to the base of the ear, rostral to the wing of atlas, extending ventrally just caudal to the mandible to the tendon of origin of the sternomandibularis muscle and external maxillary vein[5,35] (Fig. 3.30). The lateral aspect of the parotid salivary gland is usually level with the masseter muscle (except for a small flat area of this gland which protrudes onto the surface of the masseter muscle (at the level of the lateral canthus) often covering some of the parotid lymph nodes. In some apparently normal horses, the parotid salivary gland may swell and protrude 1–3 cm

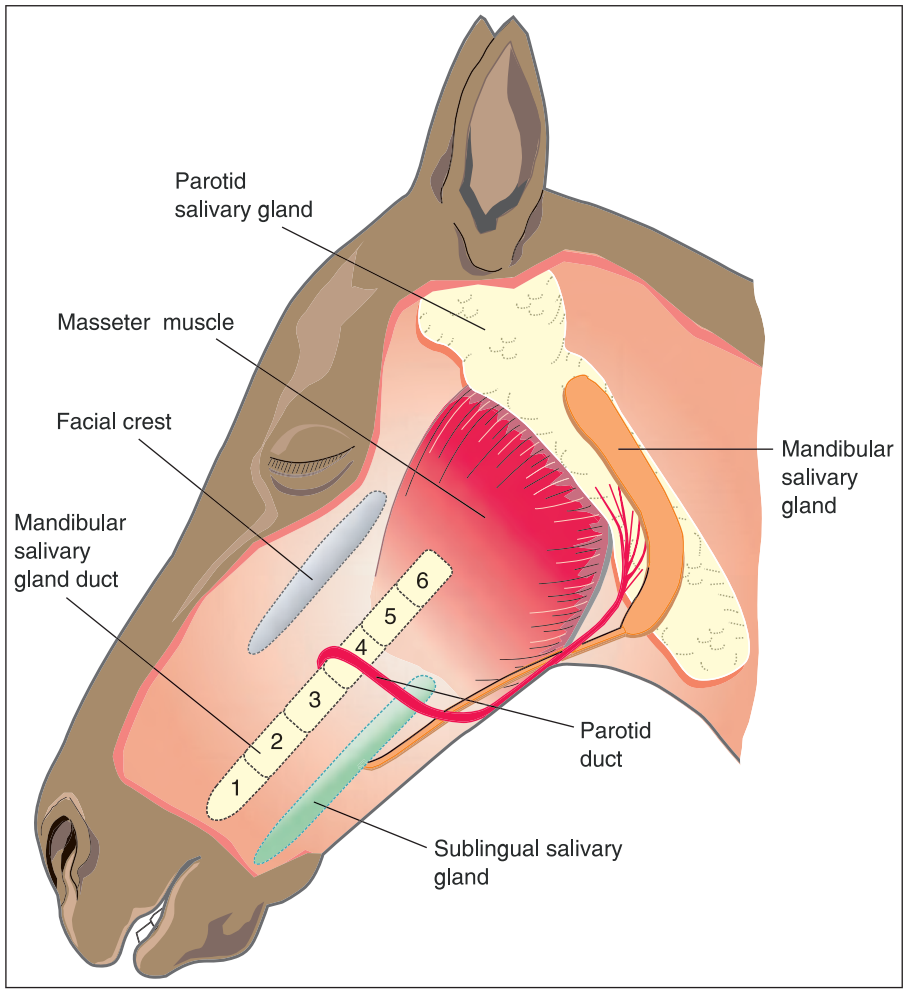


Figure 3.30. Diagram outlining the three (paired) equine salivary glands.

above this level when turned out to grass, in the ill-defined condition termed idiopathic parotitis (colloquially termed 'grass glands').

The jugular vein is often embedded in this gland and the medial aspect of the parotid salivary gland covers the stylohyoid bone, carotid artery, facial nerve, guttural pouch and the origins of the brachiocephalicus and sternocephalicus muscles – the latter separate the parotid from the mandibular salivary gland.[5] The dorsal aspect of the parotid salivary gland contains lymphatic tissue within its substance or lying beneath it, which can become focally distended following purulent infections of that region, especially strangles. The parotid duct originates from an amalgamation of three or four large ducts that converge in its rostroventral aspect, and this large duct then runs on the medial aspect of the pterygoideus muscles (and mandible), until it crosses the ventral aspect of the mandible behind the facial artery and vein. It then moves dorsally, moving rostral to the accompanying vasculature and perforates the cheek at the level of the upper 08s[35] (Fig. 3.30).

The smaller (20–25 cm long and 2–3 cm wide, approximately 50 g weight) mandibular gland lies deep to the mandible and parotid salivary gland and so is not palpable. It curves around beneath the parotid salivary gland and mandible, extending from the base of the atlas as far ventrally as the basihyoid bone. Its duct arises on its concave aspect and travels almost the full length of the oral cavity in the sublingual fold, beside the sublingual salivary gland rostrally, and enters into the oral cavity on the lateral aspect of the sublingual caruncle.[5] The long, thin sublingual salivary gland lies superficially in the floor of the mouth beneath the sublingual fold of the oral mucosa. It is situated between the tongue and mandible, extending from the mandibular symphysis to the lower 09 and drains through multiple small ducts into the oral cavity.[35]

REFERENCES

1. Berkovitz BKB and Moxham B (1981) Development of dentition: Early stages of tooth development. In: *Dental Anatomy and Embryology,* ed. JW Osborn, Blackwell Scientific Publications, Oxford, pp. 166–174.
2. Mitchell S, Kempson SM and Dixon PM (2003) Unpublished observations.
3. Butler P (1991) Dentition in function. In: *Dental Anatomy and Embryology,* ed. JW Osborn, Blackwell Scientific Publications, Oxford, p. 345.

4. Baker GJ (1985) Oral examination and diagnosis: management of oral diseases. In: *Veterinary Dentistry,* ed. CE Harvey, WB Saunders, Philadelphia, pp. 217–228.
5. Dyce KM, Sack WO and Wensing CJG (1987) *Textbook of Veterinary Anatomy*. WB Saunders, Philadelphia, pp. 473–477.
6. Easley J (1996) Equine dental development and anatomy. In: *In-Depth Dentistry Seminar. Proceedings of the American Association of Equine Practitioners,* 42, 1–10.
7. DeLahunta A and Habel RE (1986) *Applied Veterinary Anatomy*. WB Saunders, Philadelphia, pp. 4–16.
8. Miles AEW and Grigson C (1990) *Colyer's Variations and Diseases of the Teeth of Animals* (rev. edn). Cambridge University Press, Cambridge, pp. 2–15, 482.
9. Kirkland KD, Baker GJ, Marretta SM, Eurell JAC and Losonsky JM (1996) Effect of ageing on the endodontic system, reserve crown, and roots of equine mandibular cheek teeth. *American Journal of Veterinary Research*, 57, 31–38.
10. Warshawsky H (1983) The teeth. In: *Histology* (5th edn), ed. L Weiss, The Macmillan Press, New York, pp. 609–655.
11. Fortelius M (1985) Ungulate cheek teeth: developmental, functional and evolutionary interrelations. *Acta Zoologica Fennica,* 180, 1–76.
12. Ten Cate AR (1994) Development of the tooth and its supporting tissues; hard tissue formation and its destruction; dentinogenesis. In: *Oral Histology* (4th edn), ed. AR Ten Cate, CV Mosby, St Louis, pp. 58–80, 111–119, 147–168.
13. Kollar EJ and Lumsden AGS (1979) Tooth morphology: The role of innervation during induction and pattern formation. *The Journal of Biological Buccale*, 7, 49–60.
14. Brescia NJ (1966) Development and growth of the teeth. In: *Orban's Oral Histology and Embryology* (6th edn), ed. H Sicker, CV Mosby, London, pp. 18–37.
15. Latshaw WK (1987) Face, mouth and pharynx. In: *Veterinary Developmental Anatomy – A Clinically Oriented Approach,* ed. WK Latshaw, BC Decker, Toronto.
16. Ferguson M (1990) The dentition throughout life. In: *The Dentition and Dental Care,* Vol. 3, ed. RJ Elderton, Oxford Heinemann Medical Books, Oxford, pp. 1–18.
17. Suga SJ (1979) Comparative histology of progressive mineralisation patterns of developing incisor enamel of rodents. *The Journal of Dental Research*, 58, 1025–1026.
18. Eisenmann DR (1994) Amelogenesis; enamel structure. In: *Oral Histology* (4th edn), ed. AR Ten Cate, CV Mosby, St Louis, pp. 218–256.
19. Baker GJ (1979) A study of dental disease in the horse. PhD Thesis, Glasgow University, pp. 3–96.
20. Kilic S, Dixon PM, and Kempson SA (1997) A light and ultrastructural examination of calcified dental tissues of horses: 11. Ultrastructural enamel findings. *Equine Veterinary Journal*, 29, 198–205.
21. Shellis P (1981) Dental tissue. In: *Dental Anatomy and Embryology,* ed. JW Osborn, Blackwell Scientific Publications, Oxford, pp. 193–209.
22. Stanley HR, White CL and McCray L (1966) The rate of tertiary (reparitive) dentin formation in the human tooth. *Oral Surgery, Oral Medicine and Oral Pathology*, 21, 180–189.
23. Jones SJ (1990) The pulp-dentin complex. In: *The Dentition and Dental Care,* Vol. 3, ed. RJ Elderton, Oxford Heinemann Medical Books, Oxford, pp. 1–18.
24. Fawcett DW (1987) *A Textbook of Histology.* WB Saunders, Philadelphia, pp. 603–618.
25. Kilic S, Dixon PM and Kempson SA (1997) A light and ultrastructural examination of calcified dental tissues of horses: III. Dentin. *Equine Veterinary Journal*, 29, pp. 206–212.
26. Kempson SA, Davidson M and Dacre IT (2003) The effects of three types of rasps on the occlusal surface of equine cheek teeth: A scanning electron microscopic study. *Journal of Veterinary Dentistry,* 20, 19–27.
27. Kilic S, Dixon PM and Kempson SA (1997) A light and ultrastructural examination of calcified dental tissues of horses: I. The occlusal surface and enamel thickness. *Equine Veterinary Journal*, 29, 191–197.
28. Holland GR (1994) Morphological features of dentin and pulp related to dentin sensitivity. *Archives of Oral Biology,* 39 (suppl), 3–11.
29. Dacre I (2004) A pathological study of equine dental disorders. PhD Thesis, The University of Edinburgh.
30. Dixon PM and Copeland AN (1993) The radiological appearance of mandibular cheek teeth in ponies of different ages. *Equine Veterinary Education*, 5, 317–323.
31. Kilic S, Dixon PM and Kempson SA (1997) A light and ultrastructural examination of calcified dental tissues of horses: IV. Cement and the amelocemental junction. *Equine Veterinary Journal*, 29, 213–219.
32. Jones SJ (1981) Human tissue: Cement. In: *Dental Anatomy and Embryology,* ed. JW Osborn, Blackwell Scientific Publications, Oxford, pp. 193–209.
33. Capper SR (1992) The effects of feed types on ingestive behaviour in different horse types. BSc Thesis, University of Edinburgh.
34. Myers JS (1994) The effects of body size, grass height and time of day on the foraging behaviour of horses. MSc Thesis, Aberystwyth University, p. 56.
35. Sisson S and Grossman JD (1953) Splanchnology. In: *The Anatomy of the Domestic Animals* (4th edn), WB Saunders, Philadelphia, pp. 406–407.
36. Walmsley JP (1993) Some observations on the value of ageing 5–7 year-old horses by examination of their incisor teeth. *Equine Veterinary Education*, 5, 195–298.
37. Richardson JD, Lane JG and Waldron KR (1994) Is dentition an accurate indication of the age of a horse? *Veterinary Record*, 137, 88–90.
38. St Clair LE (1975) Teeth. In: *Sisson and Grossman's the Anatomy of the Domestic Animals* Vol. 1 (5th edn), ed. R Getty, WB Saunders, Philadelphia, pp. 460–470.
39. Floyd MR (1991) The modified Triadan system nomenclature for veterinary dentistry. *Journal of Veterinary Dentistry,* 8, 18–20.
40. Goody PC (1983) *Horse Anatomy*. JA Allen, London, pp. 38–41.
41. Eisenmenger E and Zetner K (1985) *Veterinary Dentistry*. Lea and Febiger, Philadelphia, pp. 55–57, 153–157.
42. Mueller POE (1991) Equine dental disorders: cause, diagnosis, and treatment. *The Compendium of Continuing Education*, 13, 1451–1460.
43. Colyer JF (1906) Variations and diseases of teeth of horses. *Transactions of the Odontological Society of Great Britain*, New Series, 38, 47–74.
44. Wafa NSY (1988) A study of dental disease in the horse. MVM Thesis, University College Dublin, pp. 1–203.
45. Huidekoper RS (1891) *Age of Domestic Animals.* FA Davis, Philadelphia and London, pp. 33–35.
46. Bradley OC (1923) *The Topographical Anatomy of the Head and Neck of the Horse.* W Green and Son, Edinburgh, pp. 85–92.
47. Perkins J (2001) Quantitative and qualitative anatomy of the equine maxillary sinuses, with particular reference to the naso-maxillary aperture. Thesis, The University of Edinburgh.
48. Tremaine WH and Dixon PM (2001) Equine sinonasal disorders: a long term study of 277 cases. Part I – Historical, clinical and ancillary diagnostic findings. *Equine Veterinary Journal,* 33, 274–282.
49. Osborn H (1966) Cement. In: *Orban's Oral Histology and Embryology* (6th edn), ed. H Sicker, CV Mosby, London, pp. 155–212.
50. Dixon PM (1997) Dental extraction in horses: Indications and preoperative evaluation. *Compendium of Continuing Education for the Equine Practitioner*, 19, 366–375.
51. Dixon PM, Tremaine WH, McGorum BC, Railton DI, Hawe C and McCann J (2004) Equine dental disease – a long term study of

400 cases: Part II – Disorders of development, eruption and variations in position of the cheek teeth. *Equine Veterinary Journal,* in press.
52. Taylor AC (2001) An investigation of mandibular width and related dental disorders in the equine oral cavity. Thesis, Coventry University.
53. Kilic S (1995) A light and electron microscopic study of the calcified dental tissues in normal horses. PhD Thesis, University of Edinburgh.
54. Hayward AF (1981) Human tissue: Pulp. In: *Dental Anatomy and Embryology,* ed. JW Osborn, Blackwell Scientific Publications, Oxford, pp. 187–190.
55. Torneck CD (1994) Dentin–pulp complex. In: *Oral Histology* (4th edn), ed. AR Ten Cate, CV Mosby, St Louis, pp. 169–213.
56. Dixon PM (2002) The gross, histological and ultrasturctural anatomy of equine teeth and their realationship to disease. *Proceedings of the American Association of Equine Practitioners,* 49, 421–437.
57. Speed JG (1951) Horses and their teeth. *The Journal of the Royal Army Veterinary Corp*, 22, 136–141.
58. Barrairon P, Blin PC and Moliner F (1980) Contribution a l'etude du mecanisme de formation de fistule des premolaires chez le jeune cheval. *Bulletin de l'Academie Veterinaire de France,* 53, 47–54.
59. Dixon PM, Tremaine WH, McGorum BC, Railton DI and Hawe C (2004) Equine dental disease – a long term study of 400 cases: Part IV – Apical infections of the cheek teeth. *Equine Veterinary Journal,* in press.

4 Dental Physiology

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Introduction

Following the Chapter 3 description of the structure of the teeth of the horse, this chapter describes how the dental system functions to prepare ingesta for digestion. It has been said that dental function in mammals is still poorly understood and that, from a functional point of view, the teeth of herbivores have had limited study.[1] There has, however, been a great expansion of scientific interest in all aspects of mastication (chewing) in mammals since the pioneer studies in which serious examinations of the occlusal relationship during chewing between the upper and lower teeth were made.[2] Subsequently, other studies have been concerned with the patterns of jaw movements. It has been noted that these patterns are a reflection of the complex relationship between muscle activity, the forces exerted on food, and the relationship between food consistency, particle size and the nervous control of mastication.[3] In comparison with the highly complex appendicular skeleton and its movement, the skeletal components of mastication are simple and consist of just a few joints with muscles.

Functional morphology

The Eocene ancestor of the family Equidae (Hyracotherium or Eohippus) was a small (26-inch), three-toed animal with a series of low-crowned (brachydont) cheek teeth, made up of four premolars and three molars. All of the teeth had simple crown patterns and chewing mainly depended on the three molar teeth. Subsequent changes in the environment during the later Miocene period produced a rapid change in the Eocene teeth. These favorable mutations were a modification of the crown pattern, an increase in the height of the crown (i.e. reserve crown development) and the development of cement. What once had been two open valleys on the teeth had now become deep, closed pits, and numerous wrinkles and spurs appeared on the side of the main crest. These changes were similar in both upper and lower jaws, but the changes were less extreme in the lower teeth.[4]

The process whereby the premolar teeth became anatomically similar to the molar teeth is described as molarization. The formation of high-crowned cheek teeth of the Merychippus in the late Miocene period was accompanied by the development of cement. This substance appeared on the outside of the enamel crown and developed to fill all its valleys, and in so doing protected the brittle enamel from cracking. Cement is also formed within the enamel infoldings of the maxillary cheek teeth as the teeth develop within the dental sac. It is interesting to note that the blood supply to the tissue within the enamel infoldings (infundibula or enamel lakes) enters from what will become the occlusal surface. Consequently, when the maxillary cheek teeth erupt, the blood supply is broken and there is no more cementogenesis. The track left by the vessels persists as a small, black hole seen on the occlusal surface within the enamel lakes (in some teeth the remnants of two vascular channels may exist). Cement is also produced around the developing roots as they are formed. In this way, a permanently erupting cheek tooth is formed with a crown height (including reserve height) of at least twice its width. Such a tooth erupts at a rate equal to the rate of the wear of the crown by attrition.

It is known that as animals get bigger there is a cubic-factor relationship between size and food requirement between animals of different sizes. A doubling of height requires eight times the food intake. It is logical to suggest that as the equid species doubled in size, and almost tripled its height, that the dental changes are a result of favorable adaptions to accommodate the requirement for increased food intake. Under free-range conditions modern-day equids spend up to 14 hours a day feeding. The complete arcade of cheek teeth in Eohippus was no more than 10 cm in mesial–distal length – essentially the same as the mesial–distal length of two cheek teeth of *Equus caballus*.

From the viewpoint of dental function, diseases and treatment, we can view the head of the horse as the body of the food processor; it houses the teeth – the blades of the food-processing machine. Molarization of the cheek teeth of the horse, and most herbivores, forms a continuous row of teeth that function as a single unit. The lips, cheeks, palate, muscles, temporomandibular joints and bones maintain the position of the teeth, and the tongue. The lips and salivary glands facilitate the process of food prehension and mastication. The muscles of mastication are all supplied by the fifth cranial nerve. They are primarily the paired lateral muscles from the maxilla and cranium to the mandible; these are the masseter and temporalis muscles that close the jaw and pull

to the acting side. The medial pterygoideus muscles also close the jaw.

In comparison to the large bulk of jaw closure muscles, the bulk of the mandible depressor muscles is small. Jaw opening results from the contraction of the anterior belly of digastricus combined with the contraction of geniohydeus, and the inferior fibers of genioglossus coupled with the sternohyoideus and omohyoideus. In many mammals, particularly carnivores, jaw opening is aided by elevation of the head. This component is not, however, as important in most herbivores.

The disproportionate muscle mass between the jaw elevators and depressors is easily understood when the nature of jaw movements in feeding is studied. The jaws close against resistance, whereas opening is a free movement synergized by gravity. Food breakdown is accomplished during jaw closure and requires forces that exceed the requirements of simply elevating the mass of the mandibular structures. Jaw muscles have faster contraction rates than most other striated muscles, with a total cycle contraction rate that ranges from 333–500 cycles/min in the pygmy goat.[5]

The muscles of the cheeks and lips are supplied by the seventh cranial nerve and consist of the levator and depressor labii maxillaris and mandibularis, the orbicularis oris, the incisivus mandibularis and maxillaris, the buccinator and zygomaticus muscles. These muscles control the functions of lip closure, elevation, retraction and depression as well as the flattening of the cheeks.

The arrangement of the teeth within the upper and lower arcades is such that the curve of the upper dental arcade is not fully accommodated by the conformation of the lower arcade, i.e. the lower arcade is straighter and the distance between the left and right arcades is less in the mandible than in the upper jaw (anisognathism). At the same time the cheeks of the horse are, compared with other species, relatively tight fitting. The temporomandibular joints (TMJ) are large and each has a thick intra-articular disk. The TMJ are synovial joints formed by the condylar processes of the mandibles and the articular tubercles of the temporal bones. Joint integrity is maintained by a tight capsule that is reinforced by lateral and caudal ligaments. The caudal ligaments have large discal attachments. Recent studies documenting the use of radiography, ultrasonography, and computer tomography have described TMJ morphology.[6] The sizes and shapes of the osseous components of the TMJ are modified proportionally with the size of the head. There is, however, a consistency about the angles of these components. The mandibular condyles angle at 15° in two planes – from laterodorsal to ventromedial and from mediocaudal to laterorostral (Fig. 4.1A, B). The temporal facets are mirror images of the mandibular condyles.

In dissections and measurements from prepared specimens of skulls, mandibles and teeth, it has been shown that the 15° angulation of the TMJ components in both planes is reflected in the angles of the cheek teeth occlusal surfaces in both a vertical and transverse plane and also in the angles of the palatine ridges.[7] In the horse, compared with many other herbivores, for example the camel, lateral excursion of the mandibles is limited. A recent study has documented the three-dimensional kinematics of the equine TMJ.[8]

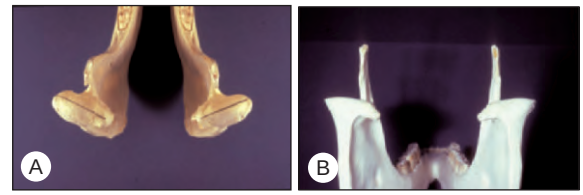


Figure 4.1. (A) Mandibular condyles, dorsal view – 15° angles. (B) Mandibular condyles, caudal view – 15° angles.

Examination of the occlusal surfaces of the cheek teeth of horses reveals a complete system of folded enamel (lophs). The arrangement of lophs in the maxillary arcade is matched (mirrored) by their occlusal counterparts in the mandibular arcade (Fig. 4.2). It is interesting to note that in a study on food processing and digestibility in horses, measurements were made of total cheek teeth enamel ridge perimeter and it was found that there was only 7 cm more in the total enamel perimeter measurement in a 1000 kg horse when compared with a 350 kg pony.[9]

The design, angles of attack and composition of horse tooth structure are ideally suited, when aided by the process of mastication, to produce shear forces on food substances that fracture stalks, leaves and grains. How these forces are created and applied may be understood from analyses of jaw movements. Using retroreflective spherical markers attached to the skull and mandible a motion analysis system recorded the location of the markers as the horses chewed hay and pellets. The data were processed and the three-dimensional displacement and rotations, described as pitch roll and yaw of the mandible relative to the skull were computed. A virtual tracking marker was created on the mandible and tracked relative to the skull. The mandible was found to have a significantly greater range of motion while chewing hay versus pellets. There was no significant difference in mandibular velocity between hay and pellet chewing.[8]

Mastication – the chewing cycle

Chewing is based upon the repetition of a cyclical movement that results from controlled rhythmic contraction of all the muscle groups associated with the opening (depression) and closing (elevation) of the jaws. In studies of the chewing cycle in many species it was found that, with the exception of man, mammals have consistent chewing patterns–consistent both individually and specifically within species. There is, however, no 'standard pattern.' What happens to the food and how it is broken down depends on the form of the cheek teeth. The literature may be confusing because of

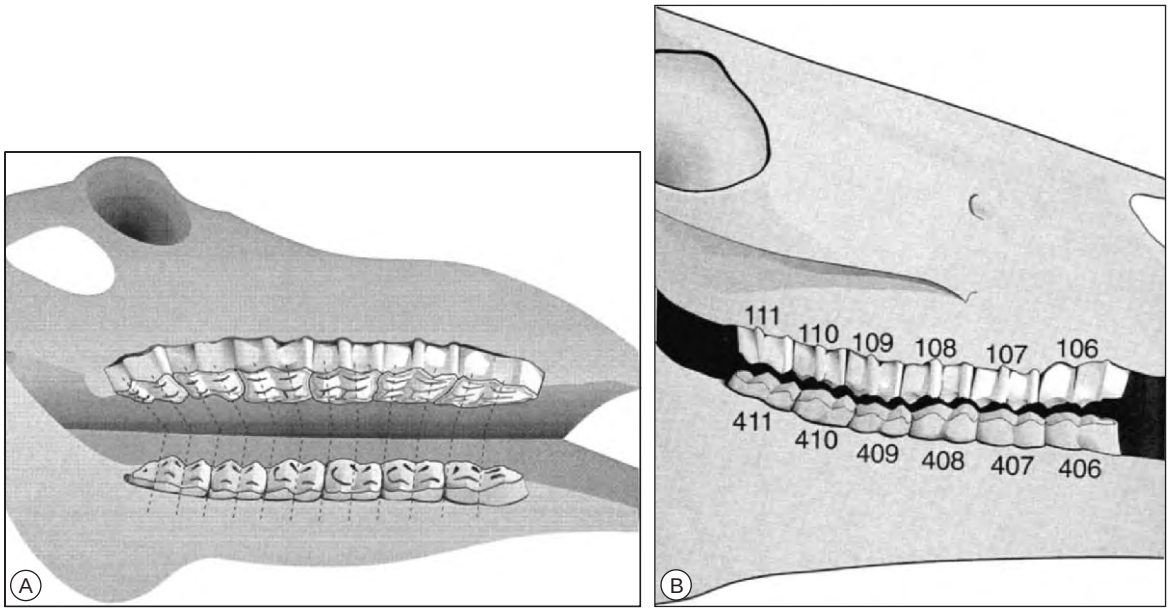


Figure 4.2. (A) Occlusal surfaces of maxillary and mandibular cheek teeth with architectural points documented. (B) Lateral view of occlusal waves.

the absence of a standard start point of the chewing cycle. In this description of the chewing cycle of the horse, the start point will be assigned to incisor contact. Other conventions use maximum incisor gap as the start point.

Recent video analysis of horses feeding and observations of masticatory movements have yielded interesting data. The masticatory movements of horses fit the general masticatory cycle of herbivorous mammals. They have been described as having a masticatory cycle of three phases: the opening stroke (O), the closing stroke (C) and the power stroke (P). It is the relative displacement of the mandible that defines the phases.[10] Figure 4.3 represents isolated frames from a video recording of equine mastication. Figure 4.4 shows the positional changes of the mandible. Note that frames 1–4 represent the opening stroke, frames 5–6 represent the closing stroke and frames 7–10 represent the power stroke. It may be observed that this analysis of the horse suggests in fact a fourth stroke – a post power 'recovery' stroke.

It has been observed for some time that horses appear to be either right-sided or left-sided chewers. This observation is not strictly accurate even though some horses consistently demonstrate major lateral mandibular excursion to one side only. The masticatory movements of 400 horses in a veterinary medical teaching hospital with a variety of feeds including fresh grass, grass hay, alfalfa hay, oats and various sweet feeds and proprietary horse diets were documented. The observations were made on a wide range of horse ages,

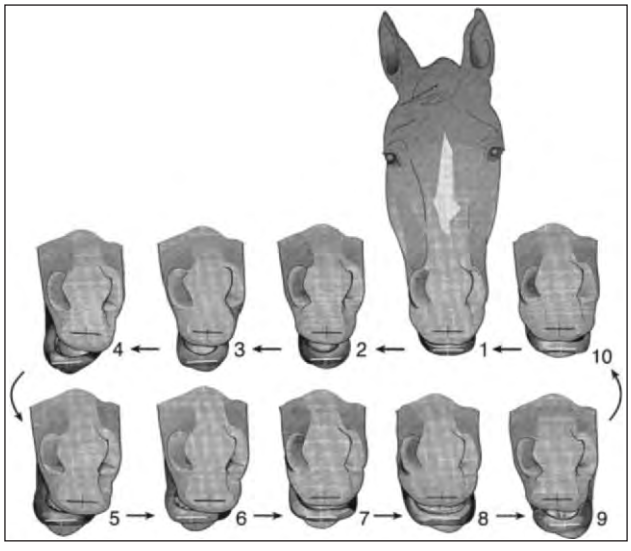


Figure 4.3. Reproduction from video recordings of chewing cycles of the horse (note in this reproduction, 'frame' relates to individually frozen time sections).

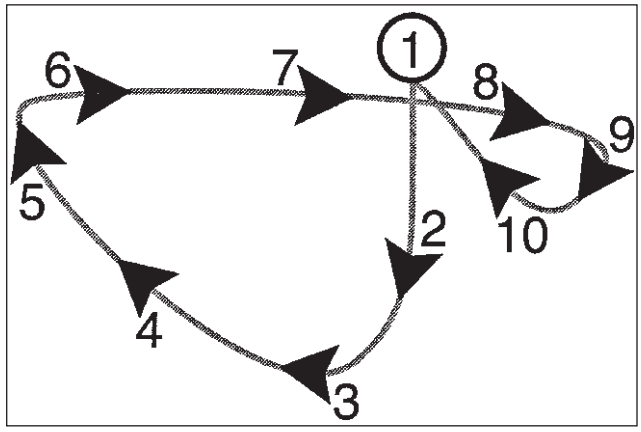


Figure 4.4. Frontal view of mandible during the chewing cycle – compare with Figs 4.3 and 4.5.

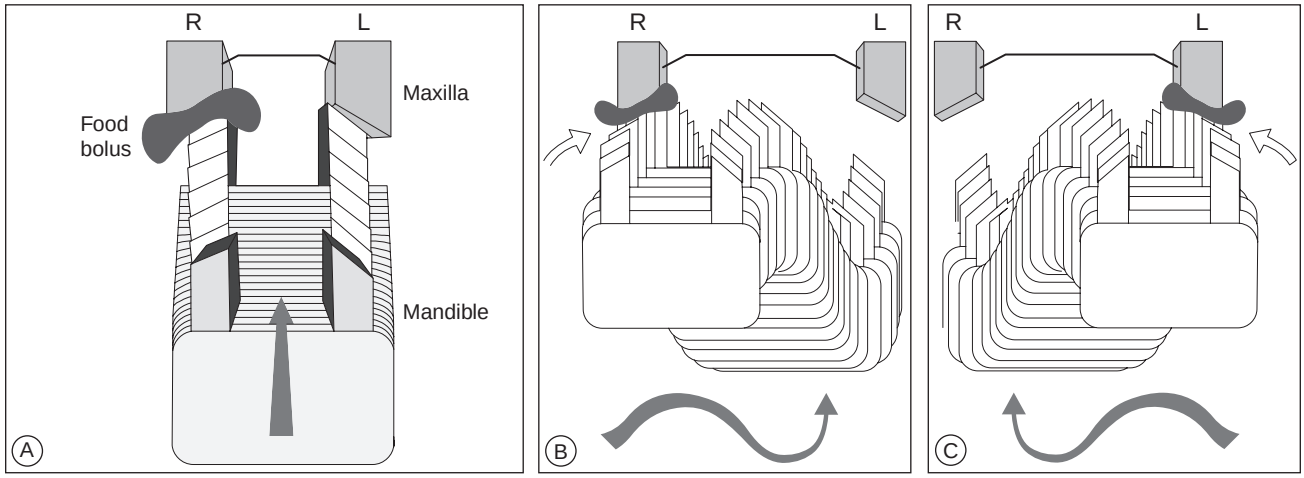


Figure 4.5. Representation of the power stroke component of the chewing cycle (from Tremaine,[12] 1997, with permission of the editor of *In Practice*).

breeds and a considerable range of medical and surgical disorders. Incomplete observations were made in 63 horses (16 percent), 45 horses (11 percent) were seen to chew both to the left and to the right, 163 horses (41 percent) moved the mandible to the right (i.e. clockwise as viewed from the front) and 131 (32 percent) chewed counterclockwise (i.e. mandibular movement to the left).

It has been suggested that during the power stroke there is only contact with one side of the arcade at a time.[1,11] However, the measured extent of anisognathism negates this observation, i.e. there has to be some contact with both sides. It is, however, true that major pressure is first applied to one side and then, as the surfaces slide across each other, is transferred to the other side (frames 5–10; Fig. 4.5). It might be concluded from these observations that there is a tendency for unequal attrition as a result of the variation in masticatory physiology. However, this cannot be confirmed by examination of the wear of occlusal surfaces at necropsy.

When eating, the horse uses its upper and lower lips as instruments to select, 'test' and pull food into the mouth between the incisor teeth. Short sliding strokes of the incisors cut or grasp the food material. This process continues until the rostral portion of the mouth is filled with food material and cheek-tooth grinding of food is initiated. Just as the food-processor analogy of mastication is valid, the 'auger' analogy may be applied to the process of the movement of food across the mouth from each arcade and distally within the oral cavity. The tight-fitting cheeks contain the feed material within the intradental oral cavity (IDOC).

The loph basins of the cheek teeth (the food channels across the occlusal surfaces) direct food as it is crushed into the IDOC. Collinson's study of molar function in horses describes how cheek teeth occlusal shape facilitates this process,[9] the canal of the entoflecid and the tray of the metaconid of the mandibular teeth direct food into the parastyle of the maxillary teeth (the terms entoflecid and metaconid refer to the specific slopes of the occlusal surfaces) (Fig. 4.6). Subsequent masticatory cycles crush and move food into the IDOC. The auger analogy is continued by examination of the morphology of the palatine ridges. There are a total of 18 pairs of palatine ridges. Each ridge is curved from lateral to midline and is incomplete and off-set in the midline. Food material is squashed into the IDOC and pressed against the palatine ridges by the tongue. The rotatory action of mastication and tongue and cheek compression moves the food caudally in a spiral fashion (Fig. 4.6).

As boluses of food collect in the oropharynx, pharyngeal constriction, elevation of the soft palate, epiglottic retraction

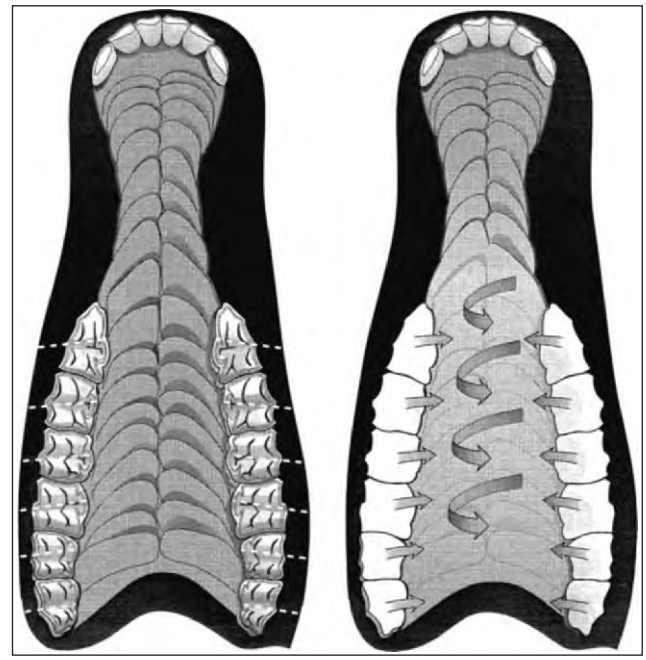


Figure 4.6. Representation of food 'channels' across the occlusal surface of maxillary cheek teeth and their relationship to palatine ridges.

Table 4.1. Mean (±SD) Values for Mastication Parameters for the Four Horses Used in the Food-processing Experiment

Mastication parameter	Fiber diet		
	Low	Medium	High
g/'mouthful'	12.1 ± 2.1	9.5 ± 2.4	8.1 ± 1.3
Chew rate/10 s	11.6 ± 0.6	11.5 ± 0.2	11.4 ± 0.2
Energy/g/chew	$9.4 \pm 4.8 \times 10^{-3}$	$8.9 \pm 6.4 \times 10^{-3}$	$1.4 \pm 1.4 \times 10^{-2}$
Duration of grind (s)	0.51 ± 0.08	0.53 ± 0.03	0.55 ± 0.02
Incisor displacement (cm)	4.4 ± 0.6	4.5 ± 0.6	4.4 ± 0.3
Premolar 4 velocity (cm/s)	10.4 ± 1.8	9.7 ± 2.4	8.5 ± 0.4

Courtesy Collinson (1994),[9] with permission.

and laryngeal contraction result in movement of food through the lateral food channels and around the larynx into the esophagus and this constitutes swallowing.

The rotatory auger analysis concept has been confirmed by observations of food-bolus shape in toothless horses. Horses without teeth can survive on special feeds—crushed and soaked feeds, for example. If, however, they try and feed on grass or hays they produce long, spiral boluses of a rope-like consistency that may lead to esophageal obstruction.

Factors that influence masticatory movements include fiber and moisture content of the diet. Chew rates have been calculated from electromyographic data (Table 4.1).[10] It was noted that horses were capable of attaining higher than 11 per second chew rates, particularly at the onset of feeding. The data in Table 4.1 were recorded over a 10 minute period. This study, however, did not confirm earlier observations that higher fiber content and lower moisture content reduces the extent of excursion of the mandible.[13]

The video analysis studies reported here did, in fact, confirm the observation that food type influences chewing pattern. The molar teeth initiate triturations ('grinding to powder') with a series of chopping actions, similar to the puncture/crushing portions of the power strokes in other species. Once the IDOC has filled with a critical mass, a 'mouthful', further lip and incisor function is limited and true arcade 'sliding' takes over, resulting in complete breakdown of food materials. Comment has been made concerning the relatively small amount of lateral excursion afforded by the shape and tight capsule and ligament structure of the equine TMJ. The degree of lateral excursion was documented in an innovative study that produced 'molographs' of the chewing pattern (the extent of lateral excursion) in horses eating different feeds.[13] The three-dimensional kinematic study also confirmed this observation.[8] It can be seen that there is greater lateral excursion with lush feeds – the drier the feed the less the lateral excursion.

Observations also suggest that there is rostral movement of one side at a time of the lower jaw during the masticatory cycle. This is achieved by the configuration of the TMJ and the contraction and relaxation of the masseter and medial pterygoid muscles. These movements result in oblique contacts with the upper and lower dental arcades.

Any changes in the architecture of the arcades or pathology of the TMJ dramatically change the efficiency of the food processing equipment of the horse. In many cases there is a chicken-and-egg relationship between oral or TMJ pathology and changes in dental architecture, i.e., it is sometimes difficult to see in specimen and case analysis which came first (Fig. 4.7).

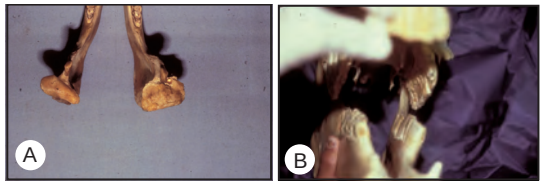


Figure 4.7. (A) TMJ osteoarthritis from shear-mouthed horse. (B) Shear-mouthed dentition. (Courtesy Dr Jack Easley. Reproduced with permission of editors *AAEP Proceedings* 2002.)

Summary

Horses have an elegantly designed and functioning masticatory apparatus that prepares feed materials for digestion. The hypsodont tooth structure and continuous eruption of reserve crown matches tooth loss from attrition caused by occlusal contact. The chewing cycle is a repetitive pattern of mandibular movement with three components – an opening stroke, a closing stroke and the power stroke. During the power stroke the exposed enamel components of the dental arcades crush and fragment feed materials. Caudal movement of feed material within the IDOC is facilitated by the valley of the cheek teeth, by the contour and angles of the palatine ridges and by the lifting and pressing actions of the tongue (Fig. 4.5).

After trituration, food is moved as an augered spiral and forms a caudal oropharyngeal bolus that is swallowed.

REFERENCES

1. Fortelius M (1982) Ecological aspects of dental functional morphology in the Pleistocene rhinoceroses of Europe. In: *Teeth, Form, Function and Evolution,* ed. B Kirsten, Columbia University Press, New York, pp. 163–181.
2. Butler PM (1952) The milk-molars of Perissodactyla with nematodes on molar occlusion. *Proceedings of the Zoological Society of London,* 121, 777–817.
3. Hiiemae KM (1978) Mammalian mastication: a review of the activity of the jaw muscles and the movements they produce in chewing. In: *Development, Function and Evolution of Teeth,* eds. PM Butler and KA Joysey, Academic Press, London, pp. 359–398.
4. Simpson GG (1951) *Horses.* Oxford University Press, Oxford, pp. 106–108.
5. Gans C and DeVree F (1974) Correlation of accelerometers with electromyograph in the mastication of pygmy goats (Capra hircus). *Anatomical Record,* 306 (Abst), 1342–1343.
6. Weller R, Taylor S, Maierl J *et al.* (1999) Ultrasonographic anatomy of the equine temporomandibular joints. *Equine Veterinary Journal,* 31, 529–532.
7. Baker GJ (2002) Equine temporomandibular joints (TMJ): Morphology, function and clinical disease. *Proceedings AAEP,* 48, 442–447.
8. Bonin SJ (2001) Three dimensional kinematics of the equine temporalmandibular joint. MS Thesis, Michigan State University.
9. Collinson M (1994) Food processing and digestibility in horses (Equus caballus). BSc Dissertation, Monash University, pp. 36–42.
10. Weijs WA and Dantuma R (1975) Electromyography and mechanics of mastication in the albino rat. *Journal of Morphology,* 146, 1–34.
11. Fortelius M (1985) Ungulate cheek teeth: developmental, functional and evolutionary interrelations. *Acta Zoologia Fennica (Helsinki),* 180, 78.
12. Tremaine H (1997) Dental care in horses. In *Practice Journal of Veterinary Postgraduate Clinical Study,* 19, 186–199.
13. Leue G (1941) Beziehungen zwischen Zahnanomalien und Verdauungsstörungen beim Pferde unter Heranziehung von Kaubildern. Veterinary Medicine Dissertation, Hanover, pp. 170–174.

5 Aging

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Introduction

The age of a horse can be an important consideration when forecasting its useful working life, when purchasing the animal, for insurance policies and for the prognosis of diseases. Furthermore, as long as no indelible identification methods for horses are imposed, age estimation contributes to the identification of an animal.

Why does the horse, of all animals, have teeth that lend themselves to age determination? Most domestic animals (cattle, carnivores etc.) have brachydont incisor teeth, i.e. low-crowned teeth that erupt fully prior to maturity and that are strong enough to survive for the life of the animal. Equine teeth, which are subjected to much higher levels of dietary abrasive forces, are hypsodont, which means that they erupt continuously over most of the horse's life. It is important in this context to make a distinction between tooth growth and tooth eruption. Tooth growth implies the lengthening of the tooth in its apical part due to the deposition of new layers of dentin and cement. Tooth eruption is the progressive protrusion of the tooth out of its alveolus. It is now generally assumed that tooth eruption is caused by a continuous remodeling of the periodontal ligament fibers and not by root lengthening as was claimed before. The deposition of bone at the bottom of the alveolus should be the result rather than the cause of tooth eruption.[1]

Equine incisors erupt lifelong, whereas their intrinsic growth ceases at the age of about 17.[2] As the total length of horse incisors remains unchanged from the age of six until the age of 17, the continuous loss of occlusal dental tissue is compensated by an equal amount of newly formed dental tissue at the apical end of the tooth. During this period of time tooth root growth makes up for occlusal wear, which is estimated to occur at a rate of 2.5 mm a year.[3] In horses aged over 17 years, occlusal wear is no longer compensated by apical tooth growth and the total incisival length diminishes progressively. In horses of this age category continuous tooth eruption is the only mechanism to provide maintenance of occlusal contact between upper and lower incisors.

Because of the marked wear on the surface of hypsodont equine teeth, occlusal exposure of dentin and cement is inevitable after the protecting enamel is worn off. This leads to the presence of alternate layers of the three calcified tissues at the occlusal surface, whose continuously changing configuration allows a macroscopic dental age estimation.[4–15]

The most appropriate teeth for aging horses are the (lower) incisors. The localization of premolars and molars interferes with a comfortable inspection but radiographic evaluation of the cheek teeth can be used as an aid in estimating age, especially in the young horse.[8] Canines are not well suited for age estimation. Because contact between upper and lower canines is seldom made, canines do not wear down in a regular way and have no age-related occlusal surface.

When estimating a horse's age by its incisors, the eruption dates and the changes in appearance of the occlusal surfaces are the main criteria. Neither is wholly dependable but the first is the more reliable, although limited in application to younger animals. The second may be used throughout the life span but becomes increasingly inaccurate with age.[16] Incisival characteristics that are frequently used for dental aging in horses are summarized below.

Eruption

In this context gingival emergence is used as a reference point for eruption.

Eruption of the deciduous incisors (Fig. 5.1)

The deciduous incisors are smaller than the permanent ones. The surface of their crown is white and presents several small longitudinal ridges and grooves. The occlusal tables of deciduous incisors are oval in the mesiodistal direction.

Eruption of the permanent incisors (Figs 5.2 and 5.3)

Permanent incisor teeth are larger and more rectangular than the deciduous incisors. Their crown surface is largely covered with cement and has a yellowish aspect. The upper incisors generally present two distinct longitudinal grooves on their labial surface; the lower incisors have only one clearly visible groove.



Figure 5.1. Arabian horse, 2 years old. The deciduous incisors have small ridges and grooves on their labial surface.



Figure 5.3. Belgian draft horse, 5 years and 7 months. All incisors are permanent and have a yellowish appearance.

Changes of the occlusal surface (Fig. 5.4)

Appearance of the dental star

The dental star is a yellow-brown structure on the occlusal surface situated between the labial edge of the incisor and the infundibular cavity or 'cup.' It consists of secondary and tertiary dentin that occludes the pulpal chamber when it risks being exposed by wear. In young animals the dental star appears as a linear stripe because the occlusal end of the original pulp cavity is not conical but elongated in a mesiodistal direction. With age dental stars become oval and then round and move towards the center of the occlusal table. These progressive patterns reflect cross-sections through the stuffed pulp cavity at various levels.

Disappearance of the cups

The infundibulum is an enamel infolding in the occlusal surface of the equine incisor. The superficial half of the infundibulum is empty or filled with food particles. This part is called the 'cup.' The bottom of the infundibulum is filled with cement. When wear has brought this infundibular cement layer into the occlusal surface, the cup is filled in or has disappeared. The exposed cement core and the surrounding enamel ring are called the 'mark.'

Disappearance of the marks

The shape of the mark generally corresponds to the contour of the occlusal table of the incisor. In young horses marks are oval in the mesiodistal direction. When wear progresses, marks become smaller and rounder and move caudally (lingually) on the occlusal surface. With age the cement of the infundibular bottom wears away and eventually the remaining enamel spot disappears from the occlusal surface.

Changes in shape of the incisors

Changes in shape of the occlusal surfaces (Fig. 5.5)

Due to extensive wear, the sequential shapes of the occlusal tables represent the cross-sections of the incisor teeth at



Figure 5.2. Belgian draft horse, 2 years and 10 months. The right permanent central incisor (401) is emerging through the gum. All other incisors are deciduous.



Figure 5.4. Standardbred horse, 8 years old. Dark-colored dental stars are present in all lower incisors. The characteristic white spot in the center of the dental star appears in the centrals (arrows). Cups have disappeared from the central incisors. The remaining marks are oval (arrowheads). Deep cups are still present on the middle and the corner incisors.

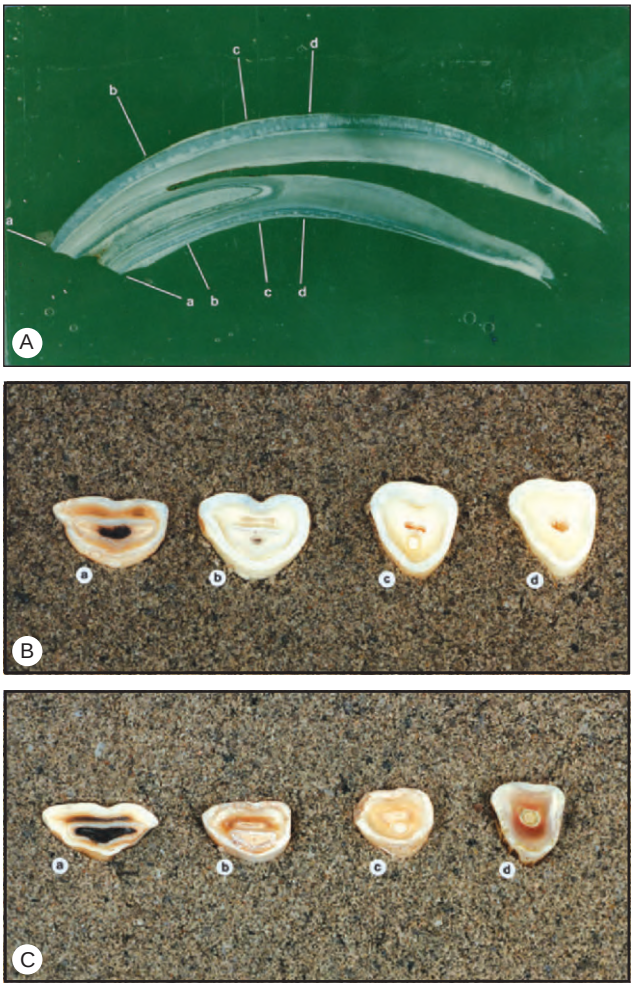


Figure 5.5. (A) Longitudinal section of the lower central incisor of a standardbred horse (4 years old). (B) Lower central incisor of a 5-year-old standardbred horse. Cross-sections at various levels as indicated in (a). In the sections c and d the pulpal cavity is open. (C) Occlusal tables of the lower central incisor of standardbreds aged: a, 5 years; b, 8 years; c, 14 years; d, 20 years, respectively. In the occlusal tables c and d the pulpal cavity is occluded by secondary dentin.

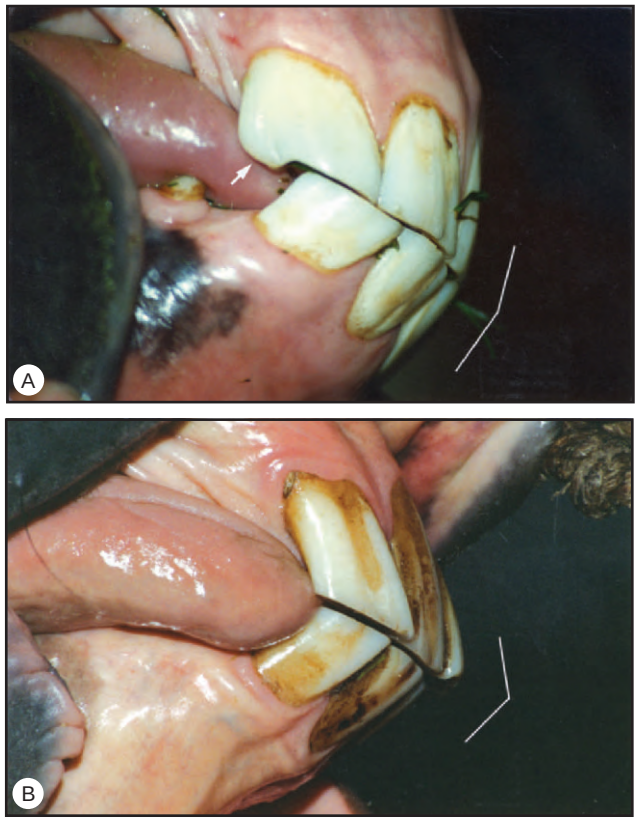


Figure 5.6. (A) Belgian draft horse, 6 years old. The upper and lower incisors are positioned in a straight line with each other. The crown of the upper corner (103) is wider than it is tall. Notice the presence of a hook on the upper corner (arrow). (B) Standardbred horse, 16 years old. The angle between upper and lower incisors is more acute. The crown of the upper corner (103) is taller than it is wide. The upper corner presents a Galvayne's groove over the entire length of its labial surface.

various levels. The sequence ranges from oval in the mesiodistal direction, to trapezoid and triangular, and finally to oval in the labiolingual direction.

Direction of upper and lower incisors (Fig. 5.6)

When incisors are viewed in profile, the angle between the upper and lower incisors changes with age. In young horses the upper and lower incisors are positioned in a straight line (angle ±180°) with each other. With age, the occlusal portions of the crowns wear off and we look at different cross-sections of the crown shape in profile. The angle between upper and lower incisors becomes therefore increasingly acute. The lower incisors are the first to obtain an oblique position followed at a later date by the upper incisor teeth.

Length versus width of the upper corner incisor (Fig. 5.6)

The shape of the upper corner incisor has been used recently to categorize a horse's age into three groups from 5–20 years of age.[8] Between 5 and 9 years of age the crown of this tooth is generally wider than it is tall. At ages 9–10, the upper corner appears square in most horses and then progresses to taller than it is wide as age increases.

The hook on the upper corner incisor (Fig. 5.6A)

The caudal edge of the upper corner sometimes exceeds the occlusal surface of the lower corner, especially when the lower incisors have acquired their oblique position. If the caudolateral portion of the upper corner is no longer in contact with its lower counterpart, it wears more slowly, forming a hook in the occlusal surface. Later, when the upper incisors obtain their oblique position and the caudal edge of the upper corner is in contact with its lower counterpart again, this notch can disappear.

The Galvayne's groove (Fig. 5.6B)

The Galvayne's groove is a shallow longitudinal groove on the labial surface of the upper corner and is filled with dark stained cement. In the unworn tooth the groove starts halfway from occlusal surface to apex and continues three-fourths of the distance to the apex. It is buried within the alveolus when the tooth first comes into wear.[17] With age, and due to the prolonged eruption of the tooth, the Galvayne's groove first appears at the gumline. As the tooth continues to erupt, it extends down the labial surface to reach the occlusal edge, then starts to disappear at the gumline and finally disappears completely. The appearance of the groove and its usefulness in aging horses was mentioned for the first time in the early 1880s by an American horse-tamer called Sample. Later, his theory was adopted by Sidney Galvayne, an Australian horseman.[18] It was in his first work *Horse Dentition: Showing how to tell exactly the age of a horse up to thirty years* (published prior to March 1886) that Galvayne described the groove, which now bears his name, on the vestibular surface of the permanent upper corner incisor.[19] The presence and length of the Galvayne's groove as an accurate guide to the age of the older horse became known throughout the English-speaking world. However, it was not until World War I that several investigations were undertaken to validate his theory.[18] Contrary to Galvayne's statements, these investigations showed that the groove may be absent in more than 50 per cent of the horses between the ages of 10 and 30 years.

Dental star morphology

The appearance of the dental star is, next to eruption times, one of the more reliable dental features, and the correlation between dental star morphology and age is stronger than for any other feature.[20]

Horses at pasture have obvious darkly colored dental stars, whereas individuals without access to pasture or grass fodder usually have pale yellowish dental stars (Fig. 5.7). This suggests that the coloration of the dental star is caused by an impregnation of grass pigments. Two small experiments support this theory:[21] (1) When equine incisors are sectioned longitudinally one can observe that the brown color of the dental star extends only a few millimeters beneath the occlusal surface and that the color intensity fades towards the pulpal chamber. This indicates that the color originates from the 'outside world' rather than from the pulp as suggested in older literature reports.[3,22] (2) When incisors with pale dental stars are stored in a mush of crushed grasses, dental stars become darkly colored after a few days; when they are stored in a buffered (pH 6.8) solution of various diphenols (caffeic acid, 3,4-dihydroxybenzoic acid and 3,4-dihydrophenylalanine [10 mmol/l]) together with thyrosinase, the dental stars obtain a deep brown color after 72 hours (Fig. 5.8). This suggests that food pigments are responsible for the dark color of the dental star.



Figure 5.7. Standardbred horse, 12 years old, deprived of fresh grass. Dental stars are yellowish (arrowheads). It is difficult to distinguish the white spot in the center of the stars.

Dental stars also present the topical coloration pattern. In young horses the dental stars have a uniform color, whereas in older individuals they are composed of a darker periphery that surrounds an uncolored central zone, the so-called 'white spot' (Fig. 5.9). The reason why absorption of food pigments occurs only in the peripheral rim of the dental star and not in the white spot nor in the surrounding primary dentin can be found by examining the diameter, extent and orientation of the dentinal tubules.

Dentinal tubules are formed as the odontoblasts retreat centripetally and leave behind a cytoplasmatic process around which the dentin matrix is deposited and mineralized. The tubules can therefore be regarded as hollow cylinders traversing the dentin. Each tubule starts peripherally at the interface between the primary dentin and the enamel, and extends centripetally toward the pulpal border. The first dentin produced by the odontoblasts is located peripherally



Figure 5.8. Lower central incisors of a 20-year-old standardbred without access to pasture. The right tooth (301) was stored in a buffered Ringer lactate solution; the left one (401) was immersed in a buffered solution of 3,4-dihydroxybenzoic acid (10 mmol/l) for 48 hours. In 401 the periphery of the dental star has obtained a brown color (arrows).



Figure 5.9. Occlusal surface of the left lower central incisor (401) of a 15-year-old draft horse. The dental star consists of a dark peripheral rim (asterisk) and a central white spot (arrow). The white spot is composed of secondary dentin (A) and a core of tertiary dentin (B).

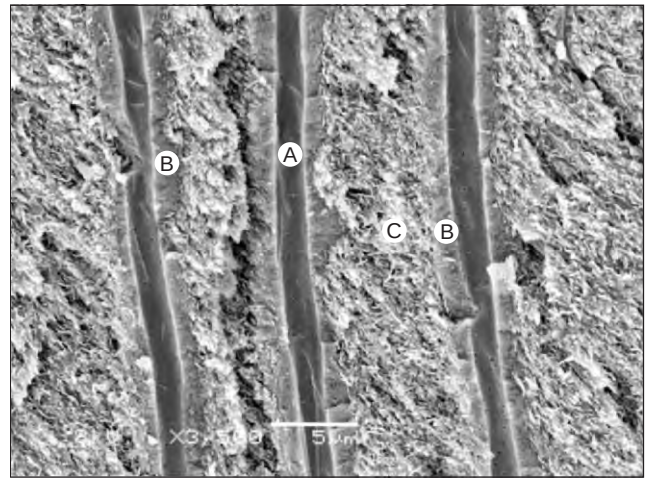


Figure 5.10. SEM image of longitudinally fractured dentinal tubules. A: tubular lumen; B: peritubular dentin; C: intertubular dentin (×3500).

in the tooth, i.e. underneath the enamel, and is called primary dentin. It surrounds the younger and more centrally located secondary dentin, whereas tertiary dentin is only formed in the restricted areas between the tip of the pulp chamber and the occlusal surface.

The only obvious feature characterizing the transition between primary and secondary dentin of equine teeth is the presence of peritubular dentin (Fig. 5.10), which is hypercalcified tissue, deposited as a collar inside the tubular walls of primary dentin. The term peritubular dentin is anatomically incorrect because this dentin forms within the dentinal tubule (not around it) and narrows the tubular lumen. It is therefore sometimes (more accurately) referred to as intratubular dentin.[23] Apart from the presence of peritubular dentin, the structure of dentinal tubules is identical in primary and secondary dentin. Peritubular dentin deposit is thickest at the outer end of the primary dentinal tubules and disappears at the transition between primary and secondary dentin.[21] The presence of peritubular dentin gives the tubules a tapered shape with the wider lumen at the pulpal side and the narrower luminal diameter near the enamel.

The dental star consists of a central core of tertiary dentin and a much broader ring of secondary dentin, in neither of which peritubular dentin is deposited. Tertiary dentin, situated in the very center of the dental star, is formed between the tip of the pulp chamber and the occlusal surface and protects the pulp from exposure to attrition. Penetration of food pigments in this zone of the dental star is prevented by the small number of tubules, which are for the most part discontinuous with those of the surrounding secondary dentin, their irregular arrangement and their small diameter (Fig. 5.11). This explains the colorless aspect of the central core of tertiary dentin inside the dental star.

The secondary dentin around the core of tertiary dentin consists of a pale inner zone and a brown peripheral zone. Both zones contain regularly arranged dentinal tubules that are continuous with those of the surrounding primary dentin and are completely devoid of peritubular dentin. The high numerical tubular density, the regular tubular arrangement and the large tubular diameters of the secondary dentin are suggestive of an easy and uniform penetration of food pigments in this area. The only difference between the pale inner zone and the dark peripheral zone of secondary dentin is the spatial arrangement of the dentinal tubules. In the periphery of the dental star, tubules end perpendicularly into the occlusal surface (Fig. 5.12). This orientation allows an optimal inflow of food pigments which is far superior to the dye penetration in the more central uncolored secondary dentin, where tubules lie nearly

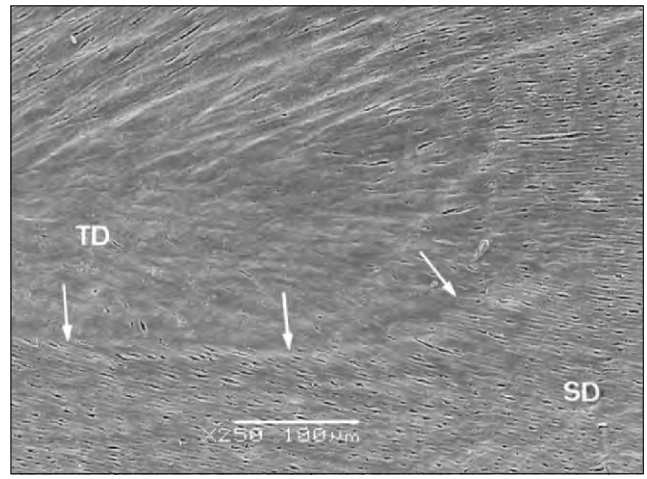


Figure 5.11. SEM image of the occlusal surface at the center of the dental star. The boundaries of the tertiary dentin are indicated by arrows. TD: tertiary dentin; SD: secondary dentin (×250).

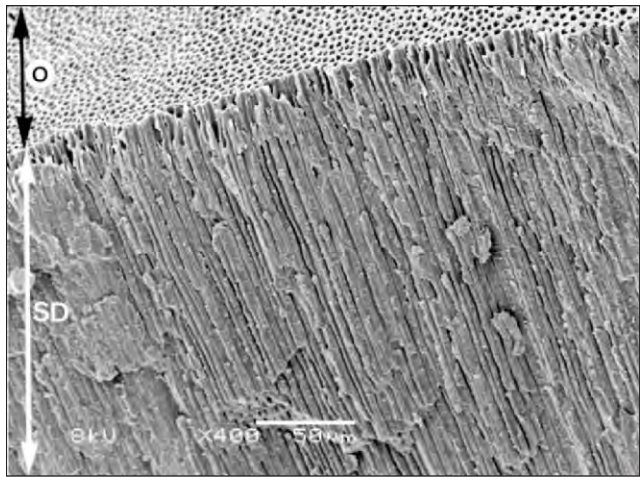


Figure 5.12. SEM image of the etched occlusal surface (O) and the subjacent secondary dentin (SD) at the dark peripheral rim of the dental star of a longitudinally fractured incisor. Dentinal tubules end perpendicular to the occlusal surface (×400).

parallel to the occlusal surface (Fig. 5.13). Penetration of food pigments in the latter zone is nearly negligible because due to the horizontal position of the tubules; the maximal penetration depth of food pigments in this zone cannot exceed the tubular diameter, which is 3 µm. Even when the horizontally exposed tubules are filled with food pigments, this 3 µm-thick mass of colored dentin is worn off in less than 1 day by the severe occlusal attrition, which amounts to 2500 µm a year. Food pigments can therefore not be accumulated in the inner zone of the secondary dentin of the dental star. This contrasts with the more peripheral zone of secondary dentin, where food pigments can permeate a longer distance in the perpendicularly debouching tubules. The pigments can accumulate within these tubules and thus cause the dark coloration of the dental star periphery. This mechanism is fully compatible with the aforementioned

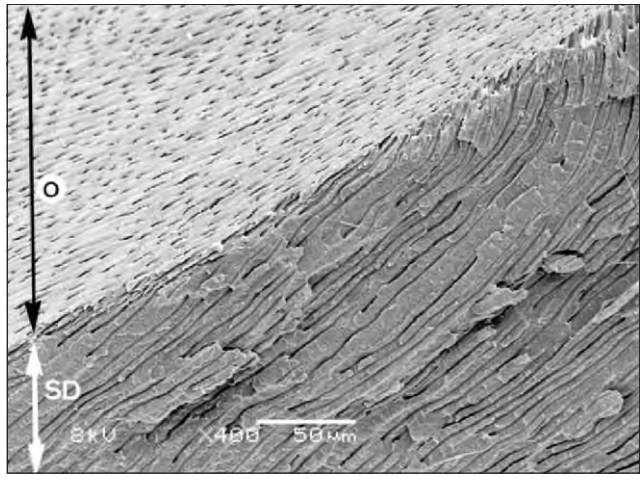


Figure 5.13. SEM image of the etched occlusal surface (O) and the subjacent secondary dentin (SD) at the pale inner zone of secondary dentin in the dental star. The orientation of the tubules is almost parallel to the occlusal surface (×400).

preliminary experiments, showing that secondary dentin acquires its dark color within 72 hours after immersion in a pigmented solution.[21]

The microstructure and spatial arrangement of the dentinal tubules can also explain the color differences between primary and secondary dentin. Primary dentin has a smaller number of tubules per area unit than secondary dentin and thus offers fewer pathways for food pigments to penetrate into the occlusal surface. In primary dentin, exposed onto the occlusal surface peripheral to the dental star, tubules debouch obliquely. For the reasons as described above, penetration of food pigments into these tubular lumina will be considerably less than in the dark peripheral rim of the dental star, where tubules end perpendicularly. Furthermore, primary dentin contains high levels of peritubular dentin and has an almost translucent appearance which is similar to the complexion of enamel, as both tissue types are highly mineralized.[24] In contrast, the less mineralized secondary dentin has a dull opaque appearance. Additionally, because tubules in secondary dentin are devoid of peritubular dentin, they have wider lumina in which plant pigments can penetrate more easily and give the dentinal tissue a dark brown color.

Dental aging in different horse breeds

Many standard textbooks dealing with aging of horses suggest that the above-mentioned characteristics give an accurate indication of a horse's true age. However, some reports are inconsistent in their guidelines and show large discrepancies in the dental features described at specific ages. A possible explanation for the non-uniformity of existing guidelines is the lack of evidence that any system was used to validate an author's recommendations for aging.[25,26] A study performed by Richardson *et al.*[27] cast serious doubts on the belief that the age of a horse can be determined accurately from an examination of its teeth. In this study a large group of horses with documented evidence of birth were examined and age was estimated both by experienced clinicians and by a computer model. There was little difference between the accuracy of the computer model and the clinical observers, but neither method was accurate when compared with the actual age. In older horses there was much greater variability between the dental age and the actual age, which means that the accuracy of dental aging declines markedly with age. Most standard texts do not provide exact data concerning breed, sex and nutrition of the examined horses. However, anatomical, physiological, environmental and behavioral differences between individuals ensure differences in rate of equine dental wear.[28] The concealment of these data may explain the discrepancies between different reports. Inaccuracies in the dental aging system of horses may also result from differences between breed and type of horse involved. Eisenmenger and Zetner

stated that the teeth of thoroughbreds erupt earlier than those of Lipizzaners and coldblood horses. Teeth of ponies may also have rates of eruption and wear that differ from the teeth of horses.[3] As for donkeys, both ancient literature data[29] and recent investigations[30] have suggested that the degree of dental attrition in donkeys is slower than in horses.

The nature of diet can also play a part in the abrasion of horse incisors. Dental wear is caused not only by grinding of opposing crowns against one another, but also by contact with abrasive particles in food such as silicate phytoliths which form part of the skeleton of grasses. Other plant-borne abrasives include cellulose and lignin.[28] In order to preclude the influence of the quality of nutrition on the rate of dental wear, it is necessary that horses that are examined for breed variability are raised and kept under similar environmental and nutritional conditions.

Based on the suggestions that the degree of attritional dental wear is correlated with the breed of horse, four unrelated horse breeds have been subjected to a comparative study.[31–33] All horses examined here were raised in Western Europe, were given access to daily pasture and were fed concentrates and hay. None of the horses was a crib-biter nor suffered from other vices with a possible influence on dental wear. The incisor teeth had not been rasped in any individual. It is evident that in practice one has to be vigilant for these considerations. Factors that are difficult to control and that could not be taken into consideration are the individual chewing habits and the amount of food intake.

A critical evaluation of the dental aging technique revealed that the rate of attritional dental wear is different in different horse breeds. Indentation hardness tests, performed with a Knoop diamond indenter, showed slight breed differences in the hardness of equine enamel and dentin. These different microhardness values seem to contribute to the differences in the rate of attritional dental wear.[34]

The following text describes the appearance of lower incisor teeth at various ages as generally seen in the Standardbred horse, the Belgian draft horse, the Arabian horse and the mini-Shetland pony population of Western Europe. It must be emphasized that this text is not a truism. When determining a horse's age one must register all dental features together and take account of clinical factors that may have influenced the aspect of the horse's teeth. The following descriptions will therefore be accurate in many cases, but may be incorrect for any individual.

Eruption of the deciduous incisors

The central incisors generally erupt during the first week of life. The middle incisors emerge through the gums at 4–6 weeks and the corners erupt between the sixth and the ninth month of life. In the mini-Shetland pony, eruption of the middle and the corner incisors is retarded. The middle incisor starts erupting at the age of 4 months, whereas the corner incisor breaks through the gums between 12 and 18 months of age.

Eruption of the permanent incisors

The upper and lower permanent incisors erupt almost simultaneously. In some horses shedding begins with the maxillary, in others with the mandibular incisor teeth. Arabian horses shed their central, middle and corner incisors at 2.5, 3.5 and 4.5 years of age, respectively. In Standardbreds and in Belgian draft horses, shedding generally occurs later, namely at nearly 3, nearly 4 and nearly 5 years of age (Fig. 5.14). In mini-Shetland ponies, eruption of the permanent incisors is still further delayed by 2 or 3 months. In male animals the canines erupt at about 4.5–5 years of age. Generally, these teeth are absent or rudimentary in mares.

Appearance of the dental star

Dental stars appear sequentially in the central, the middle and the corner incisors. In standardbreds and in Arabian horses they appear on the centrals at 5 years, on the middles at 6 years and on the corners at 7–8 years. In Belgian draft horses and mini-Shetland ponies, stars appear somewhat earlier, namely on the centrals at 4.5 years, on the middles at 5.5 years and on the corners at 6.5–7 years (Fig. 5.14). With age the characteristic white spot becomes visible in the center of the dental star (Figs 5.15–5.17). In Standardbreds and in Arabian horses this white spot appears on the central incisors from the age of 7–8 years onwards, and on the middle incisors from the age of 9–11 years onwards. In Belgian draft horses and in mini-Shetland ponies the white spot becomes visible on the centrals at the age of 6–7 years and on the middles at the age of 8. In all breeds, the appearance of the white spot in the dental star of the corner incisors is variable and occurs between 9 and 15 years.

Disappearance of the cups

The disappearance of the cups is an unreliable feature for age determination because it does not occur between narrow age limits. In all breeds, cups on the central incisors disappear at the age of 6–7 years, whereas cups on the middle incisors are filled in variably between 7 and 11 years and those on the corners between 9 and 15 years. The variations in the age at which the cups disappear may be due to a difference in the depth of the cup. The accumulation of cement in the infundibulum is variable, i.e. superabundant in some individuals and almost non-existent in others.

Changes in shape of the marks

On the central incisors big oval marks are visible until the age of 6–7 years. These marks become oval to triangular from the age of 7–8 years onwards in Belgian draft horses, from the age of 8–10 years onwards in Standardbreds and Arabian horses, and from the age of 10 years onwards in mini-Shetlands. Round marks on the central incisors are visible at 9–10 years in Belgian draft horses, at 13–14 years in Standardbreds and mini-Shetlands and at 15–17 years in Arabian horses (Fig. 5.17).

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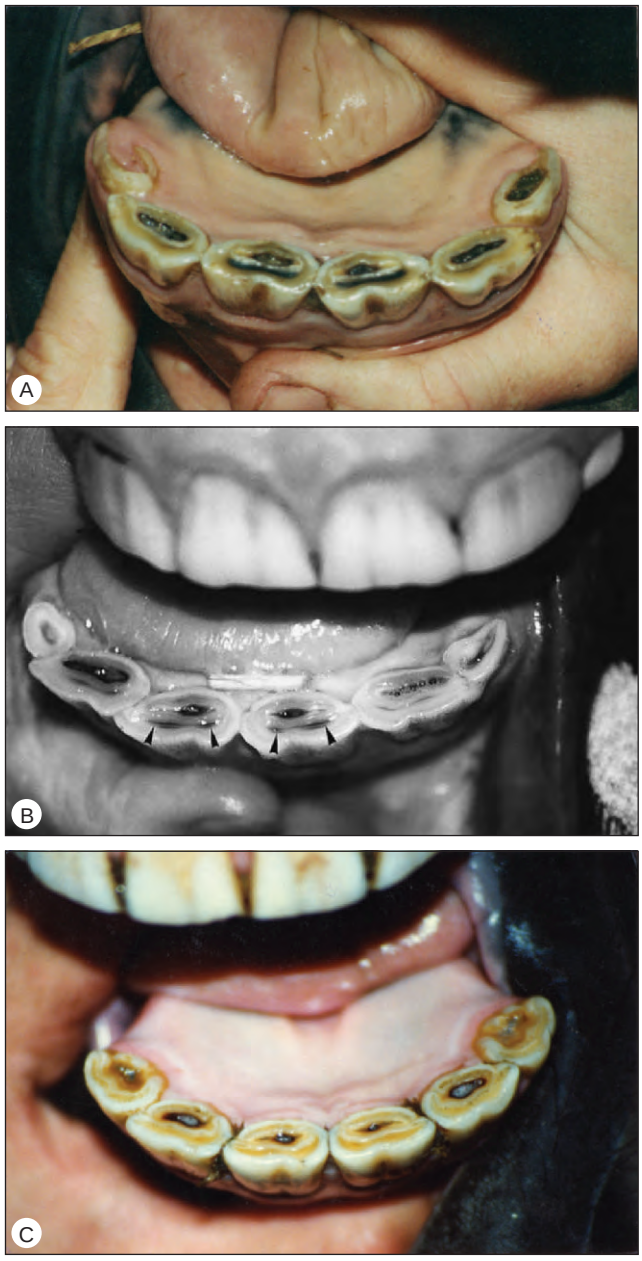


Figure 5.14. (A) Standardbred horse, 5 years old. The permanent central and middle incisors are in place, the corner incisor is emerging through the gums. The dental star is present on the centrals, absent on the middles and the corners. All lower incisors have deep cups. (B) Belgian draft horse, 4 years and 8 months. The permanent central and middle incisors are in place. The corner incisors are still deciduous. Dental stars are present on the centrals (arrowheads) and appear also on the middles. Cups are present in the central and the middle incisors. (C) Arabian horse, 5 years old. All lower incisors are permanent, the corners are not yet fully in wear. There are no obvious dental stars. Deep cups are present on all lower incisors.

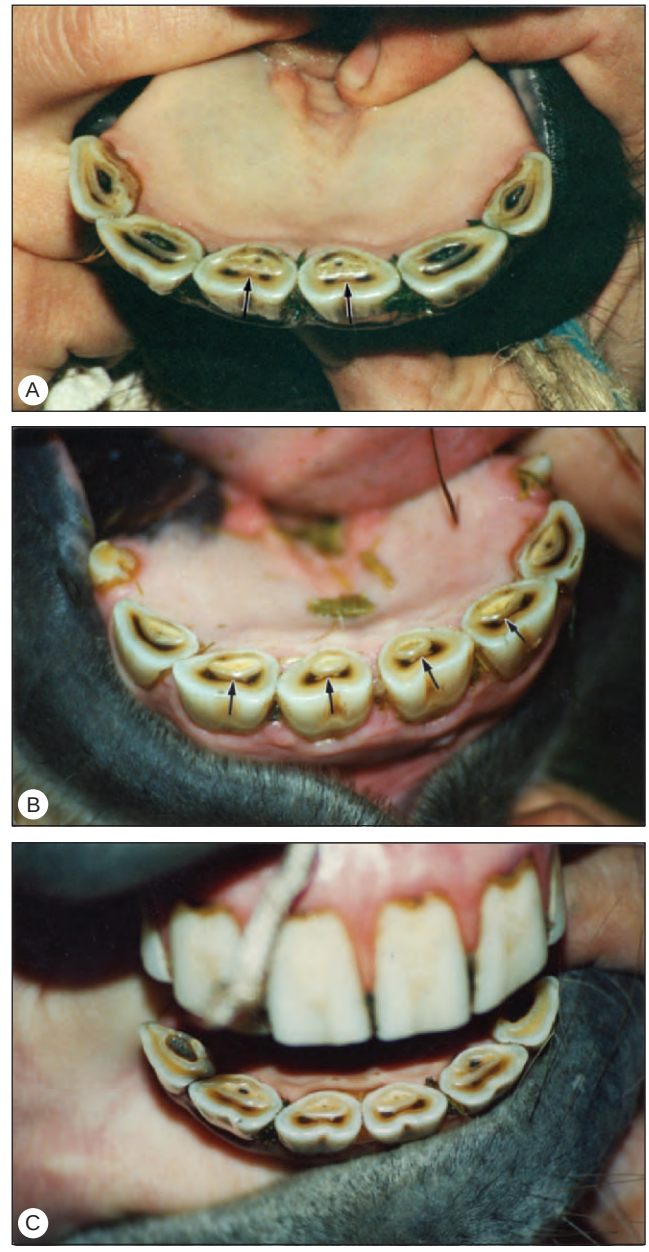


Figure 5.15. (A) Standardbred horse, 8 years old. Dental stars are present on all incisors. In the central incisor the white spot in the dental star becomes apparent (arrows). Cups are filled-in on the centrals. On the middles and the corners, cups are still present. The occlusal tables of the central incisors are becoming trapezoid, those of the middles and the corners are still oval. (B) Belgian draft horse, 8 years and 6 months. Dental stars are present on all incisors. In the central and the middle incisors the white spot in the dental star becomes apparent (arrows). Cups are filled-in on all lower incisors. The remaining marks are oval. The occlusal tables of the centrals and the middles are becoming trapezoid. (C) Arabian horse, 8 years and 6 months. Dental stars are present on the central and the middle incisors. The white spot in the dental star is appearing in the central incisor. Cups on the centrals and the middles have nearly disappeared, the remaining marks are oval (middles) to triangular-shaped (centrals). Deep cups are still present on the corner incisors. The occlusal tables of the centrals become trapezoid.

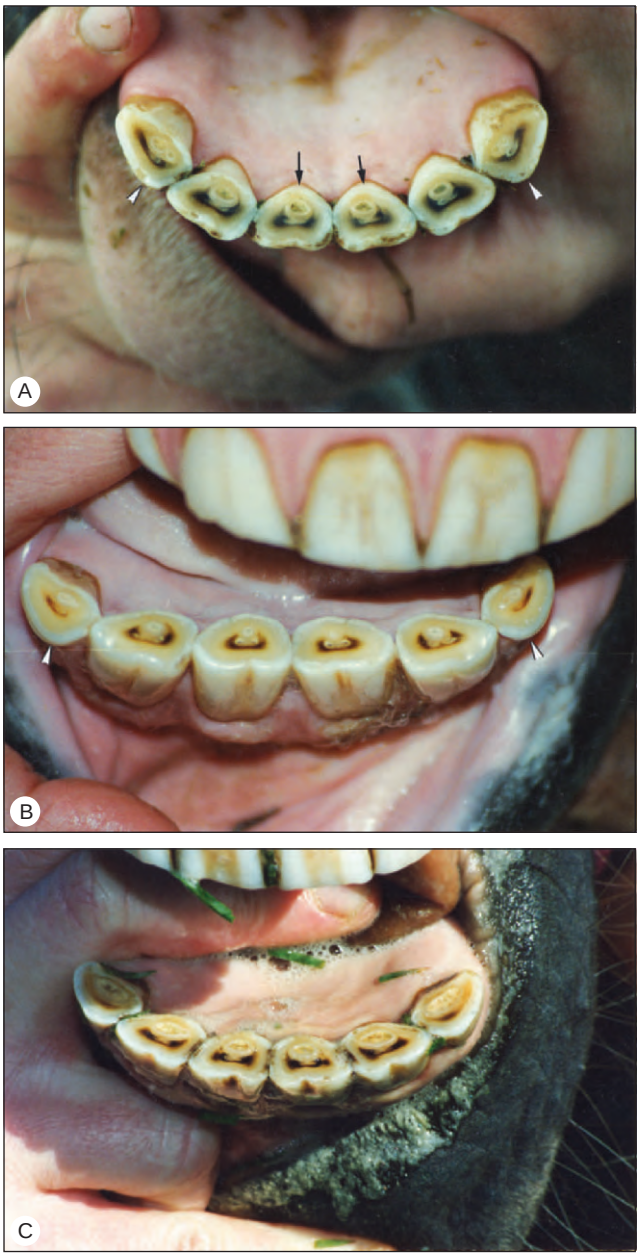


Figure 5.16. (A) Standardbred horse, 12 years old. Dental stars, consisting of a white spot and a dark periphery, are present on all lower incisors. Cups have disappeared and the marks are small oval to rounded. The occlusal tables of the central and the middle incisors are trapezoid. On the central incisor the lingual apex is visible (arrows). The corner incisors have an apex on the labial side (arrowheads). (B) Belgian draft horse, 12 years old. Dental stars, consisting of a white spot and a dark periphery, are present on all lower incisors. Marks are rounded and on the central incisors they have almost disappeared. The occlusal tables of the centrals and the middles are trapezoid. On the corner incisor the labial apex is obvious (arrowheads). (C) Arabian horse, 12 years old. Dental stars are present on all lower incisors. On the central and the middle incisors the white spot in the dental star is visible. Cups have disappeared. The remaining marks are oval and still clearly visible. The occlusal tables of the centrals and the middles are trapezoid. The corner incisor presents a labial apex.

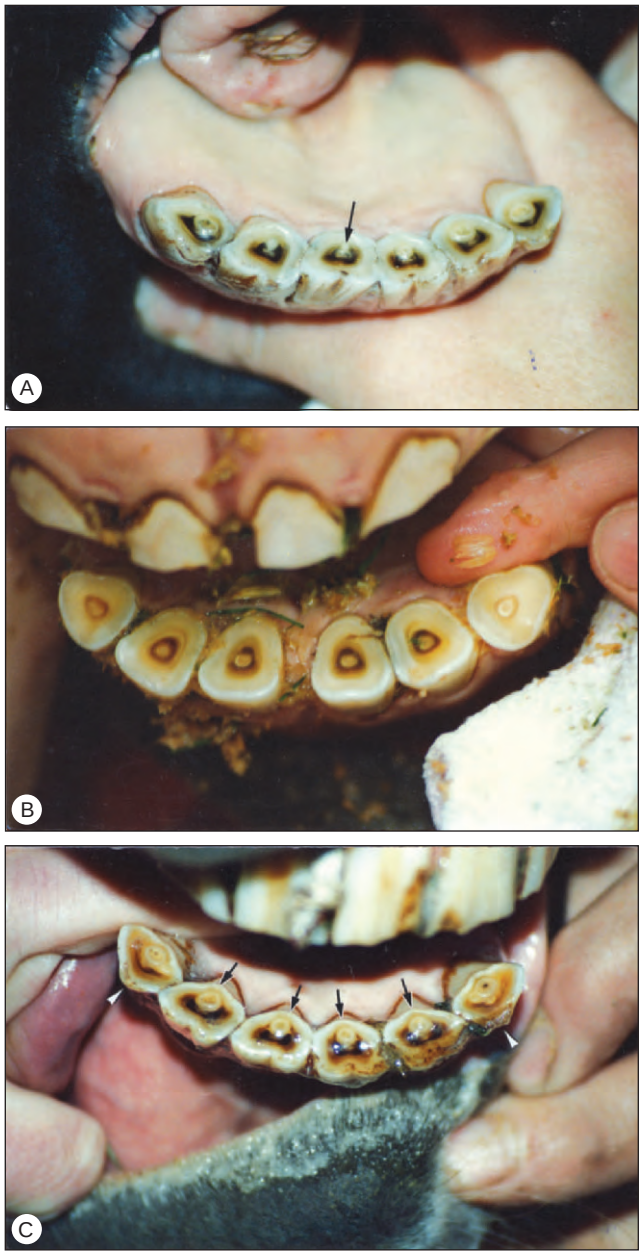


Figure 5.17. (A) Standardbred horse, 18 years old. Marks are small and rounded. On the central incisors they have almost disappeared (arrow). The occlusal tables of the centrals and the middles are trapezoid, those of the corner incisors are triangular with an apex to the labial side. (B) Belgian draft horse, 18 years old. Marks have disappeared from all lower incisors. The occlusal surfaces are triangular; those of the central and the middle incisors have a lingual apex and those of the corners have a labial apex. (C) Arabian horse, 18 years old. Round marks are still clearly visible on all lower incisors. The occlusal surfaces of the centrals and the middles are trapezoid with a lingual apex (arrows), those of the corner incisors are oval with a labial apex (arrowheads).

Disappearance of the marks

From all age-related dental characteristics, the disappearance of the marks is the one with the highest interbreed variability. In draft horses, marks on the central incisors disappear from the age of 12–15 years, those on the middles and the corners from the age 14–15 years onwards. In mini-Shetland ponies, marks on the central, the middle and the corner incisors disappear at the age of 15, 16 and 17 years, respectively. In Standardbred horses, marks disappear some years later. On the centrals they vanish in 18-year-old horses while disappearing on the middle and the corner incisors in 19- to 20-year-olds. In Arabian horses, marks on the lower incisors may persist for a very long time. They start disappearing at the age of 20 but exhibit considerable individual variations (Fig. 5.17C).

Changes in shape of the occlusal surfaces

Changes in shape of the occlusal surfaces of the lower incisors are useful but inaccurate indicators of age. The changes are difficult to judge objectively because successive shapes shade off into one another and are not easily distinguishable. The sequential shapes of the tables of the central and the middle incisors are oval, trapezoid, triangular with the apex pointing to the lingual side and biangular. A survey of the most important changes is given in Tables 5.1–5.4. It is striking that the shape of the lower corner incisor does not conform to the

Table 5.1. Aging Belgian Draft Horses

	I1	I2	I3
Shedding	±3 y	±4 y	±5 y
Appearance of the dental star	4.5 y	5.5 y	6.5 y–7 y
Appearance of the white spot in the dental star	6 y–7 y	7 y–8 y	11 y–13 y
Disappearance of the cup	5 y–8 y	7 y–11 y	9 y–15 y
Shape of the mark:			
oval	until 6 y		
oval-triangular	≥7 y–8 y		
round	≥ 9 y–10 y		
Disappearance of the mark	12 y–15 y	14 y–15 y	14 y–15 y
Shape of the occlusal table:			
oval	until 6 y	until 7 y	until 10 y
trapezoid	≥7 y	≥8 y–9 y	–
trapezoid with lingual apex	≥7 y	≥9 y	–
labial apex on 303 or 403			≥9 y–10 y
Hook on 103 or 203			≥5 y
Galvayne's groove			≥11 y

Table 5.2. Aging Standardbred Horses

	I1	I2	I3
Shedding	±3 y	±4 y	±5 y
Appearance of the dental star	5 y	6 y	7 y–8 y
Appearance of the white spot in the dental star	7 y–8 y	9 y–11 y	11 y–13 y
Disappearance of the cup	6 y–7 y	7 y–11 y	9 y–15 y
Shape of the mark:			
oval	until 6 y		
oval-triangular	8 y–10 y		
round	≥13 y		
Disappearance of the mark	18 y	19 y–20 y	19 y–20 y
Shape of the occlusal table:			
oval	until 6 y	until 7 y	until 12 y
trapezoid	≥7 y	≥8 y–9 y	–
trapezoid with lingual apex	≥ 9 y	≥10 y	–
labial apex on 303 or 403			≥10 y–11 y
Hook on 103 or 203			≥5 y
Galvayne's groove			≥11 y

Table 5.3. Aging Arabian Horses

	I1	I2	I3
Shedding	±2.5 y	±3.5 y	±4.5 y
Appearance of the dental star	5 y	6 y	7 y–8 y
Appearance of the white spot in the dental star	7 y–8 y	9 y–11 y	13 y–15 y
Disappearance of the cup	7 y	7 y–11 y	9 y–15 y
Shape of the mark:			
oval	until 7 y		
oval-triangular	≥8 y–10 y		
round	≥15 y–17 y		
Disappearance of the mark	≥20 y	≥20 y	≥20 y
Shape of the occlusal table: oval	until 6 y	until 7 y	until 12 y
trapezoid	≥8 y–9 y	≥9 y–11 y	–
trapezoid with lingual apex	≥10 y–11 y	≥14 y	–
labial apex on 303 or 403			≥12 y
Hook on I03 or 203			≥5 y
Galvayne's groove			≥11 y

Table 5.4. Aging Mini-Shetland Ponies			
	I1	I2	I3
Shedding	≥3 y	±4 y	±5 y
Appearance of the dental star	4.5 y	5.5 y	6.5 y–7 y
Appearance of the white spot in the dental star	6 y–7 y	8 y	10 y–12 y
Disappearance of the cup	7 y–8 y	8 y–12 y	9 y–13 y
Shape of the mark:			
oval	until 8 y		
oval-triangular	≥10 y		
round	≥13 y		
Disappearance of the mark	15 y	16 y	17 y
Shape of the occlusal table:			
oval	until 6 y	until 7 y	until 10 y
trapezoid	≥7 y	≥8 y–9 y	–
trapezoid with lingual apex	≥11 y–12 y	≥14 y	–
labial apex on 303 or 403			≥9 y–10 y
Hook on 103 or 203			≥5 y
Galvayne's groove			≥11 y

sequential changes described above. The lower corners remain oval for a long time and gradually develop an apex at the labial side. In Belgian draft horses and mini-Shetland ponies a labial apex appears at the age of 9, and in Standardbreds at the age of 11. In Arabian horses the apex is a constant characteristic in individuals aged over 12 years (Figs 5.16 and 5.17).

Direction of upper and lower incisors

The arch formed by the incisors of the opposing jaws as they meet, when viewed in profile, changes as the teeth advance from their alveoli and undergo attrition (Fig. 5.6). In young horses the upper and lower incisors are positioned in a straight line (±180°). From the age of approximately 10 years onwards, the angle between upper and lower incisors becomes more acute. Because exact measurements of the age-related incisival angle are not available, the evaluation of the angle provides only a rough estimate of an animal's age. The same applies for the curvature of the dental arch formed by the lower incisive tables. In young horses this arch is a semicircle, whereas in older individuals it forms a straight line. In view of the gradual character of this change in direction, however, it is impossible to determine the exact age at which it occurs.

The hook on the upper corner

The hook on the caudal edge of the upper corner incisor has long been considered as the typical characteristic for a 7- or 13-year-old horse. However, hooks on 103 and 203 are seen in a minority of horses and occur at practically any age over 5 years. Only 13 per cent of all 7-year-olds and 8 per cent of all 13-year-olds that were examined for this study presented a hook on one or both upper corners. On the other hand, hooks were also seen in 14 per cent of the 5- and 6-year-old horses, in 22 per cent of the horses aged between 8 and 12, and in 13 per cent of all horses aged over 13 years. As the presence of hooks on 103 and 203 cannot be related to any specific age category it is considered irrelevant for the estimation of age in horses.

The Galvayne's groove

The Galvayne's groove is a feature that is most often observed in horses aged over 11 years. However, as its presence, length and bilateral symmetry are variable and inconsistent, the groove is considered to be of little value for the age determination in horses.

Conclusion

Teeth provide a practical available tool for estimating age in horses. Aging an individual horse from its dentition, however, is a complex process and all above-mentioned features should be carefully examined. It must be emphasized that dental aging in horses can only provide an approximate guess rather than an exact evaluation. In older horses most of the so-called characteristic features can only be judged subjectively. It is obvious that the accuracy of the dental age determination declines markedly with age.

An important factor that can prevent an accurate dental age determination in horses is the breed-dependence of the attritional dental wear. A comparison of the dental criteria in different breeds revealed that, in general, the incisor teeth of draft horses and mini-Shetland ponies are more liable to attrition, whereas the incisors of Arabian horses wear more slowly than those of standardbred horses.

A variety of other factors such as nature and quality of food, environmental conditions, heredity, injury and disease can also influence dental wear. It is therefore important that equine clinicians do not claim levels of accuracy that are unjustifiable. As it is impossible to assign specific ages to each dental feature, accuracy of age estimation in certain individuals can be very low.

Therefore it is advisable to make written records at the time of examination to show the dental features upon which the age estimate was made. In some countries there have been legal guidelines established to distance veterinarians from trying to state the age of a horse from solely dental findings. In case of insurance policies or legal questions the veterinarian should indicate explicitly that he is providing an 'estimate of age.' It is also advisable that the incisor tables are photographed. When necessary, the pictures can be submitted to others for a second opinion and can be stored with appropriate identification for further use as well.[8]

REFERENCES

1. Ten Cate AR (1998) Physiologic tooth movement, eruption and shedding. In: *Oral Histology, Development, Structure and Function* (5th edn), ed. AR Ten Cate, CV Mosby, St Louis, pp. 289–314.
2. Muylle S, Simoens P and Lauwers H (1999) Age-related morphometry of equine incisors. *Journal of Veterinary Medicine A*, 46, 633–643.
3. Eisenmenger E and Zetner K (eds) (1985) *Veterinary Dentistry*. Lea & Febiger, Philadelphia, pp. 2–26.
4. Kertesz P (1993) In search of Mr Bishop. *Veterinary Record*, 133, 608.
5. Zipperlen W (1871) Over de ouderdomskennis van het paard of de tandleer. In: *Geïllustreerd veeartsenijkundig handboek*. B. Dekema, Utrecht, p. 171.
6. Dupont M (1901) *L'âge du cheval*. Librairie J.B. Baillière, Paris.
7. Frateur JL (1922) *De ouderdomsbepaling van het paard door het gebit*. E. Marette, Brussel.
8. American Association of Equine Practitioners (2002) *Official Guide for Determining the Age of the Horse* (6th edn). Fort Dodge, Iowa.
9. Barone R (1997) Dents. In: *Anatomie comparée des mammifères domestiques. Tome 3* (3rd edn). Vigot, Paris, p. 91.
10. Dyce KM and Wensing CJ (1980) *Anatomie van het paard*. Scheltema-Holkema, Utrecht, p. 14.
11. Willems A (1980) *Ouderdomsbepaling van het paard* (5th edn). Van de Sompele, Oud-Heverlee, p. 14.
12. Habermehl KH (1981) Wie sicher ist die Altersbestimmung beim Pferd? *Berliner und Münchener Tierärztliche Wochenschrift*, 94, 167.
13. McMullan WC (1983) Dental criteria for estimating age in the horse. *Equine Practice*, 5, 10, 36.
14. Walmsley JP (1993) Some observations on the value of ageing 5–7-year-old horses by examination of their incisor teeth. *Equine Veterinary Education*, 5, 295.
15. Sack WO (1994) *Rooney's Guide to the Dissection of the Horse* (6th edn). Veterinary Textbooks, Ithaca, p. 182.
16. Dyce KM, Sack WO and Wensing CJ (1996) *Textbook of Veterinary Anatomy* (2nd edn). WB Saunders, Philadelphia, p. 491.
17. St Clair LE (1975) Teeth. In: *Sisson and Grossman's The Anatomy of the Domestic Animals* (5th edn). WB Saunders, Philadelphia, p. 460.
18. McCarthy PH (1987) Galvayne: The mystery surrounding the man and the eponym. *Anatomia Histologia Embryologia*, 16, 330.
19. Galvayne S (1886) *Horse Dentition: Showing how to tell exactly the age of a horse up to thirty years*. Thomas Murray, Glasgow.
20. CD Equus-Vetstream Ltd, Three Hills Farm, Bartlow, Cambridge CB1 6EN, UK.
21. Muylle S, Simoens P and Lauwers H (2002) A study of the ultrastructure and staining characteristics of the dental star of equine incisors. *Equine Veterinary Journal*, 34, 230–234.
22. Joest E and Becker E (1970) Zähne. In: *Handbuch der speziellen pathologischen Anatomie der Haustiere* (3rd edn), Verlag P Parey, Berlin, pp. 83–315.
23. Torneck CD (1998) Dentin-pulp complex. In: *Oral Histology, Development, Structure and Function* (5th edn), ed. AR Ten Cate, CV Mosby, St Louis, pp. 150–196.
24. Kilic S, Dixon PM and Kempson SA (1997) A light microscopic and ultrastructural examination of calcified dental tissues of horses: III Dentine. *Equine Veterinary Journal*, 29, 206–212.
25. Richardson JD, Cripps PJ, Hillyer MH, O'Brien JK, Pinsent PJN and Lane JG (1995) An evaluation of the accuracy of ageing horses by their dentition: a matter of experience? *Veterinary Record*, 137, 88.
26. Richardson JD, Lane JG and Waldron KR (1994) Is dentition an accurate indication of the age of a horse? *Veterinary Record*, 135, 31.
27. Richardson JD, Cripps PJ and Lane JG (1995) An evaluation of the accuracy of ageing horses by their dentition: can a computer model be accurate? *Veterinary Record*, 137, 139.
28. Hillson S (1986) *Teeth*. Cambridge University Press, Cambridge, p. 183.
29. Marcq J and Lahaye J (1943) *Extérieur du cheval*. J Duculot, Gembloux. p. 11.
30. Misk NA (1997) Radiographic studies on the development of incisors and canine teeth in donkeys. *Equine Practice*, 19, 23.
31. Muylle S, Simoens P and Lauwers H (1996) Ageing horses by an examination of their incisor teeth: an (im)possible task? *Veterinary Record*, 138, 295–301.
32. Muylle S, Simoens P, Lauwers H and van Loon G (1997) Ageing draft and trotter horses by their dentition. *Veterinary Record*, 141, 17–20.
33. Muylle S, Simoens P, Lauwers H and van Loon G (1998) Ageing Arab horses by their dentition. *Veterinary Record*, 142, 659.
34. Muylle S, Simoens P, Verbeeck R, Ysebaert M and Lauwers H (1998) Dental wear related to the microhardness of enamel and dentine. *Veterinary Record*, 144, 558–561.

6 Abnormalities of Development and Eruption

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Introduction

The essentials of the development and embryonic origins of the teeth have been reviewed in Chapter 3. The impact of both genetic and environmental factors may result in the abnormal development of teeth as well as contribute to maleruptions.

The essential forces which result in the penetration of the gum by teeth have been the subject of much theory and experimentation. Physiologic tooth movement may be described in general terms under three headings: (a) pre-eruptive tooth movement made by both deciduous and permanent tooth germs within the tissue of the jaw before they begin to erupt, (b) eruptive tooth movement made by a tooth to move from its position within the bone of the jaw to its functional position in occlusion and (c) post-eruptive tooth movement which maintains the position of the erupted tooth in occlusion while the bones of the jaw and head continue to grow, and also movements that compensate for occlusal wear and/or loss of teeth.[1]

We can recognize the results of pathological changes associated with all three of these physiological categories in foals, in adult horses and in ponies.

Physiology of dental eruption and maturation

Radiographic evidence of enamel formation (secretion by ameloblasts in the tooth bud) within the deciduous cheek teeth can be seen as early as 112 days of fetal life. The ultimate shape of any tooth is orchestrated by the foldings that occur within the ameloblast layer and the subsequent deposition of enamel and contacting dentinal tubules produced by the odontoblasts (Fig. 6.1). During this process there is a large increase in the size of the developing tooth and induced changes within the surrounding tissues to accommodate this increase and the changes in shape. By this process, what will eventually be recognized as the alveolar (tooth) socket assumes its unique profile, a profile that matches the complexity of the folds of the enamel and eventually the reserve crown and tooth roots.[2] Nutrition of all the tissues of the developing tooth is afforded by vessels in the apical 'pulp' and in the maxillary teeth and the incisors from vessels that descend from the coronal surface into the enamel invaginations that are designated enamel (cement) lakes and infundibula (cups), respectively (Fig. 6.2).

There are profound differences between the physiological movements of simple (brachydont) and complex (hypsodont) teeth. In both, however, it should be noted that root formation and maturation continues long after there is penetration of the gum by the erupting tooth. Four possible mechanisms have been described to explain the process of tooth eruption. These mechanisms are (a) root growth, (b) hydrostatic pressure, (c) selected deposition and resorption of bone and (d) the pulling of the periodontal ligament.[1] These four mechanisms require further evaluation for hypsodont (continuously erupting) teeth. Experiments on the continuously erupting incisors of rodents suggest that the forces for eruptive tooth movement reside in the periodontal ligament.[3] The complete absence of true root formation at the time of eruption of a horse's teeth would confirm that root growth, i.e. a 'pushing' of the tooth toward the gum, is the least significant of these four mechanisms in this species. All horse owners and veterinarians are familiar with the mandibular and maxillary changes that occur in young horses and which reflect both the vascular and bone remodeling changes that accompany tooth eruption. They have been referred to as teething bumps or pseudocysts (Figs 6.3 and 6.4).[4] In most cases, such swellings are benign and resolve as the musculoskeletal components of the head enlarge and mature. In some instances however, there may be internal nasal swellings that cause respiratory obstruction or there may be true dental impactions and subsequent periapical pathology (see pp. 121–125).

Developmental abnormalities

Cemental hypoplasia

Cemental hypoplasia is seen in all incisors and maxillary cheek teeth. There is no evidence that it is pathological in incisors, but it may predispose some cheek teeth to endodontic disease (see Chapter 11). It is unusual to see defects of peripheral coronal or reserve crown and root cement, although inflammatory disease associated with periodontal disease may result in lysis of cementum, hyperplasia of cement and the formation of nodules of cement.[5]

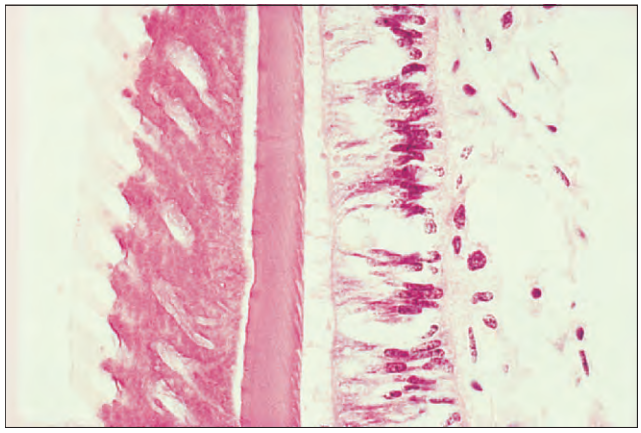


Figure 6.1. Predentin separating odontoblast and ameloblast layers (hematoxylin-eosin, magnification ×950).

The process of cementogenesis within the enamel invaginations of the maxillary cheek teeth can be recognized histologically at least 160 days prior to eruption. These cementoblasts originate from the mesenchymal cells of the dental papilla and they break through the epithelial sheath and come to rest beneath the ameloblast layer. In this way, a collar of cementoblasts forms within the enamel invagination (Fig. 6.2). At first the cement that is formed is sponge- or coral-like in appearance but it continues to mature, becoming more dense as long as the tissues receive a vascular supply. Once the tooth erupts, however, this vascular supply is broken and cement formation in this locus stops.

It has been seen that although all maxillary teeth show some degree of cemental hypoplasia, the 'severity' of the condition cannot be ascertained by studying the occlusal surfaces of the maxillary cheek teeth.[5] Vertical sections of cheek teeth through the enamel lakes demonstrate the true extent of hypoplasia of cementum.[6]

Prior to the detailing of maxillary cementogenesis[6] the condition of cemental hypoplasia had been misnamed as caries[7–9] and infundibular necrosis.[4] The significance of hypoplastic cement within the enamel lakes of the maxillary cheek teeth has been reviewed by a number of authors. In summary it can be said that in most cases the condition is benign. In many cases it can be seen that during mastication, food material can become pressed into the depths of the hypoplastic tissue via the previous vascular pathways (Fig. 6.5). Subsequent microbial fermentation and dissolution of existing cement (caries) is possible. Such a relationship was recognized in some of the earlier German studies of equine dental disease.[10]

Teeth with extensive occlusal exposure of lake cement hypoplasia may exhibit abnormal wear and subsequent 'cupping out,' that is, a concavity of the occlusal surface. Such changes can, if uncorrected, initiate 'wave-mouth' formation. In turn, changes in shape of the occlusal surface may result in stress concentration during mastication and can create of shear forces that lead to fracture of the affected tooth.

As has been stated, for the most part, hypoplasia of cement is benign and the pulp is protected as the occlusal surface is worn away and deeper areas of hypoplasia are exposed. Ultimately the enamel invaginations and the contained cement are worn away (Fig. 6.6).

Oligodontia

The absence of a tooth or teeth (oligodontia) is seen frequently in many abattoir specimens. Tooth absence is usually either the result or the sequel of periodontal or dental disease. In some cases, however, a developmental abnormality may have occurred that resulted in the failure of the formation of a tooth bud, hence no tooth forms. When a single tooth is missing there will be movement of adjacent teeth so that the result will be a shortening of the mesial–distal length of the arcade rather than a gap in the dentition. Such a shortening will cause abnormal occlusion and changes in wear across the teeth. Some cases may require extraction of the 'extra' tooth or teeth in the normal arcade or, in the case of incisor teeth, attention will need to be given to the bite alignment of the incisor arcades (Fig. 6.7).

Dental dysplasia and oligodontia were reported in a thoroughbred colt that was monitored from birth to 28 months of age.[11] Serial radiographs revealed the absence of 708, 808, 308 and 408. 106, 206 and 207 were positioned in an abnormally rostral location. 309 and 409 became progressively more caudally angulated. Teeth 310, 311 and 411 had an abnormal coned appearance (Figs 6.8 and 6.9).

The complete dental complement for the horse is 44 teeth. We recognize that canine teeth are absent in females and that 305 and 405 (mandibular wolf teeth) are rarely seen. Such oligodontics are normal in the horse.

Polyodontia

Polyodontia, or extra teeth, may be the result of the splitting of developing tooth buds from trauma, for example fractures, tooth avulsions or as a result of a developmental abnormality in which extra tooth germs are budded from the dental lamina. Traumatic division of tooth buds leads to the formation of misshapen, malformed and often misplaced, malerupted teeth or tooth components. These are treated by extraction.

Single extra teeth, for example seven incisors or seven cheek teeth in a single dental arcade will result in occlusal abnormalities similar to those that are seen in cases of oligodontia.

Cases have been described, however, in which there is true duplication of all teeth in an arcade, for example upper or lower incisors or permanent molars. Such conditions usually result in malocclusions, periodontal disease and difficulty in chewing. Early extraction of extra teeth may restore normal occlusal contact but, in fact, most cases of true polyodontia have been recorded from terminal subjects or from specimens that have been collected and for which no clinical information was available.

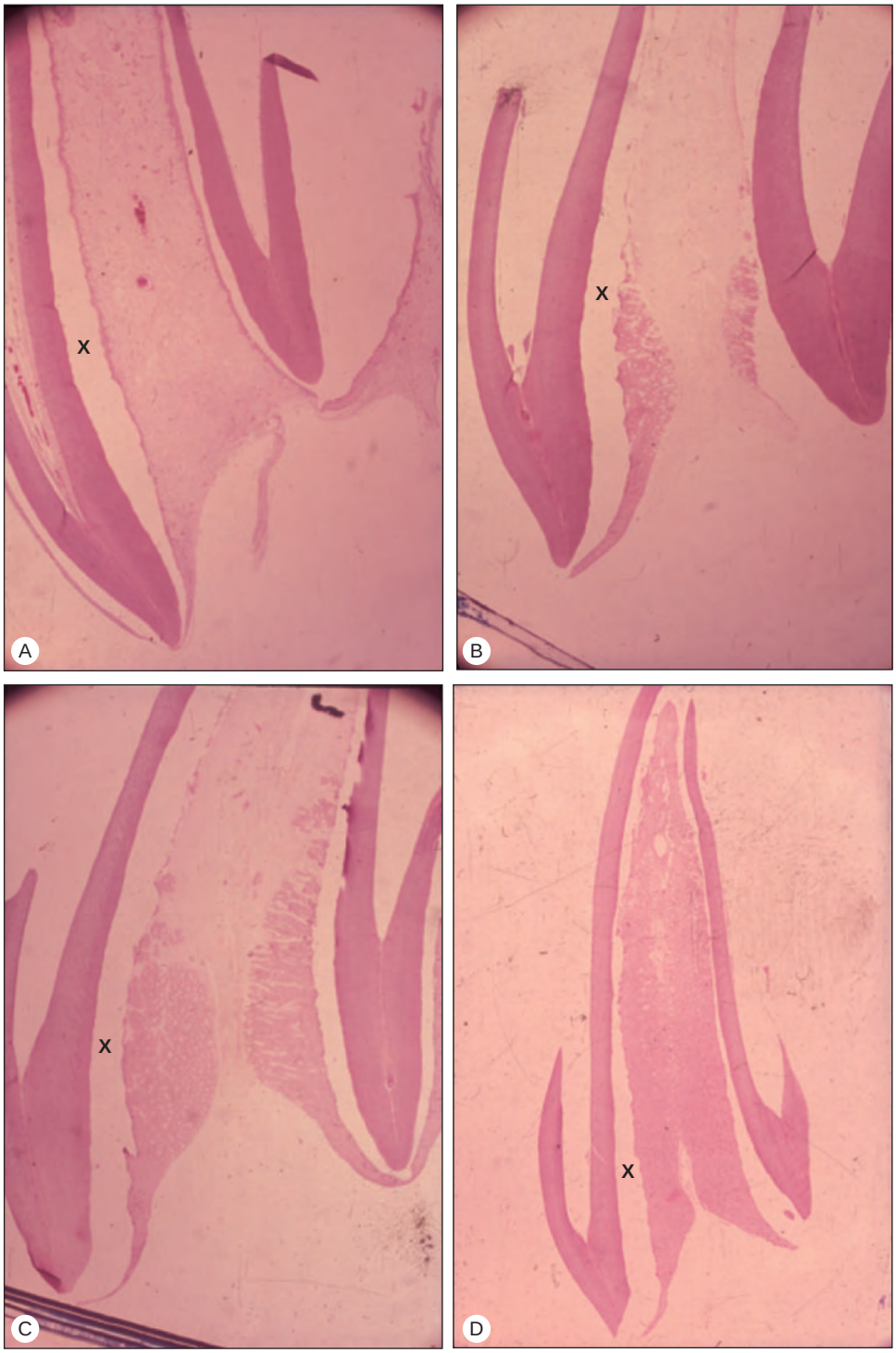


Figure 6.2. Cementogenesis in the maxillary molar enamel lakes: (A) 240-day fetus (magnification ×8.25); (B) 270-day fetus (magnification ×8.25); (C) 300-day fetus (magnification ×8.25); (D) 49-day neonate (magnification ×4.25). X = enamel lost by decalcification.

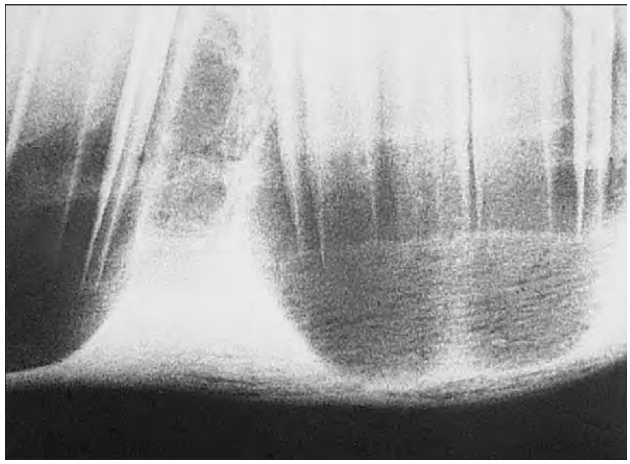


Figure 6.3. Radiograph of erupting 308 illustrating hydrostatic and vascular changes associated with tooth eruption in the horse (eruption cyst).





Figure 6.4. (A) and (B) Mandibular swellings associated with eruptions of permanent cheek teeth (from Tremaine WH (1997) *In Practice,* vol. 16 no. 4, with permission of the editor).

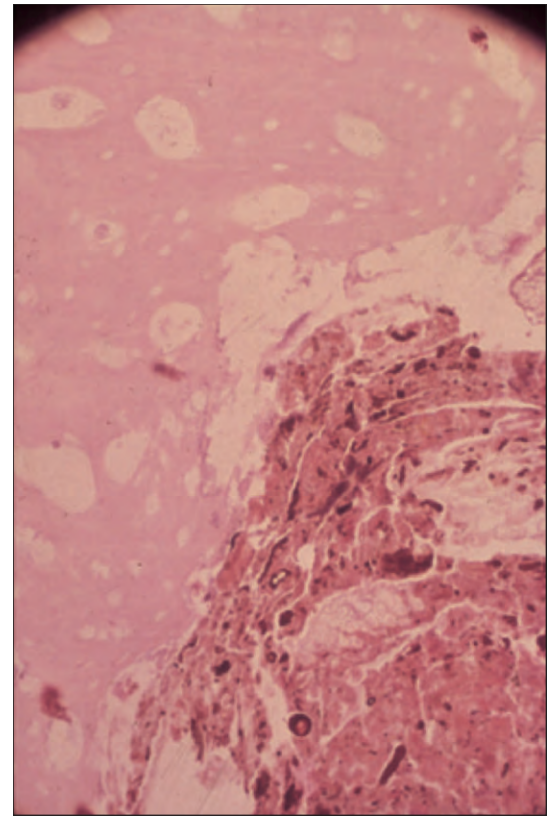


Figure 6.5. Food debris within hypoplastic cementum of maxillary tooth enamel lake (decalcified hematoxylin-eosin, magnification ×350).

Enamel hypoplasia

Failure of enamel formation is a factor in numerous dental tumors. Partial failure, enamel hypoplasia, may be the result of the impact of teratogenic drugs or may be idiopathic. In appearance, there are defects in the teeth in which not only is enamel absent, but there is a defect in coronal cementum. The condition is not of any clinical significance.

Brachygnathia (parrot mouth)

In this condition, the lower jaw is shorter than the upper jaw leading to an overjet or overbite (depending on severity). In most cases, the defect is limited to incisor malocclusions,

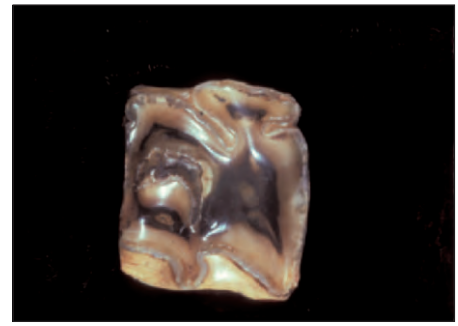


Figure 6.6. Attrition of cheek tooth with loss of enamel lakes – note distal lake replaced by dentin at the bottom of the infundibulum.



Figure 6.7. Incisor malalignment caused by absence of 403.

that is, the defect is a shortening of the rostral component of the mandible and a shortening of the interdental space, and there is often only a minor malocclusion of the cheek teeth (Fig. 6.10). The condition has been the subject of much discussion as to its etiology. For example, is there genetic control? In some countries and in some breeds there are restrictions on the use of stallions for breeding (e.g. Thoroughbreds in the UK and Quarterhorses in the USA). Many people are familiar with particular stallions who appear to throw a high number of 'parrot-mouthed' offspring (see Chapter 17). It is unusual for parrot-mouthed individuals to have difficulty feeding or grazing except for those cases in which the lower incisors impact and start to lacerate the palatine mucosa. Parrot-mouthed horses tend to develop incisor-related periodontal disease and an exaggeration of rostral hooks on 106 and 206 and caudal hooks on 311 and 411. Continuous eruption of the mandibular incisor teeth in severely parrot-mouthed horses results in compression of these teeth into the palatine mucosa. The result may be pressure ulcers and sores that



Figure 6.8. Lateromedial radiograph of the left maxilla and mandible taken at post mortem (from Ramzan *et al.*,[11] with permission, *Equine Veterinary Journal*).

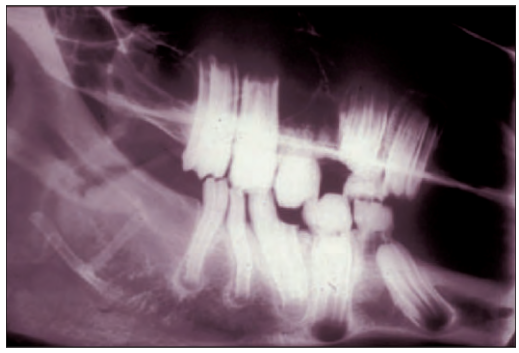


Figure 6.9. Lateromedial radiograph of the right maxilla and mandible taken at post mortem (from Ramzan *et al.*,[11] with permission, *Equine Veterinary Journal*).

create pain for the horse and may interfere with mastication. Such overgrowths need to be reduced by removal of incisor erupted crowns. This is best done under sedation and with the use of a mouth gag placed in the intradental space to give unencumbered access to the incisors. Reduction may be achieved by marking the line to be cut with a diamond disk and the use of incisor cutters or by grinding down with a power disk driver (see Chapter 16). In recent years, techniques have been described to limit the extent of this congenital and developmental condition. These may use orthodontic devices to inhibit growth of the premaxilla or bite plane devices to both inhibit premaxillary extension and encourage mandibular extension (see Chapter 17).

Prognathia (sow mouth, monkey mouth)

This condition is seen less commonly than parrot mouth. Small horse breeds, ponies and miniature horses form the group in which moderate to severe underbites may be seen (Fig. 6.11). In severe cases there may be nasal or nostril deformity as a result of shortening of the premaxilla and maxillary bones. Consequently, nostril collapse, obstruction and stertorus breathing may occur.

Owners should be advised to change breeding strategies if their programs result in a number of foals with underbites. As with parrot-mouthed animals, individual cases will require regular attention to incisor alignment and incisor (upper) reduction to avoid mucosal pressure sores on the mandibular diastema.



Figure 6.10. Parrot mouth in a 4-year-old Thoroughbred.

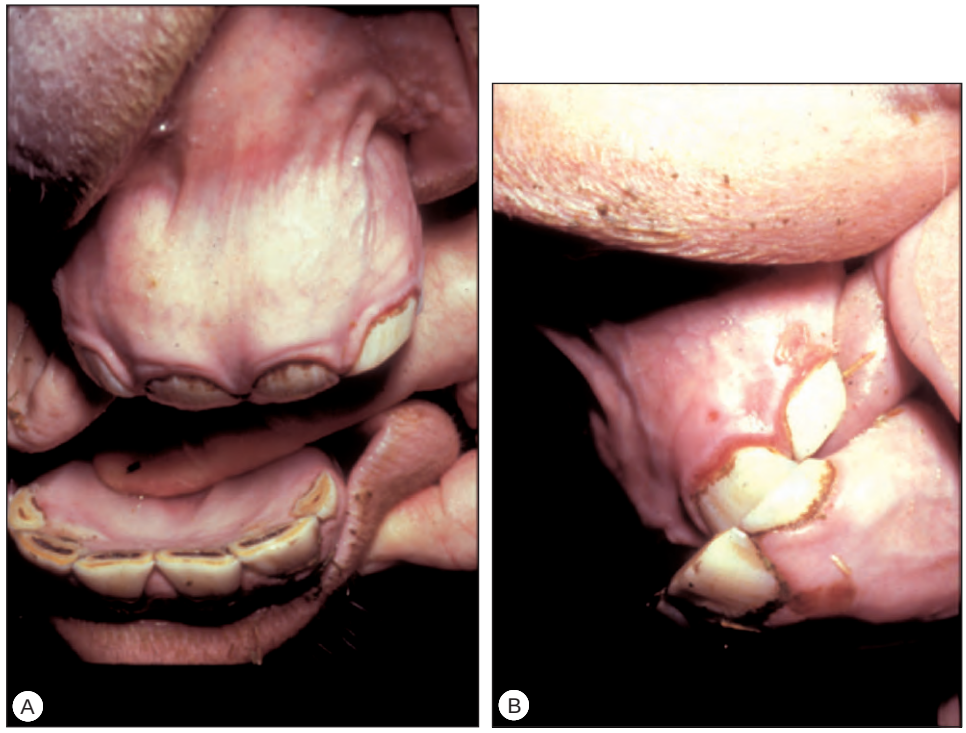


Figure 6.11. Monkey mouth: (A) rostral and (B) lateral views.

Campylorrhinus lateralis (wry nose)

This is a condition in which there is a major developmental (and congenital) deformity of one side of the rostral region of the face (Fig. 6.12). There is a dysplasia of one side of the maxilla and premaxilla so that there is lateral deviation of the nose to the dysplastic side. Close examination will often also show an enhanced concavity of the palate in the area rostral to the cheek teeth. The deformity also affects the congruity of the nostrils and the nasal septum with, in some cases, severe nasal obstruction as a result, caused by septal deviation.

The condition occurs occasionally in all breeds and may be related to fetal malpositioning. There seems to be a higher incidence in the Arabian breed and it is suggested that in this breed at least there may be a genetic cause.

The condition can be treated by facial reconstruction involving a frontal plane division of the face at a line from the first cheek tooth. Incisions are made to elevate the palatine mucosa, periosteum and the palatine arteries. The palatine maxillary bones and nasal septum are then divided. The nose may then be realigned and stabilized with external fixators. Postoperatively, a tracheostomy is essential and follow-up rhinoplasty procedures may be required to alleviate residual nasal obstruction (see Chapter 17).



Figure 6.12. Wry nose in a 3-week-old Arabian foal.

Rudimentary teeth

Teeth that do not assume normal maturity and shape are defined as being rudimentary. In mares canine teeth are usually absent; this is a sex-linked genetic control. In some



Figure 6.13. 'Molarized' wolf teeth in a zebra (from WR Cook with permission).

cases, however, there is partial formation of a simple structured tooth or teeth – a single or pair of rudimentary canines. They are usually small, from 2–6 mm in circumference and some 3–8 mm in length. They are sometimes unerupted and may only be detected by palpation through the overlying mucosa.

It is a common custom for maxillary wolf teeth (105 and 205) to be extracted. Consequently, data relating to the prevalence of the rudimentary first cheek teeth, gathered from the examination of necropsy specimens, is likely to give a false incidence (13 per cent).[6] An examination of thoroughbred fetal skulls demonstrated that only 20 per cent contained wolf teeth.[6] Examination of yearling standardbreds revealed an incidence of 60 per cent. No explanation can be afforded for these differences. In other equine species (e.g. the plains zebra), the incidence is nearly 100 per cent (WR Cook, personal communication, 1974). In the plains zebra it is not uncommon to find that the maxillary wolf teeth are large and often make occlusal contact with the first mandibular cheek teeth (306 and 406) and so are still functional in mastication (Fig. 6.13). Mandibular wolf teeth occur only rarely in *Equus caballus* and are also rare in other equid animals (e.g. zebras).

Abnormalities of eruption

The essential forces which result in the penetration of gums by teeth may be described in six anatomical stages.[10]

Stage I: preparatory stage (opening of the bony crypt)
Stage II: migration of the tooth toward the oral epithelium
Stage III: emergence of crown tip into the oral cavity (beginning of clinical eruption)
Stage IV: first occlusal contact
Stage V: full occlusal contact
Stage VI: continuous eruption and movement.

The mechanism for tooth eruption in the horse has been reviewed and those components based on the selected deposition and resorption of bone, the involvement of vascular and intraosseous hydrostatic pressure changes, and the pulling of the periodontal ligament are the most important. Stage II is facilitated by the lysis of bone and tissue on the oral aspect of the erupting tooth – the eruption pathway. Abnormalities of eruption may occur at any stage and may be traumatic, genetic, viral or teratogenic. Evidence from other species suggests that the formation of the eruption pathway is orchestrated after the formation of the complete enamel anlage of the developing tooth.

Trauma to developing teeth and surrounding bones may lead to malorientation of the tooth bud and subsequent misplacement or maleruptions (Fig. 6.14). Whenever possible, care should be taken when repairing fractures or avulsions that involve teeth to try and avoid damage to the developing permanent dentition when placing the transfixing pins of external fixators or other osteosynthetic devices.

Reference has already been made to the vascular and hydrostatic forces that accompany tooth eruptions. The general pattern of the sequence of eruption of mammalian teeth is based on a mesial to distal progression (Table 6.1). This sequence applies to both deciduous and permanent teeth, but it can be seen that there is an overlap in the time sequence so that permanent teeth 108, 208, 308 and 408 (premolar 4s or third cheek teeth) have a tendency to be crowded because they erupt into a potentially crowded space that is made by the 07 and 09 teeth (premolar 4s and first molars). Examination of lateral radiographs of erupting and maturing teeth within the mandibles and maxillae show the direction of eruption to be more curved for the caudal teeth



Figure 6.14. Incisor maleruption and malposition subsequent to mandibular symphyseal fracture and fracture repair.

Table 6.1. Eruption Sequence of Cheek Teeth		
Cheek tooth number	Mammalian eruption sequence	*Equus caballus*
(100, 200, 300, 400)		
06	4	3
07	5	4
08	6	6
09	1	1
10	2	2
11	3	5

and straighter for the more rostral teeth. The center of the circle that fits the curve of eruptions is the center of the temporomandibular joints.

Dental impactions

It should be appreciated that the sequences of the anatomical stages of eruption, particularly stages II–IV, are mutually dependent. Therefore if the eruption pathway is not complete, if there is a minor change in position of the erupting permanent tooth in replacing its deciduous predecessor or if the space is too small, then there is the possibility of disease processes resulting from this impaction.

In the case of incisor teeth, caudal eruptions of permanent incisors are seen quite commonly (Fig. 6.15). In most cases these positional changes are corrected spontaneously when the deciduous tooth is shed. In other cases, the displacement may be severe enough to warrant extraction of the deciduous incisor (seen particularly with 302 and 402) and even grinding down the mesial and distal margins of the adjacent teeth. Once a space has been created the normal pathway of eruption is reduced and the permanent incisor is able to move rostrally into its correct location. Such anomalies are more frequently seen in the mandibles and involve 302 and 402.

In horses (stallions and geldings) there is often a 6–8 month delay between the eruption of the first and of the second of a pair of canine teeth. This may result in the formation of an inflammatory fibrous reaction over the unerupted crown of the impacted tooth. These swellings may be painful on palpation or cause abnormal reactions to bit contact. The clinician has two options to relieve the problem of an impacted canine. Under sedation and local analgesia the overlying tissue should be divided in a cruciate pattern, using a scalpel and an elevator, thus enhancing the eruption pathway and enabling entry of the tooth into the mouth. If there is continued failure to erupt the second choice is to extract the tooth (see Chapter 18).

Similar local reaction is seen occasionally over the crown of an impacted or misplaced wolf tooth. In these cases extraction of the tooth is the recommended course of treatment.

The most frequent sites of cheek teeth impaction are seen associated with the eruptions of the 8s (i.e. 108, 208, 308 or 408). The result is an expansion of the 'eruption cyst' and, in many cases, a break-out of the inflammatory process to periapical structures. This may result in dental fistula formation, periradicular disease or sinusitis depending upon the location of the tooth involved.

Impaction of 108 and/or 208 may result in palatal displacement of the affected permanent tooth or teeth (Fig. 6.16). This will result in an irregular arcade and the formation of a buccal cheek pocket or pouch. As food is chewed it will spill laterally into this space and there will be local periodontal disease. Treatment is based on amelioration of the pocketing. It can be accomplished by grinding or sloping the distal margins of 107 or 207 and the mesial



Figure 6.15. A common form of incisor maleruption in a 2.5-year-old standardbred horse.



Figure 6.16. Palatal displacement of 108.



Figure 6.17. Dental caps on right maxillary arcade.

margins of 109 or 209. In this way, the shape of the pocket is changed and food accumulation is avoided. It may, in some cases, be necessary to extract the misplaced tooth (108 or 208).

The most frequently seen disease associated with dental impactions is, as has been stated, the effect of impacted 8s. It is the author's opinion that this problem is the single most important cause of apical osteitis and the subsequent development of mandibular dental fistula formation (see Chapter 10).

Delayed eruptions, impactions and maleruptions of incisors and cheek teeth occur more commonly in ponies and miniature horses. Consequently the eruption times of teeth as quoted in the literature may not apply in these groups.

Deciduous caps

As the deciduous teeth are worn away and permanent teeth erupt to replace them, the occlusal remnants of the deciduous teeth are referred to as deciduous caps (Fig. 6.17). The caps are normally shed as part of the eruptive process and do not cause problems. In some cases however, they may become unstable, change position and result in oral or buccal discomfort. Loose caps and displaced or rotated caps should be removed. They may be elevated with an elevator, or even the end of a screwdriver, and removed. It is important to keep in mind that early removal of caps will stop cementogenesis within the enamel lakes of the maxillary cheek teeth and thereby enhance hypoplasia of cementum at these sites.

Summary

Abnormalities of dental development and eruptions occur quite commonly in the horse and result in a wide range of clinical signs and symptoms.

REFERENCES

1. Ten Cate AR (1989) Physiologic tooth movement, eruption and shedding. In: *Oral Histology, Development, Structure and Function* (3rd edn), ed. AR Ten Cate, CV Mosby, St Louis, pp. 275–298.
2. Kupfer M (1937) *Tooth Structure in Donkeys and Horses*, Gustav Fischer, Jena, pp. 1–103.
3. Berkovitz BKB (1975) Mechanisms of tooth eruption. In: *Applied Physiology of the Mouth*, ed. CLB Lavelle (1st edn), J Wright, Bristol, pp. 53–72.
4. Baker GJ (1971) Some aspects of dental radiology. *Equine Veterinary Journal*, 3, 46–51.
5. Baker GJ (1970) Some aspects of equine dental disease. *Equine Veterinary Journal*, 2, 105–110.
6. Baker GJ (1979) A study of equine dental disease. PhD Thesis, University of Glasgow, pp. 36–38.
7. Colyer F (1931) *Abnormal Conditions of the Teeth of Animals in Their Relationship to Similar Conditions in Man*. London, The Dental Board of the United Kingdom, pp. 36–38.
8. Hofmeyer CFB (1960) Comparative dental pathology (with particular reference to caries and paradontal disease in the horse and dog). *Journal of South African Veterinary Medical Association*, 29, 471–480.
9. Honma K, Yamakawa M, Yamouchi S and Hosoya S (1962) Statistical study on the occurrence of dental caries in domestic animals. 1. Horse. *Japanese Journal of Veterinary Research*, 10, 31–36.
10. Beck E (1939) Treatment of dental disease in the horse. *Deutsche Tierarztt*, 6, 29–31.
11. Ramzan PHL, Dixon PM, Kempson SA *et al.* (2001) Dental dysplasia and oligodontia in a Thoroughbred colt. *Equine Veterinary Journal*, 33, 99–104.

7 The Management of Oral Trauma

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Oral anatomy

The mouth is the most rostral portion of the alimentary canal. It is bounded laterally by the cheeks, dorsally by the hard and soft palates and ventrally by the mandible, tongue and mylohyoid muscles. It is a long cylindrical cavity and when closed is almost entirely filled by the tongue and teeth. The oral cavity communicates caudally with the oropharynx through the isthmus faucium.

The mucous membrane of the mouth is continuous at the margin of the lips with the skin and caudally with the mucosa of the oropharynx. It is of squamous type with multiple small papillae, which are the openings of the ducts of the labial glands. The lips are muscular folds surrounding the orifice of the mouth. The angles of their union are called the commissures and are situated at the level of the first premolar cheek teeth.

The cheeks are muscular in nature and the gums are composed of dense fibrous tissue united intimately with the periosteum of the mandible, maxilla and with the dental structures which it surrounds.[1] Motor innervation of the cheeks and lips is provided by the seventh and oral sensation by the fifth cranial nerves.

The hard palate is bounded rostrally and laterally by the dental arcades. Although its mucous membrane is smooth there are 18 curved transverse ridges along its length.[1] The blood supply to the mouth is well developed and formed from the facial and buccinator arteries. There is a rich venous plexus beneath the mucosa of the hard palate supplied by the palatine arteries and veins.

The tongue is a muscular structure situated in the floor of the oral cavity between the horizontal rami of the mandibles and is supported by a muscular sling formed by the mylohyoid muscles. Caudally it is attached to the hyoid apparatus. It is covered with a squamous epithelium and the dorsal surface is covered with a variety of papillae. On either side of its root sits a dense aggregate of lymphoid tissue, the lingual tonsils. The blood supplied to the tongue is by the lingual and sublingual branches of the external maxillary artery and its corresponding veins. Motor supply to the tongue is by the twelfth cranial nerves. Sensation to the rostral two-thirds of the tongue is provided by the fifth and seventh cranial nerves and to the caudal one-third, by the lingual branch of the ninth cranial nerve.[1]

There are three pairs of salivary glands which have ducts entering the oral cavity. The parotid ducts enter the mouth via papillae situated approximately at the level of the fourth upper premolar teeth (108 and 208). The mandibular ducts open into the floor of the mouth at the level of the lower canine teeth (304 and 404) via flattened papillae, the sublingual caruncles. The sublingual glands enter the oral cavity by a series of approximately 30 ducts in the sublingual fold. Other smaller salivary glands are found throughout the mouth.[1]

Oral examination

Injuries to the oral cavity can be assessed by visual inspection of the mouth. Obvious external injuries to the lips and cheeks are readily assessed; however, lesions within the oral cavity itself can be more difficult to evaluate. Examining the mouth can usually be performed by gently grasping the tongue and rotating it within the mouth at the level of the interdental space.[2] Great caution should be observed if there is any possibility of a lingual injury. A pen torch may be used to illuminate the oral cavity, but a good head lamp is much better as it allows the clinician the freedom to use both hands, enabling careful palpation of dental arcades, gums, tongue, cheeks and other oral structures. Great caution must always be observed when inserting a hand into a horse's mouth as serious injury to the examiner may result from Haussmann's equine teeth. A much better and safer option is the use of an oral speculum such as a Haussmann's gag, or a variety of similar types of specula which are readily available. Although such a speculum can be used in accommodating patients without need for sedation, it is preferable when examining oral injuries to sedate the patient to reduce the risk of operator injury and to facilitate the accurate assessment of the extent of the injury. In the presence of a severe oral injury the pain associated may preclude use of a metal speculum, even in a heavily sedated patient, and necessitate an examination under general anesthesia. A speculum permits a safe means of examining even the most caudal part of the oral cavity, although accurate visualization may be more difficult because of the intrusion of soft-tissue structures in the area.

An endoscopic examination of the nasal passages and nasopharynx is of great value in assessing full thickness

defects of the hard or soft palate and the identification of most pharyngeal foreign bodies which protrude through the intrapharyngeal ostium. An endoscopic examination of the oral cavity may be carried out in a sedated horse with a speculum in place. However, the risk of instrument damage means that extensive intra-oral endoscopy should probably be carried out under general anesthesia. The endoscopic light allows not only inspection of an injury, but facilitates the lavage of blood and debris from the area, improving the view. In addition the illumination it affords may permit the surgical repair of some otherwise inaccessible wounds.

Radiographic views of the head are important in assessing the extent of oral injuries when damage to bones or teeth is suspected. Lateral or oblique radiographic views obtained with the horse in the standing position are helpful. Intra-oral views of the mandibular or maxillary incisor arcade may be of particular value in demonstrating injuries to these areas. Such views require the horse to be sedated heavily and it may be preferable with more fractious patients to carry this out in the anesthetized horse (see Chapter 14).

The use of oral barium sulfate may permit the identification of an oronasal fistula and plain radiographic views should demonstrate the presence of metallic foreign bodies within the tongue, cheeks, or gums.[3,4] It should be remembered that artifactual shadows can be created by restraining devices such as head collars. A rope halter is probably the most satisfactory means of restraining a horse for radiographic examination of the head. Assessing functional disturbances of swallowing requires a dynamic study which is best achieved by using image intensification or fluoroscopy. Barium sulfate can be used as a contrast agent either mixed together with food or water, or administered as a suspension on its own.

Oral trauma

A variety of injuries affecting the oral cavity are commonly encountered in equine practice. Lacerations of the lips may be associated with wire or bit injuries and occasionally are caused by kicks or other forms of direct trauma (Fig. 7.1). Horses are prone to grasping fixed objects such as doors, mangers, or buckets and this may lead to fractures of the rostral mandible or maxilla, usually associated with avulsion of incisor teeth. Occasionally the incisor teeth may be damaged without significant injury to the surrounding bone and this is particularly likely when deciduous teeth are involved in such injuries.

Injuries may occur to the interdental space where the bit sits. This may be the result of overzealous handling by a rider or by the use of strong restraining devices such as a chiffney. Such injuries are often accompanied by ulceration of the gum just rostral to the premolar teeth and in some cases damage to the underlying bone may result in sequestration (Fig. 7.9). More severe fractures of the mandible or maxilla may occur at this level as a result of direct trauma.

Injuries to the tongue are less common but may also follow overzealous restraint using a chiffney. Severe laceration and even transection of the tongue is not unknown under such circumstances. Perhaps more commonly an injury to the lingual frenulum may be the consequence of grasping the tongue during an oral examination, or if used as a method of restraint for fractious patients. There was one famous account of a farrier`s assistant who literally pulled a horse`s tongue out of its mouth while restraining the animal for shoeing! Less severe injuries can be caused by stable hooks or clips, twisted or loose deciduous premolar teeth, or by foreign bodies in the feed.

Injuries to the hard or soft palate are less common but may follow the incorrect or careless use of dental instruments, for example during wolf tooth extraction. Iatrogenic soft-palate damage may follow surgical treatment of horses for epiglottic entrapment or dorsal displacement of the soft palate.

Injuries to salivary structures are rare. However, trauma to the face may result in damage to the parotid salivary gland or duct with consequent development of a salivary facial fistula. Such animals can produce a spectacular jet of saliva during eating!

The oral environment and healing of oral injuries

The oral cavity is exposed to the external environment and is bathed in saliva. Saliva is composed of mucus and serous fluids containing electrolytes and proteinaceous enzymes, for example amylase. It facilitates mastication and deglutition. Saliva also contains glycosaminoglycans and glycoproteins, which are responsible for its lubricant characteristics. Saliva is secreted in large volumes (50 ml/hour from a single parotid gland) as a result of mastication and its production is controlled by the autonomic nervous system.[5] The mouth is frequently in contact with food material and subjected to the movements of mastication. In the wild, horses graze almost continuously except when they are asleep. There are large numbers of bacteria within the oral cavity, many of which are anerobic. All these factors have a considerable bearing on the effects of injury to the mouth and to the management of oral trauma.

Many minor lacerations to the lips, cheek and tongue will heal by second intention without the need for surgical reconstruction, because of the excellent oral blood supply. However, in certain situations surgical reconstruction of oral structures is indicated.[6–8] Typically, injuries involving complete laceration of the lips, cheeks or tongue are suitable candidates for repair, as second intention healing often produces cosmetically and functionally unacceptable results.[9] Management of such injuries should involve careful wound preparation with particular attention given to the removal of foreign material. In most cases oral lacerations quickly become filled with food as well as clotted blood.

This material should be first flushed away to allow a more accurate assessment of the extent of the injury. Localized infections may subsequently produce necrosis at the site and in some cases bone fragments may become sequestrated. Careful debridement of the wound must be performed. If the wound is extensive or inaccessible in the standing horse, a general anesthetic should be administered to ensure that appropriate wound assessment and management can be carried out. Removal of all necrotic material and debris is essential, producing healthy tissue margins which bleed freely. The use of pulsating lavage (i.e. Water Pik system) has been recommended as it is very effective in removing debris without creating further trauma.[10] When significant injury to the lips or cheeks occurs, the wound should be closed in layers.[7,8] At the very least, separate skin and mucosal closure is required. In most cases, a third layer which incorporates the muscular tissue and fascia should also be repaired using absorbable suture material. Tissues should be reconstructed with accurate anatomical apposition of the layers and it is preferable to use non-absorbable suture material or stainless steel staples in the skin.

Following repair, care must be observed in preventing self-mutilation by the horse. While most horses seem unbothered by sutured facial wounds, others seem to find such wounds irritating. In these circumstances cross-tying, the use of a muzzle or even long-term sedation can be helpful in preventing failure of the repair or worsening of the original damage.

The management of horses with facial trauma

A horse with major facial trauma should be assessed immediately for life-threatening respiratory obstruction, nasal hemorrhage and any other indication of major dysfunction such as neurological or ocular disturbance. Obviously maintenance of a clear airway must be a priority and insertion of an emergency tracheotomy tube should be carried out without delay if required. Most nasal hemorrhage associated with facial injury will cease without need for particular action. Secondary infection of the paranasal sinuses is common following facial fracture and appropriate antibiotics should be administered, and sinus lavage may also be of benefit as part of any treatment regime.

Multiple radiographic projections should be obtained in horses with major facial wounds to assess the extent of osseous or dental injury, the presence of radiodense foreign bodies,[3,4] and before any attempt is made to repair the overlying soft tissues.

Consideration of tetanus prophylaxis, insurance status and discussions with the owner or agent regarding the likely prognosis for restoration of function, are clearly priorities that must be addressed before undertaking any major surgical treatment, or the administration of a general anesthetic to a patient. However, sometimes it may not be possible to make an accurate assessment of the full extent and implications of the injury until the horse has been anesthetized and inspection of more inaccessible structures carried out. This should be explained in clear terms to owners, agents and insurers, whenever they are involved, to minimize the risks of misunderstanding or even litigation at some later date. Consultation with appropriate specialists such as an ophthalmologist or neurologist may be appropriate before any treatment is undertaken.

While the treatment of facial and oral trauma follows the general principles of wound and injury management in other anatomical areas, injuries to mouth, nose, and eyes in particular seem to be associated with greater client concern and feelings of anthropomorphism, than with injuries elsewhere. The clinician must therefore be prepared to respond to questions about cosmetic as well as functional outcome in such patients.

Treatment of specific soft tissue injuries

The lips and cheeks

Injuries to the lips and cheeks are readily treated by suturing after preparation of the wound. If the wound is extensive and involves all the layers of the lips, the horse should either be sedated heavily or given a general anesthetic. The wound



Figure 7.1. A severe laceration to the upper lip that extends into the oral cavity. This a full thickness laceration which should be repaired in layers.



Figure 7.2. Repair of the oral cavity using a continuous suture of 4 metric polyoxanone, same case as in Fig. 7.1.

should be repaired as accurately as possible in layers (Figs 7.1–7.5).

It is preferable to repair the oral mucosa first and this should be done with simple interrupted or a continuous appositional suture of absorbable material such as polyglactin 910 or polydioxanone (Fig. 7.2). Knots can be tied within the oral cavity. In most cases a second layer which should incorporate the muscular and fascial tissue should be carried out using the same material (Fig. 7.5A). Lavage of the incision with polyionic fluid containing soluble penicillin at this stage will assist in minimizing the risk of wound sepsis. The skin of the lips can then be repaired using non-absorbable suture material such as monofilament nylon, sheathed polyamide or stainless steel skin staples. In small lesions the use of a simple interrupted pattern is preferred. However, with more extensive defects simple interrupted sutures should be alternated with vertical mattress sutures to assist in relieving the tension on the wound and therefore reducing the risk of subsequent dehiscence. If extensive cheek laceration has occurred, great care must be taken in determining whether the parotid salivary duct is involved. If this structure has been damaged its wall may be repaired to prevent the development of a facial salivary fistula. Similarly, extensive facial wounds may have involved injury to branches of the facial nerve. Major facial nerve injuries carry a poor prognosis and may result in permanent disability.

When the edge of the lip is involved, care should be taken to restore complete function by accurate repair of the orbicularis oris muscle. The close proximity of the skin, mucosa and musculature at this site potentially creates excessive movement at the suture line, particularly during prehension. To minimize this the skin and mucosa should be separated from the adjacent musculature at the edges of the wound[7,8] (Fig. 7.4).



Figure 7.3. An extensive laceration of the lower lip which also has a full thickness defect through the left cheek into the mouth.

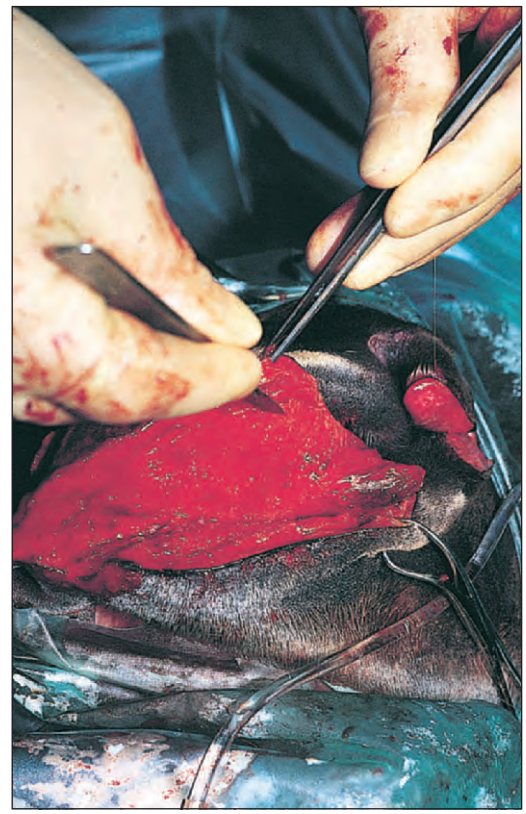


Figure 7.4. Separating the skin from the underlying musculature.

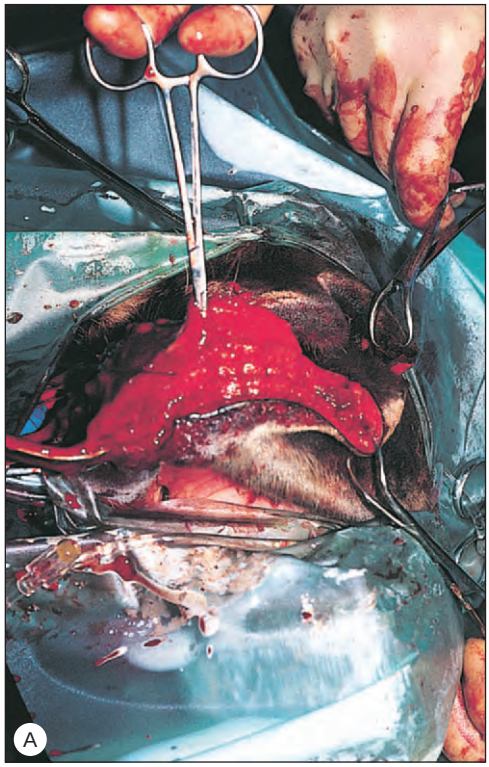




Figure 7.5. (A) Repair of facial musculature using a continuous suture of 4 metric polydioxanone. (B) Although the lesion was repaired in layers, partial wound dehiscence resulted in an orofacial fistula. This was successfully repaired by a second operation.

This facilitates closure in layers and reduces the incidence of dehiscence. The wound edges should be aligned accurately to ensure healing is satisfactory and to minimize the risk of subsequent dehiscence and consequent disfigurement, which might affect function.

Should the repair of a lip or cheek laceration subsequently dehisce, the wound may be left to heal by second intention. However, if extensive defects develop (Fig. 7.5B) an attempt should be made to carry out secondary repair of the wound. It may be necessary to use tension-relieving devices such as a quill of polythene tubing from a giving set for fluid administration. These are incorporated into vertical mattress sutures to protect the wound from dehiscence. When carrying out a secondary repair, it is of paramount importance to perform radical debridement of necrotic material down to healthy bleeding tissue. Creation of tissue planes prior to repair may also facilitate healing. In general the prognosis for healing of lacerations to the lips and cheeks is very good. However, in those cases where secondary or tertiary repair is undertaken there may be a cosmetic blemish at the lip margin. Occasionally an orofacial fistula may remain (Fig. 7.5B).

The tongue and oropharynx

Injuries to the tongue mostly involve lacerations caused by damage from bits or restraining devices such as a chiffney. Minor splits to the tongue will heal readily without need for repair. Occasionally a horse is examined which has previously sustained a severe laceration of the tongue which has healed without complication, leaving a large defect in its dorsum or lateral border. Lacerations of the lingual frenulum, which usually occurs when excessive traction is applied to the tongue, need not be repaired and usually heal without complication. However, most severe lacerations involving the body of the tongue are best managed surgically.[8]

The horse should be given a general anesthetic and the wound assessed carefully to ensure tongue viability. A gauze bandage may be used as an effective tourniquet when applied caudal to the wound. Gentle traction to it also allows good exposure of the more caudal parts of the tongue. Glossectomy may be necessary if the tongue tip is considered unviable and removal of tissue up to the level of attachment of the frenulum is unlikely to affect function.[8] Intravenous administration of sodium fluoroscein has been recommended as an aid to assessing lingual viability. The tongue can be examined under a Woods lamp 5 minutes after injection of 4 or 5 g of the compound.[8] Oversewing the lingual body with simple interrupted or a continuous suture of polyglactin 910 or polydioxanone should be attempted after removal of the necrotic tip. Severe lacerations are repaired using simple interrupted and vertical mattress sutures applied alternately (Fig. 7.6). The latter should incorporate a significant bulk of lingual musculature to take up some of the tension and to ensure more satisfactory healing. All dead space should be obliterated if possible. Multiple layer closure may be required. It should be remembered that the tongue is very mobile and the risk of wound dehiscence is significant unless care is taken to align the tongue correctly and to repair the injury accurately. If the injury is not dealt with in the acute situation, a degree of necrosis and wound contamination may occur. In such circumstances all devitalized tissue must be debrided carefully to minimize the risk of wound dehiscence. When placing vertical mattress tension sutures care must be taken to avoid damage to the lingual blood supply. Although this is good, vascular compromise may result in necrosis of the tongue, particularly when the tip is involved. The dorsum of the tongue has a much stronger mucosa than its ventral aspect and suture retention is better in this site. Tension sutures should therefore be placed in this area.[6]



Figure 7.6. This severely lacerated tongue has been severed almost completely. The injury was repaired using simple interrupted sutures of 4 metric polydioxanone alternated with vertical mattress sutures of the same material. The wound healed by primary intention and the horse regained normal use of its tongue.

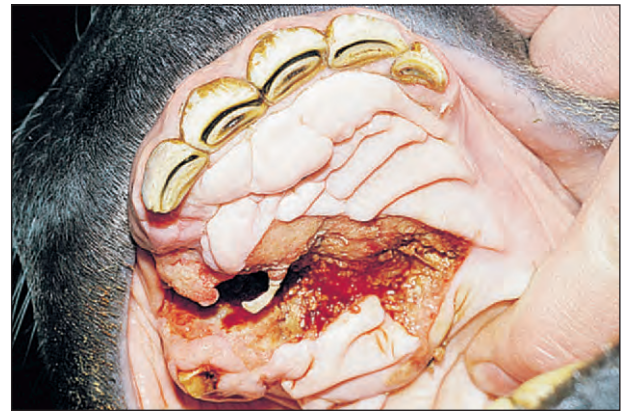


Figure 7.7. This horse sustained severe trauma to the maxilla which resulted in a fracture of the premaxillary bone and laceration of the hard palate. There was direct continuity between the oral and nasal cavities.

Lesions to the base of the tongue and the oropharynx are much more difficult to evaluate and certainly more difficult to repair due to their inaccessibility. However in most circumstances inaccessible wounds will heal well without the need for repair. Daily lavage of the oral cavity with a saline solution may be of value in reducing wound contamination with food material.

Rarely, horses may be encountered which have developed subepiglottal infections or granulomatous abscesses. These are presumably the result of earlier mucosal penetration or an injury which has been undetected. Surgical removal of granulomatous swellings at this site is difficult but may be achieved via an oral approach or through a ventral midline pharyngotomy. This site is also prone to damage by ingested foreign bodies, which are usually twigs or pieces of wood. In most circumstances affected horses show oral discomfort, dysphagia, inappetance, hyperptyalism and occasionally epistaxis. Foreign bodies within the oropharynx can often be detected by nasopharyngeal endoscopy as they frequently protrude through the intrapharyngeal ostium. Foreign bodies are usually retrieved manually with the horse under heavy sedation or general anesthesia. No repair of the mucosal injury is usually necessary. Parenteral antibiotics and non-steroidal anti-inflammatory medication should be administered for several days following removal of foreign bodies from this region.

The hard and soft palates

Injuries to the hard palate are rare but may accompany severe head trauma (Fig. 7.7). In some circumstances there may be an underlying fracture of the palate which involves the palatine processes of the premaxilla and/or the maxilla. Mostly these can be left as open wounds to heal by second intention. However, an oronasal fistula should be repaired surgically. A suspected oronasal fistula may be confirmed by a combination of a thorough clinical and endoscopic examination and by radiography after oral administration of barium sulfate. While such a fistula may resolve by second intention healing, surgical repair should be attempted if the site is accessible. It may be possible to repair the defect by simply suturing the palatal mucosa with interrupted sutures of polydioxanone (Fig. 7.8). If the injury can not be repaired adequately in this manner, it may be possible to close the defect by creating a mucoperiosteal flap, or by making tension-relieving incisions in adjacent portions of the palate. Care should be taken to avoid damage to the palatine blood supply.[6]

Post-repair feeding should be carried out by nasogastric intubation for the first 4 or 5 days to reduce the risk of suture dehiscence.

If the rostral portion of the skull is grossly unstable following a maxillary or premaxillary injury, the fractures may require surgical repair (see Chapter 21). However, it is surprising how frequently horses with this sort of major injury respond successfully to conservative management.

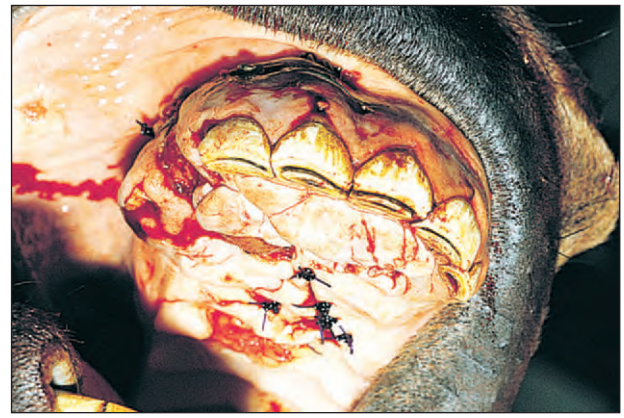


Figure 7.8. This is the surgical repair of the injury illustrated in Fig. 7.7. The fracture has been reduced with cerclage wire after radical debridement of the site. The palate has been repaired partially using simple interrupted and vertical mattress sutures of 4 metric polydioxanone. The horse made an uneventful recovery and the cerclage wire was subsequently removed.

Congenital defects of the hard palate are uncommon and in the author's opinion surgical repair is not indicated. Injuries to the hard palate and more specifically the palatine artery may follow removal of a wolf tooth or repulsion of cheek teeth. In such circumstances moderately severe hemorrhage may occur. Control of hemorrhage by pressure is usually effective. If there is a large enough defect it may be possible to insert some gauze bandage packing.

It may become apparent that an oral wound is not healing as rapidly as might be expected. In some cases healing may be accompanied by an exuberant granulation response. In such circumstances, bone or even dental sequestration should be suspected (Fig. 7.9). Obtaining the relevant radiographic projection may confirm this suspicion. Careful exploration of the wound may produce the sequestrum and after debridement and gentle curettage of the involucrum the wound will usually heal rapidly.

Injuries to the soft palate are uncommon but may follow attempted surgical treatment of the soft palate or adjacent structures, such as in cases of epiglottal entrapment. In an attempt to divide axially the displaced mucosa using a curved bistoury, the soft palate may become inadvertently damaged. Full-thickness injuries to the soft palate inevitably result in the development of an oronasal fistula because of contamination of food and saliva. Surgical repair of such injuries should be attempted as soon as possible after the injury has been identified. Access for surgical repair is very limited. A general anesthetic should be administered and the mouth opened maximally using an oral speculum. Good lighting should be provided and the area may be examined by a fiberoptic or videoendoscope to assess the extent of the injury. Long-handled retractors should be used to depress the base of the tongue and retract the cheeks in order to help in evaluating the injury and in its subsequent repair. Long-handled needle holders, forceps and scissors are also of great help when attempting repair of such an injury. Separating the oropharyngeal mucosa from its overlying musculature will facilitate closure in two layers, enhancing the likelihood of achieving first intention healing. Wherever possible at least two layer closure should be attempted. The musculature and nasopharyngeal mucosa are closed as one, and the oropharyngeal mucosa as the other. The author prefers polydioxanone as a simple, continuous suture for each layer.



Figure 7.9. This is an oral ulcer in the interdental space of the right mandible of a horse which suffered an injury following restraint with a chiffney. A large sequestrum can be seen which was removed. The horse made a complete recovery.

Even though such defects may be closed effectively, dehiscence is common. This is because of the highly mobile nature of the palate during mastication and deglutition, and the presence of food and saliva within the oral cavity and oropharynx. It is not unusual for second and even third attempts at repair of full-thickness palatal defects to fail. However, small fistulae may heal spontaneously. As described above, feeding such cases by nasogastric intubation is important to reduce the risk of dehiscence of the suture line. Similarly, this method of feeding helps in the conservative management of such a fistula and should be combined with muzzling of the patient, in an attempt to reduce contamination of the airway by food material.

Clefts and other defects of the soft palate are more common than those affecting the hard palate but in the author's opinion are not suitable candidates for surgical repair. The affected animal should be destroyed if the degree of disability (dysphagia or exercise intolerance) is more than slight.

Injuries to salivary tissue

Injuries to the salivary glands may occur as a result of direct trauma. The parotid gland is the most vulnerable because of its size and location behind the angle of the jaw. Such injuries can be repaired by wound debridement, cleansing and closure of the skin. Salivary cutaneous fistulae are rare after this sort of injury.[5]

Injuries to the parotid duct are more common and are usually associated with direct trauma to the ventral border of the mandible, at which point the duct crosses before entering the oral cavity. In some cases there may be little evidence of an injury to the duct and there may be little need for specific treatment as the wound may heal readily.

However, in a proportion of casest there is direct continuity between the duct and a skin wound. This often results in the development of a salivary facial fistula. Such injuries often have the dramatic consequence of creating a profuse discharge of saliva during eating and mastication. Although saliva tends to have an inhibitory effect on healing, most of these wounds will eventually close in time, without need for specific treatment. It is the author's practice to manage all

parotid duct fistulae conservatively in the certain knowledge that in the vast majority of cases the fistula will heal uneventfully.

In those unusual cases in which the fistula does not close, surgical repair may be effective. The duct should be dissected from the edge of the fistula and closed with simple interrupted sutures of 2 metric polyglactin 910. Insertion of a catheter into the parotid duct may facilitate suturing, making accurate repair less problematic.

Injury to the parotid duct may occur inadvertently during facial or dental surgery. However, an understanding of the local anatomy should preclude such an occurrence. Transection of the parotid duct may be performed electively when carrying out a buccotomy technique for removal of mandibular or maxillary cheek teeth. In such cases, an end-to-end anastomosis can be carried out using simple interrupted sutures of 2 metric polyglactin 910. A parotid duct fistula may follow surgical removal of sialoliths which are occasionally encountered in older horses. Secondary closure of such wounds may be effective or alternatively they may be left to heal by second intention.

Injuries to the mandibular or sublingual salivary glands or ducts are very rare. The author has encountered one horse with a ranula associated with the sublingual salivary duct. This was managed successfully by oral marsupialization.

REFERENCES

1. Anon (1975) *Equine Digestive System*, ed. R Getty, WB Saunders, Philadelphia.
2. Baker GJ (1985) Oral examination and diagnosis: management of oral diseases. In: *Veterinary Dentistry*, ed. CE Harvey, WB Saunders, Philadelphia, pp. 217–234.
3. Engelbert TA and Tate LP (1993) Penetrating lingual foreign bodies in three horses. *Cornell Vet*, 83(1), 31–38.
4. Kiper ML, Wrigley R, Traub-Dargatz J and Bennett D (1992) Metallic foreign bodies in the mouth or pharynx of horses: seven cases (1983–1989). *Journal of the American Veterinary Medical Association*, 200(1), 91–93.
5. Auer JA (ed.) (1992) *Equine Surgery*. WB Saunders, Philadelphia, pp. 306–308.
6. Scott EA (1982) Surgery of the oral cavity. *Veterinary Clinics of North America Large Animal Practice*, 4, 3–31.
7. Howard RD and Stashak TS (1993) Reconstructive surgery of selected injuries of the head. *Veterinary Clinics of North America Large Animal Practice*, 9, 185–198.
8. Robinson NE (ed.) (1997) *Current Therapy in Equine Medicine*, Vol.4. WB Saunders, Philadelphia, pp. 148–149.
9. Hague BA and Honnas CM (1998) Traumatic dental disease and soft tissue injuries of the oral cavity. *Veterinary Clinics of North America Large Animal Practice*, 14, 333–347.
10. Modransky P, Welker B and Pickett JP (1989) Management of facial injuries. *Veterinary Clinics of North America Large Animal Practice*, 5, 665–682.

8 Dental Trauma

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Introduction

Horses, particularly young horses and foals, are curious animals. They are kept in fields, paddocks, barns, sheds and stalls and frequently explore their surroundings with their lips and tongues. Such explorations often result in accidents and wounds to the lips, mouth, tongue, teeth and jaws. The management of oral trauma is described in Chapter 7 and mandibular and maxillary fracture osteosynthesis is detailed in Chapter 21.

Other causes of dental trauma may well be the sequelae of dental work by veterinarians and equine dental technicians. Inadvertent tooth fracture during the reduction of the crowns of overgrown teeth, as a result of the improper application of molar or incisor cutters, may require extraction of the traumatized tooth. Inadvertent exposure of the occlusal aspects of the pulp chambers may also occur in crown reduction procedures on overgrown teeth. In an *in vitro* study it was shown that prolonged power-grinding reductions, without the use of water cooling, can create destructive hyperthermia of pulp tissues.[1] In a 2-minute contact procedure, an increase of an average of 24.3°C was recorded from thermocouples implanted 15 mm from the contact point and sited at the dentino-pulp junction.[2] It is known that temperature increases of 5°C create cellular damage to pulp tissues and that a 16.7°C temperature increase causes irreversible pulp necrosis[2] (see Chapter 11).

Dental fractures

Tooth fractures can be classified based on anatomic location, tooth number and position (mesial, distal, lingual or buccal, exposed crown, reserve crown and root), as well as on etiopathogenesis. They may occur as a result of direct trauma, from kicks and falls, from avulsions as a result of the subject playing with or grasping objects and then panicking and pulling back (Fig. 8.1A,B) if their teeth get stuck, and from inadvertent tooth fracturing during reduction procedures by veterinarians and dental technicians. In many cases of maxillary incisive bone and mandibular bone fractures (see Chapter 21) there is often accompanying dental trauma and tooth fractures. The teeth may also fracture as a sequel to impactions, misplacements and dental decay.

In a long-term study of 400 equine dental cases, 101 horses were diagnosed with abnormalities of wear, traumatic damage, idiopathic fractures or tumors of their cheek teeth.[3] There were 26 cases of traumatic damage with a median age of 6 years (range 1–20) and 24 cases of idiopathic fractures (Figs 8.2 and 8.3) with a median age of 10 years (range 2–27). Of the traumatic cases, seven were the result of iatrogenic damage (two mandibular tooth cases and five maxillary teeth cases). In the idiopathic fracture cases, carious lesions were found in seven maxillary cheek teeth and one mandibular cheek tooth. In the other 16 cases, there were 14 lateral slab fractures, three medial slab fractures and two transverse crown fractures.[3] In the same series of 400 equine dental cases, there were 11 cases of incisor tooth fractures. It is interesting to note that in this series of 11 cases, the median age was 2 years (range 1–9), being significantly younger than the horses with traumatic fractures of the cheek teeth.[4] In that series, treatment of fractured incisor teeth was symptomatic, antibiotic and anti-inflammatory therapy followed by extraction of teeth and stabilization of incisive bone and mandibular fractures. Cheek tooth fractures were also treated conservatively unless they were very loose or grossly infected, when oral extraction was indicated and used.

In the large study based on oral examinations of 30,000 cavalry horses, a clinical incidence of 0.71 per cent of equine dental fractures was reported. Radiographic evidence of tooth fractures was found in 3.5 per cent of 500 skulls.[5,6]

Diagnosis

The case history should be followed by a thorough oral and dental examination with careful irrigation of the traumatized areas and palpation and manipulation of the surrounding bones and tissues. This is facilitated by the use of sedation and analgesics. In most cases, radiographs of the traumatized area should be taken to assist in the documentation of the complete nature of the trauma to the teeth and their supporting structures.

Acute exposure of pulp tissue from exposed crown fractures is recognized by the presence of bleeding spots

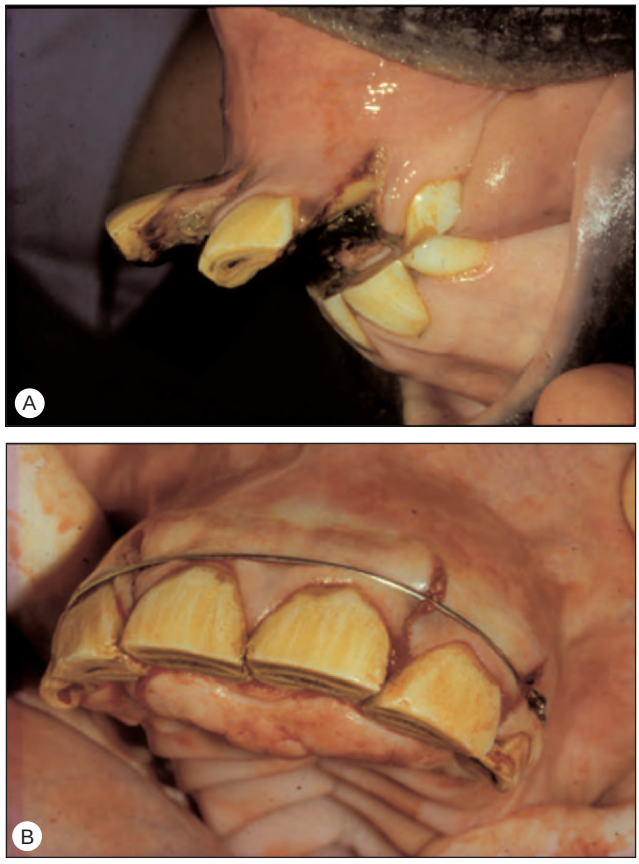


Figure 8.1. (A) Incisor alvulsion fracture and (B) repair and wire stabilization.

and/or extrusion of pulp tissues. During power reduction of overgrown crowns, the reduction process should be monitored closely and, as soon as a pink 'blush' is seen, e.g., in creating bit seats on the 6s, the reduction grinding should be stopped (see Chapter 11). Chronic pulp exposure may lead to pulp necrosis and periapical osteitis and dental decay. It is therefore recommended that pulp protective procedures be used (see Chapter 19).

It is not uncommon for draining mandibular fistulous tracts and perapical osseous swellings to be blamed upon previously undiagnosed or unrecognized mandibular and/or dental trauma. This is most unlikely. Such cases are much more likely to be the result of eruption abnormalities and dental impactions (see Chapter 6).[7]

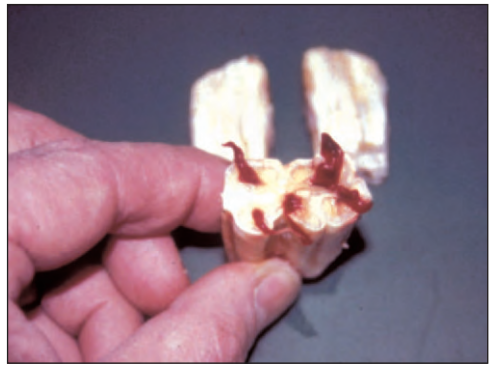


Figure 8.2. Fractured mandibular cheek tooth caused by oral extraction of an adjacent tooth (from Dixon *et al.*[3] with permission, *Equine Veterinary Journal*).

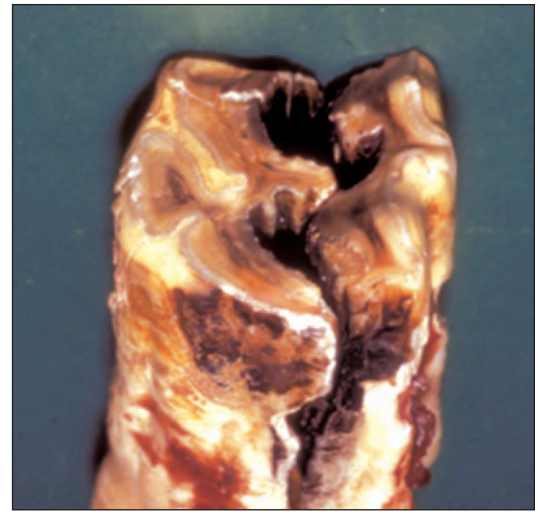


Figure 8.3. Mandibular tooth extracted because of draining fistula following dental fracture (from Dixon *et al.*[3] with permission, *Equine Veterinary Journal*).

Dental healing

As was described in Chapter 3, a significant amount of tooth structure material is inorganic in nature. Subsequently, fractures of inorganic material do not heal. However, the organic components of teeth, in particular the pulp chambers, have cellular elements, nerves, blood and lymph structures that respond in a classic healing fashion to traumatic insult.

When the pulp is injured as a result of exposure from dental fractures, the undifferentiated mesothelial cells, fibroblasts adjacent to the exposure, undergo proliferative activity.[8] During the repair phase, differentiation of some cells into bone- or dentin-producing cells occurs and hard tissue is elaborated. These odontoblasts lining the pulp chambers produce reparative dentin and an intrapulpal dentin bridge is formed. Provided there is no active pulpal infection, this internal bridge stabilizes the traumatized tooth. The author has compared this process in horses' teeth to the formation of internal callous formation in primary bone healing under ASIF (Association for the Study of Internal Fixation) conditions.[9]

Clinical management of dental trauma

As in all traumatic events, a complete history and full physical evaluation should precede specific examination of the oral cavity, lips, tongue, teeth and supporting bony structures.

In acute cases, the use of sedation and analgesia will be required in order to complete a thorough oral examination (see Chapter 13). Individual teeth should be inspected and manipulated. Loose tooth fragments should be removed. Badly fractured teeth may have to be extracted. However, less severe fractures, rotation and loosening can be managed conservatively. In all cases, appropriate tetanus prophylaxis and antibiotic therapy should be used. In many cases, dental trauma will be accompanied by oral soft tissue injuries (see Chapter 7).

General anesthesia greatly facilitates both the oral examination and the planning process for the surgical repair of oral wounds and for operative dental procedures.

Principles of treatment

Care must be taken to stabilize loosened teeth and to eliminate the occlusal contact of their erupted crowns. This is best done by diamond disk or burr reduction of the erupted

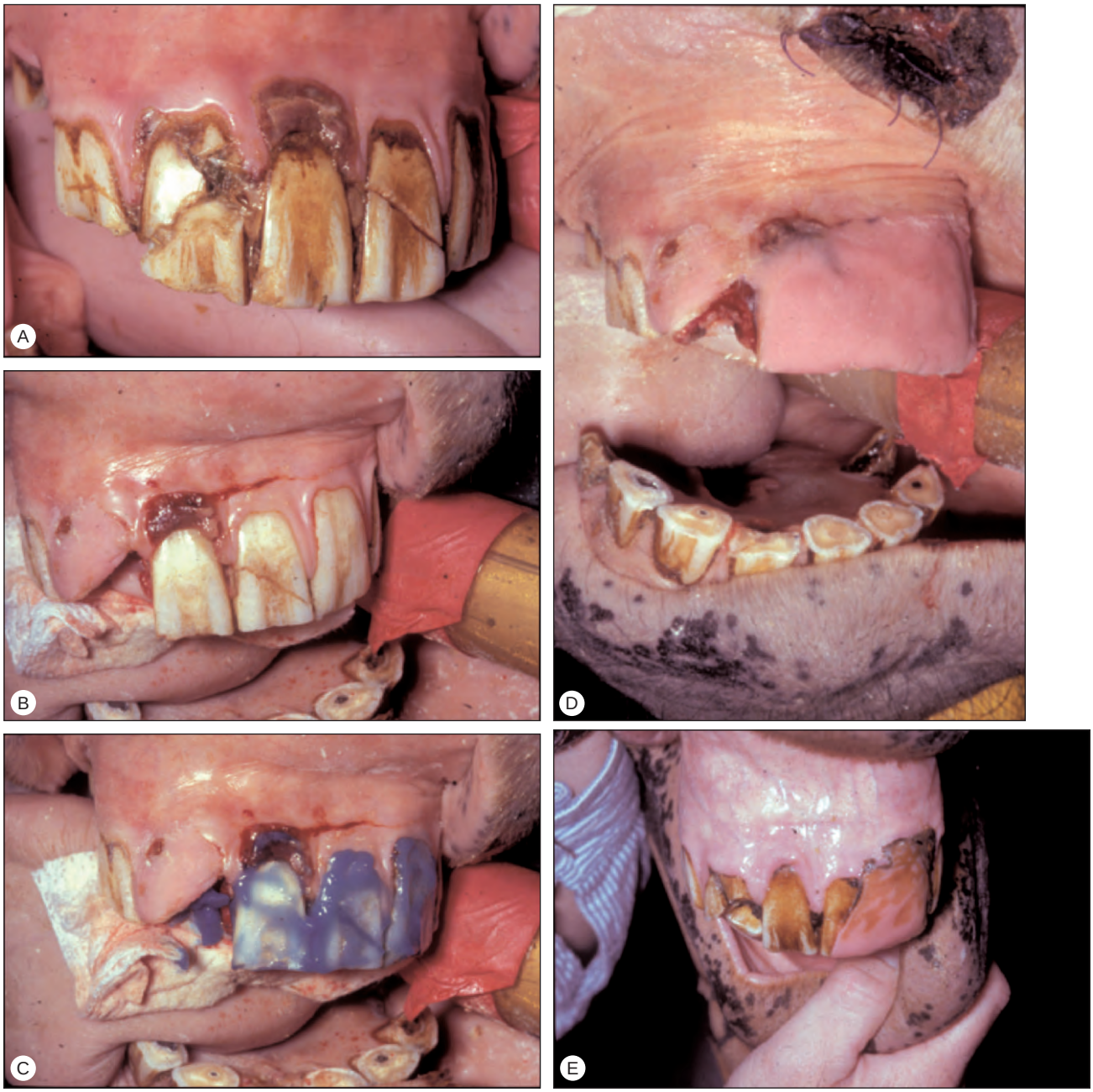


Figure 8.4. (A) Fractured 101, 102, 201 and 202; (B) surface cleaning; (C) etching; (D) application of Jet Acrylic; (E) 3 months postoperative evaluation.

crown and its occluding partner, e.g., if 208 is the traumatized tooth the distal portion of 207 and mesial portion of 209 should be reduced as well as the whole of the occlusal surface of 208. A mirror image reduction should be made of 107, 108 and 109. The injured tooth should be inspected for pulp exposure and, if detected, appropriate pulp-capping treatments should be used (see Chapter 19).

Traumatized or loose teeth can be stabilized using contact wires and bridges attached to the injured tooth and bridged to mesial and distal partners. The use of acrylics (Jet Acrylic, Henry Schein Inc., Melville, NY) may be used to reinforce the stabilization process (Fig. 8.4A–E). The tooth surfaces are cleaned ultrasonically and etched with phosphoric acid before applying the acrylic powder and accelerator (see Chapter 20 for details of dental materials and their application).

After repair and stabilization, it is recommended that a soaked concentrate diet be used for at least 3 months. The stabilization should be monitored by the owner and, if there is evidence of any instability, then the fixators should be removed and replaced. In most cases the absence of complications, such as draining fistula or sequestration, for a 3-month period is good clinical evidence of success. Radiographs are helpful in monitoring the healing process.

In the absence of success, tooth loosening, etc., extraction of the affected tooth or teeth is indicated.

Summary

Dental trauma is a frequent event in equine health. Cases should be evaluated thoroughly and every attempt should be made to preserve the traumatized tooth or teeth. Not all cases will be successful, but anti-inflammatory agents, antibiotics and restorative technologies will be useful in saving teeth that otherwise would be treated by exodontia.

REFERENCES

1. Baker GJ and Allen ML (2002) The use of power equipment in equine dentistry. *Proceedings AAEP,* 48, 438–441.
2. Allen ML (2001) An in-vitro study of heat production in equine teeth from power reduction. MS Thesis, University of Illinois.
3. Dixon PM, Tremaine WH, Pickles K *et al.* (2000) Equine dental disease Part 3: a long term study of 400 cases: disorders of wear, traumatic damage and idiopathic fractures, tumours and miscellaneous disorders of the cheek teeth. *Equine Veterinary Journal,* 32, 9–18.
4. Dixon PM, Tremaine WH, Pickles K *et al.* (1999) Equine dental disease Part 1: A long term study of 400 cases: disorders of incisor, canine and first premolar teeth. *Equine Veterinary Journal,* 31, 369–377.
5. Becker E (1945) Cited by Becker E (1962) Zähne. In: *Handbuch der Speziellen Pathologischen Anatomie der Haustiere* (3rd edn), Band V, eds. J Dobberstein, G Pallaske and H. Stunzi, Verlag Paul Parey, Berlin, p. 130.
6. Geres V (1962) Cited by Becker E (1962) Zähne. In: *Handbuch der Speziellen Pathologischen Anatomie der Haustiere* (3rd edn), Band V, eds. J Dobberstein, G Pallaske and H. Stunzi, Verlag Paul Parey, Berlin, p. 265.
7. Baker GJ (1979) A study of dental disease in the horse. PhD Thesis, University of Glasgow.
8. Fitzgerald M (1979) Cellular mechanics of dentinal bridge repair using 3H-thymidine. *Journal of Dental Research,* 58 (Special Isssue D), 2198.
9. Baker GJ (1999) Dentistogenic pulpitis in equine dentistry. *Proceedings Veterinary Dental Forum,* 269–272.

9 Equine Dental Pathology

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Introduction

Equine dental disorders are a major part of equine practice in the United Kingdom, as indicated by a British Equine Veterinarian Association (BEVA) survey in 1965 that showed 10 per cent of equine practice time was spent on dental-related work.[1] More recently, a survey in the United States ranked dental disorders as the third most common equine medical problem encountered by large animal practitioners.[2] Despite its importance to veterinary practice, a survey of 150 adult horses with no history of dental disease showed that 24 per cent of these horses did in fact have dental abnormalities.[3]

Gross anatomical features of equine dentition have been recorded as far back as 600 C.E.[4] These include eruption times of deciduous and permanent dentition, presence of incisor 'hooks' and grooves, and the presence of features such as the 'dental star' on the occlusal surface of incisors that were used in aging horses.

Imaging

Previous reports on equine dental imaging have concentrated mainly on radiology of clinical cases or post-mortem specimens.[5–9] Recently, there has been an increased use of alternative dental imaging techniques such as computerized tomography,[10] nuclear scintigraphy[11] and magnetic resonance imaging.[12] Intra-oral endoscopy has also been used to gain an enhanced view of dental clinical crowns.[13]

Although use of these imaging modalities has been of great use in assessment of cases, recent advances in light and scanning electron microscopy techniques have more fully increased our knowledge on the morphology of both normal and diseased teeth. Most of the techniques for undertaking equine dental histology have come from those used in brachydont teeth studies; however, when dealing with equine teeth there are additional considerations. The most obvious of these is the size of equine teeth. With cheek teeth (CT) being over 100 mm long in some young adults (*cf.* 25 mm in adult human teeth), and weighing over 120 g (*cf.* 2.5g for an average human molar) there is considerably more tissue to process and examine histologically compared with a human tooth.

It is often difficult to locate a focal area of disease within a tooth using standard equine dental radiology. Without being able to recognize important pathological areas prior to dissection, the area of interest may be destroyed during histological processing. When compared with human teeth, the overlapping complex pattern of enamel infolding present in equine dentition, particularly in CT, will often obscure dental anomalies. Even obvious conditions such as pulpar exposure will usually be missed radiologically.

A problem encountered when processing decalcified equine dental sections, in both transverse and longitudinal planes, is that following decalcification, enamel is lost (being 98 per cent calcium hydroxyapatite crystal) and so ceases to support the adjacent dentine and cementum. Consequently, during further processing, decalcified sections are prone to fall apart into individual components of cementum and dentine unless the operator is careful. Cementum and dentine, having a 30–35 per cent organic component (principally of collagen fibres) retain their structure and together with pulp are the tissues of interest in decalcified histological sections. Despite these difficulties, histological sections of equine teeth can now be processed to an equal quality as human dental sections (i.e. to 3–5 μm thick in decalcified, and 40 μm thick in undecalcified sections).[14,15]

Interpretation of equine dental histology may be inaccurate if the horse's clinical history is not available, e.g. the actual age of the animal, or the presence of dental pain. Bender showed that 80 per cent of human cases involving histological changes to the dental pulp had associated pain,[16] but the presence of dental pain is difficult to detect clinically in the horse. Often microscopic lesions such as 'cracked tooth syndrome' cause dental pain, yet challenge the human dental practitioner to diagnose, despite being able to thoroughly question the patient.[17] With equine patients, the veterinarian faces an even more challenging task and this may reflect why dental cases often have advanced pathology when examined histologically.

Although equine practitioners seldom request histological evaluation of teeth, a great deal of information may be gathered by grossly examining an extracted tooth. In the past this has been performed using manual hacksaws or even hammers; however, a much better result is gained by using commercial diamond cutting wheels or blades, e.g. attachments to Dremel® powered instruments or tile-cutting saws,

with water or oil as a lubricant. As more cases are investigated, the profession is becoming better placed to evaluate equine dental pathology.

Endodontic anatomy

In discussing dental pathology it is beneficial to review equine endodontic anatomy. Anatomists and paleontologists have given precise terminology to anatomical features of the equine tooth from an evolutionary point of view (protocone, mesostyle, hypoconuclid etc.); however, it is more practical for the clinician to consider the tooth based upon its endodontic anatomy. Even in older teeth, where the pulp horns are completely filled with secondary dentine, it is still very useful to have a reference system for these anatomically distinct dental regions. There has been only limited research into equine pulpal anatomy and this could explain why, to date, endodontic therapy has been of limited success in treating equine apical infections – one of the most significant equine dental diseases.[18]

During dentogenesis (i.e. tooth development, *cf.* dentinogenesis – production of dentine), there is a progressive deposition of dentine on the periphery of the pulp chamber. The initial common pulp chamber subsequently divides into pulp horns. The age at which this takes place varies between individual pulps, individual teeth, and individual animals. This is dependent on the rate of dentine deposition, and factors affecting this such as blood supply and alkaline phosphatase activity.[19]

Separation of the common pulp chamber into individual horns begins occlusally, moving in an apical direction (Figs 9.1 and 9.2). It had previously been thought that equine incisors, canines and wolf teeth had a single pulp chamber throughout their life; however, recent work has shown splitting or divergence of the principal pulp horn and presence of accessory canals, particularly in older incisor teeth (David Klugh 2003, unpublished data).

The middle four (maxillary and mandibular) CT (Triadan 07–10) contain five pulp horns arising from a common pulp chamber. Use of an endodontic numbering system, combined with standard anatomical directional terminology, enables accurate location of any ultrastructural anatomical or pathological features within the tooth. In maxillary CT (Fig. 9.3), the rostrobuccal pulp horn is numbered 1, the caudobuccal 2, rostropalatal 3, caudopalatal 4, and midpalatal 5.[20] In mandibular CT rows (Fig. 9.4), the rostrolingual pulp horn is numbered 1, midlingual 2, caudolingual 3, rostrobuccal 4 and caudobuccal 5. The first CT (Triadan 06) in all four rows has an additional smaller pulp horn, rostral to the others, numbered 6. The sixth CT in maxillary rows (Triadan 1/211) has an irregular caudal extension of the second pulp horn, and when discrete, is numbered 7. An additional pulp horn may be present palatal to a caudal peripheral enamel infolding, which is numbered 8. The sixth CT in mandibular rows (Triadan 3/411) has an additional caudal pulp horn, numbered 7.

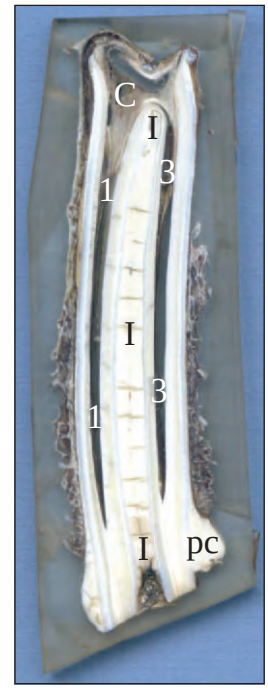


Figure 9.1. A longitudinally sectioned maxillary cheek tooth of a young horse embedded in resin showing two pulp horns (1 and 3) extending occlusally from a common pulp chamber (C). The rostral infundibulum (I) descends from the occlusal surface to the area where the pulp horns divide. Peripheral cementum (pc) is markedly thicker at the gingiva and clinical crown.

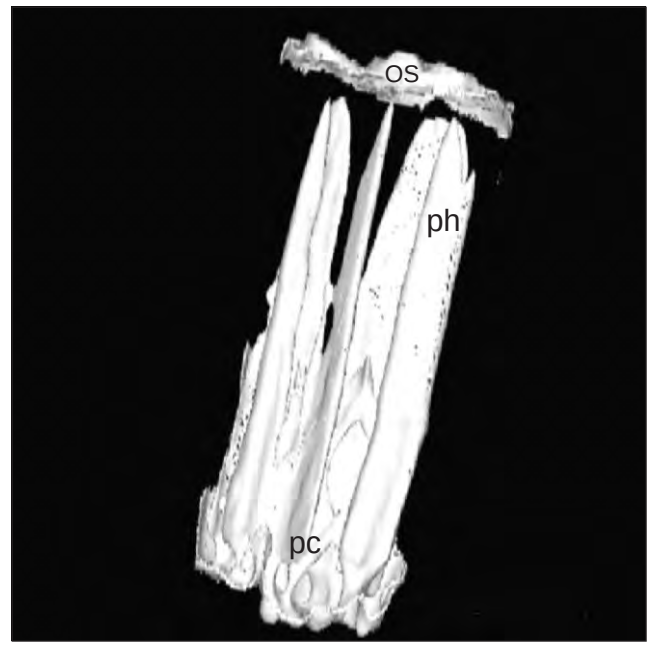


Figure 9.2. Computer-assisted tomograph of an intact mandibular cheek tooth having removed the imaged cementum, dentin and enamel from the image based on their radiodensities, leaving the pulp horns (ph) extending toward the remaining organic pellicle on the occlusal surface (os) from the apical common pulp chamber (pc).

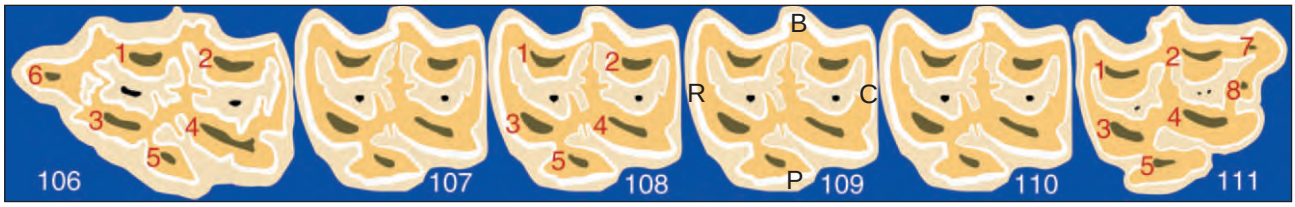


Figure 9.3. Diagrammatic representation of right maxillary cheek teeth row's occlusal surface. Pulp horn positions are indicated by the overlying secondary dentin (shown here as dark brown, with enamel being white, primary dentin tan, and cementum cream). Pulp horn numbers 1–8 are identified to the left of the pulp horn. Teeth 107, 109 and 110 all have similar pulp horn numbering to 108. Pulp horns 7 and 8 are not always present on tooth 111. (B) buccal; (P) palatal; (R) rostral; (C) caudal. The left maxillary CT row is a reflection of this image.

Ultrastructural anatomy

The ultrastructural anatomy of the calcified equine dental tissues is complex and varied.[21–24] All equine teeth comprise three calcified tissues and pulp, as discussed in Chapter 3.

Dentinopulpal complex

The dentinopulpal complex is so termed because of the intricate association between the dental pulp and dentine. As these tissues are frequently involved in many equine dental pathological processes, their anatomy is briefly revisited here. Additionally, as dentine is laid down progressively, it may also record events (physiological or pathological) that occur throughout the lifespan of a tooth, much as the rings in the trunk of a tree do.

The dental pulp consists of an intricate network of loose connective tissue supported on a fibrous skeleton with blood vessels, lymphatics and nerves that enter and exit at the dental apex. The outermost layer of pulp consists of odontoblast cells, responsible for continuous dentine production. This occurs soon after the formation of the enamel 'scaffolding' within the dental sac, and continues over the life of the equine tooth (Fig. 9.5). Odontoblast cells leave long tubular processes within the dentine (odontoblast processes within dentinal tubules) as they retreat centrally into the pulp, with progressive deposition of dentine.[23,25,26] Odontoblast processes extend horizontally to the amelodentinal junction and vertically to the occlusal surface of teeth[23] (Fig. 9.6). They may transmit normal (physiological) stimuli from the occlusal or interproximal surfaces regulating the rate of dentine deposition in the underlying pulp,[27] or transmit noxious stimuli (e.g. from physical, chemical or thermal trauma or caries) to initiate tertiary dentine production.

Reclassification of equine dentine

There is some confusion concerning the classification of human dentine as being primary, secondary or tertiary. Ten Cate describes primary dentine as the dentine laid down 'until the external form of the tooth is completed' with secondary dentine being laid down thereafter.[19] Torneck, however, defines secondary dentine as that which 'develops after root formation has been completed.'[28] Neither of these definitions is applicable to equine (hypsodont) teeth, with their continued deposition of peripheral cementum at levels of the reserve crown and roots,[29] thereby changing the external shape of the tooth throughout its life. When discussing equine dentition it may therefore be most practical to use Kierdorf's definition of secondary dentine as that which is deposited once a tooth is in full occlusal contact.[30]

Numerous incremental lines are present in human dentine (and enamel) indicating the phasic nature of mineral deposition.[31] They may be seen under light microscopy of undecalcified (ground) and decalcified dental sections, reflecting their presence in mineralized and matrix components of these hard tissues respectively. A neonatal line is seen in humans reflecting stress upon dentine production around birth. Similar lines are seen following febrile diseases of childhood. Such hematoxyphilic lines in bone are found to contain less calcium and phosphate than surrounding bone, but more sulfur. Reversal lines are also seen within cementum and bone (but not dentine or enamel as they do not remodel) indicating a change from mineral deposition to resorption.

Resting, reversal and incremental lines have been observed in equine dental histological sections; however, their presence is not constant and not yet fully understood (Figs 9.7 and 9.8) despite their long use as archeological tools for assessing

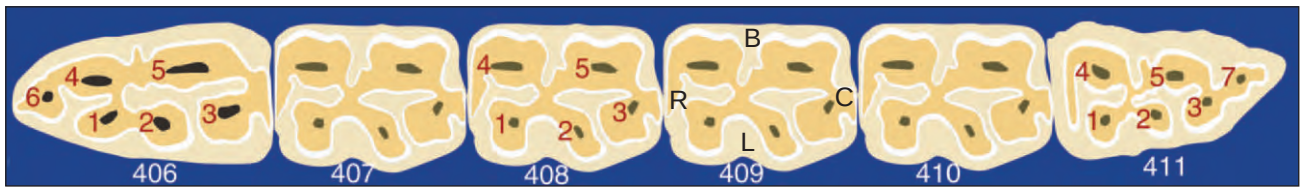


Figure 9.4. Diagrammatic representation of right mandibular cheek teeth row's occlusal surface. Pulp numbering is the same as for Fig. 9.3. (B) buccal; (L) lingual; (R) rostral; (C) caudal.

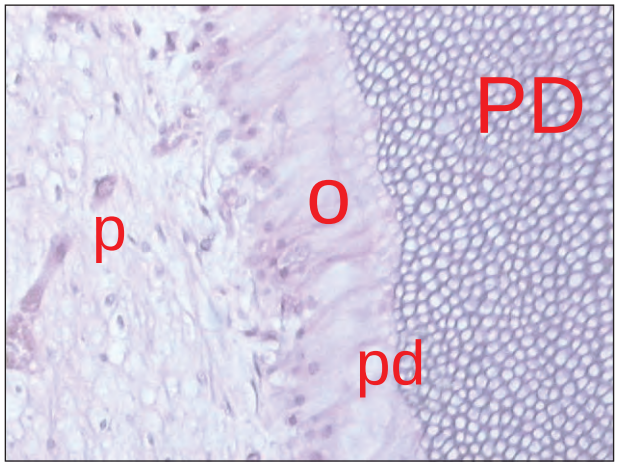


Figure 9.5. Odontoblast cells (o) withdrawing toward the center of the pulp (p) laying down a secretory matrix, i.e. a paler staining layer of predentin (pd) adjacent to the primary dentin (PD).

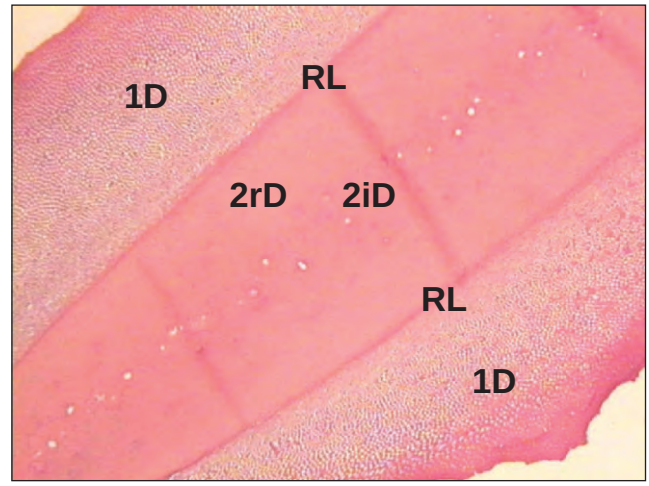


Figure 9.7. Decalcified transverse histological section through a mandibular CT pulp horn that has become completely filled with dentin. Primary equine dentin (1D) is peripheral to regular secondary dentin (2rD) and irregular secondary dentin (2iD) with a resting line (RL) present between them (H and E).

such factors as seasonality of death in fossilized equine remains.

In equine primary dentine the odontoblast processes are surrounded by dentinal tubules, which are filled with peritubular dentine (more precisely called intratubular dentine), the latter being present only in primary dentine (Fig. 9.9).[23,25] As intratubular dentine confers resistance to wear, it is unclear why it should be absent from secondary dentine. The mechanism of intratubular dentine formation is not currently understood. A thin layer of intertubular dentine (which has a honeycomb appearance when etched sections are viewed by scanning electron microscopy) in turn surrounds intratubular dentine.

Secondary dentine is further classified as being either regular or irregular, with irregular secondary dentine also being referred to as reparative, reactive or tertiary dentine. Following eruption, regular secondary dentine is laid down by odontoblasts throughout the majority of the life of the tooth, with the odontoblast cells withdrawing centripetally (toward the center of the pulp) from the previously laid down primary dentine (Fig. 9.10). Secondary dentine is continuous with primary dentine, sharing the same odontoblast process within a continuation of the same dentinal tubule. Under normal circumstances 'regular' secondary dentine will be laid down within the pulp chamber by the odontoblasts until the pulp chamber is almost completely occluded.

Irregular secondary (reparative, reactive or tertiary) equine dentine has previously been classified as being laid down in response to noxious stimuli. However, recent work (Dacre 2003, unpublished data) has shown irregular secondary

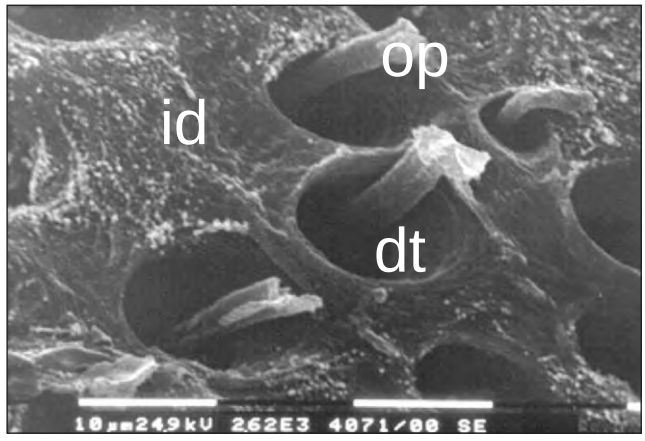


Figure 9.6. Odontoblast processes (op) may be seen emerging from dentinal tubules (dt) on the occlusal surface. Intertubular dentin (id) lies between the dentinal tubules. It is assumed these odontoblast processes are sclerotic, and that the remaining space within the dentinal tubule was originally filled with highly mineralized intratubular dentin that was removed following acid etching of the tissue during processing of this section for scanning electron microscopy.



Figure 9.8. In comparison to Figure 9.7 this decalcified transverse section shows a transition zone (TZ) between primary (PD) and secondary (SD) dentin and no resting line (H and E).

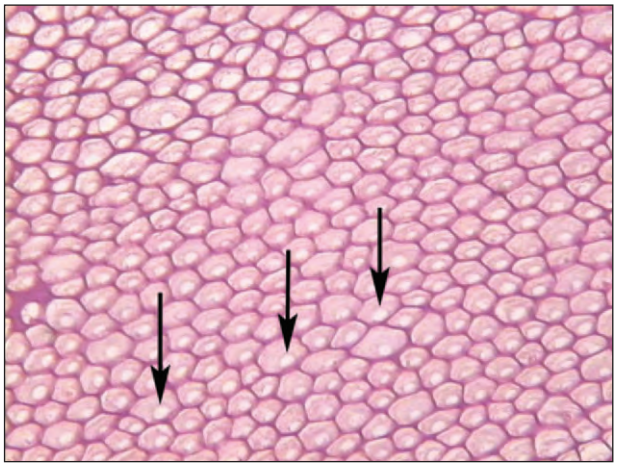


Figure 9.9. Normal equine primary dentin showing the typical honeycomb pattern of intertubular dentin. Within this network lighter staining organic remnants of now demineralized intratubular (previously called peritubular) dentin remain, with pale circular areas (arrow) previously occupied by odontoblast processes still evident (decalcified H and E transverse section).

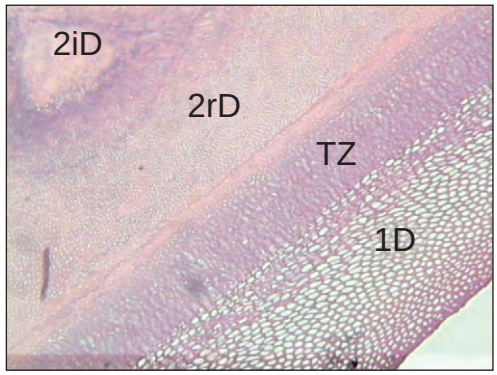


Figure 9.11. As odontoblasts retreat toward the center of the pulp they lay down primary dentin (1D) prior to eruption, regular secondary dentin (2rD) following occlusion and finally irregular secondary dentin (2iD) that fills the pulp horn completely under normal conditions. A transition zone (TZ) between primary and regular secondary dentin is often present (H and E)

dentine to exist in all normal equine teeth that have no evidence of prior exposure to noxious stimuli. Secondary irregular dentine is laid down in the most central part of the pulp horn when this region is undergoing its final physiological stage of pulp replacement by dentine (Figs 9.11 and 9.12). The continued formation of both regular and irregular secondary dentine prevents pulpar exposure on the occlusal surface.

Tertiary (otherwise termed reactive or reparative) dentine is laid down focally, in response to specific local noxious stimuli[19] (Fig. 9.13). It is not continuous with primary or secondary dentine, with the dentinal tubules within its matrix being distorted by cells trapped within this rapidly laid down tissue. It is laid down by recently differentiated mesenchymal cells from within the dentinal pulp, when the overlying odontoblast cells have been destroyed.[32]

Where areas of dentine have failed to mineralize sufficiently, interglobular dentine is seen (Fig. 9.14). Being a failure of mineralization rather than matrix formation, we see the normal architectural pattern of the dentinal tubules running through zones where the globular calcospherites have failed to fuse into a homogenous mass within the maturing dentine. Interglobular dentine is thought to arise in human teeth in vitamin D deficiency or following exposure to high fluoride levels at the time of dentine formation.[28]

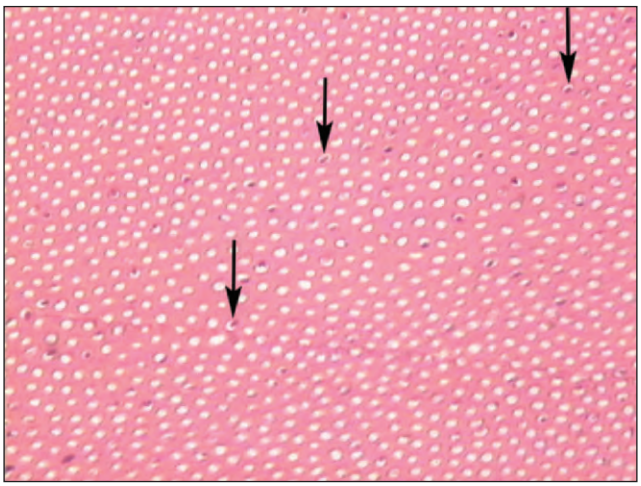


Figure 9.10. Normal equine regular secondary dentin in transverse section. The absence of intratubular dentin and greater proportion of intertubular dentin makes this appear very different to that of primary dentin. Remnants of odontoblast processes still remain (following processing) in some of the dentinal tubules (arrow) (H and E).

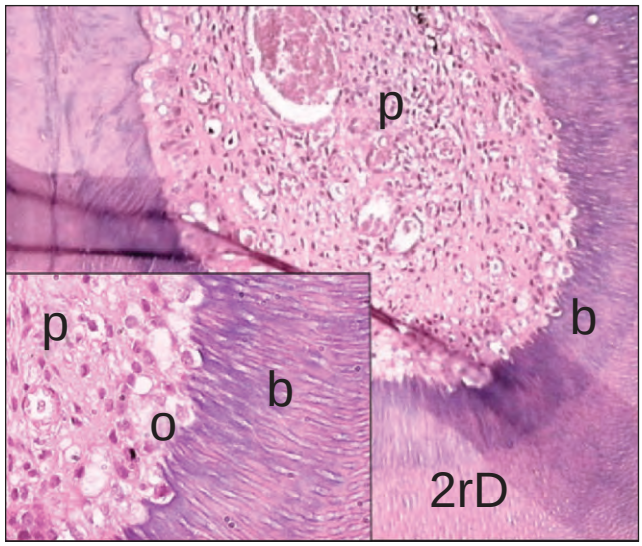


Figure 9.12. A basophilic staining line (b) occurs where the dentin changes orientation from regular secondary dentin (2rD) to what will become irregular secondary dentine. Odontoblasts (o) are seen at the margin of the pulp (p) as shown more clearly in the insert. Note the change in orientation of the dentinal tubules within the basophilic line as shown by their being sectioned in a more longitudinal plane (H and E).

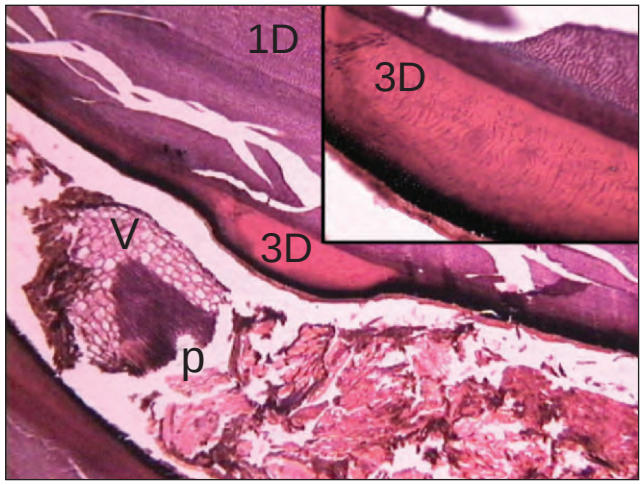


Figure 9.13. Tertiary dentin (3D) has been laid down within the pulp of this equine cheek tooth in response to focal noxious stimuli. As seen more clearly in the inset (top right) there is no continuation of dentinal tubules into this tertiary dentin (as occurs between primary and secondary dentin). The pulp (p) has been exposed latterly through dental attrition resulting in the pulp horn filling with vegetable material (V) and infection and destruction of any pulp that remained (decalcified transverse H and E section).

Sclerotic dentine is formed when dentinal dental tubules become occluded with calcified material. The degree of dentinal sclerosis increases with age in human teeth without any clearly identifiable external influence and by several different mechanisms. In horses, where dentine is exposed on the occlusal surface we would expect the degree of dentinal sclerosis to be high, preventing bacterial invasion of such exposed dentinal tubules. Kilic demonstrated the presence of large numbers of odontoblast processes in occlusal surface dentine.[33] It was not clear at the time if these processes were calcified (i.e. sclerotic). The presence of open dentinal tubules and odontoblast processes on the occlusal surface has been proposed to be part of a sensory mechanism to limit the stress applied to teeth (with fluid movement within dentine during tooth compression) and thus may facilitate discrimination of particle hardness during mastication.[34] This was previously believed to be principally regulated via mechanoreceptors within the periodontal ligament.

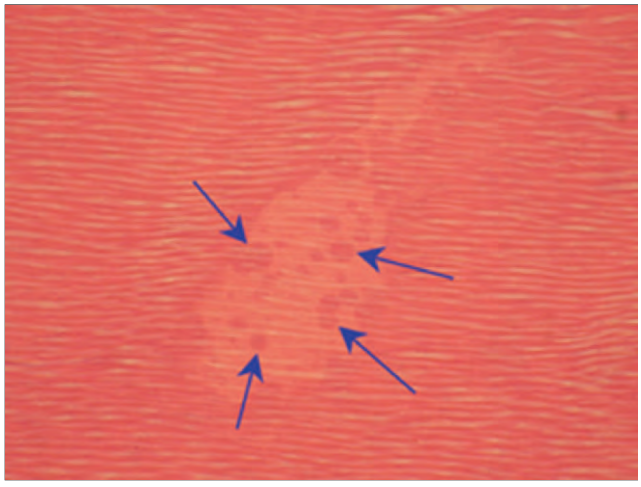


Figure 9.14. Globular dentin is present in areas of rapid dentin deposition. Circular calcospherites (arrows) are seen within the less mineralized area of dentin (paler region) in this longitudinal dental section giving a 'globular' appearance (H and E ×400).

Dental pathology

It has long been recognized that our knowledge of equine dental pathology has been very limited, as exemplified by a statement of the eminent Victorian veterinarian William Dick in 1862: 'of the disease of the teeth in the horse we know little.'[35] Until recently the horse was considered to have a low incidence of dental disease compared with other species, possibly due to the difficulties of performing a thorough dental examination of the oral cavity (prior to the introduction of modern sedatives).[3]

Previous studies on equine dental pathology have concentrated on gross findings, with many of these examining skulls with unknown dental histories collected from abattoirs. Even so, much useful data has been gained and has shown equine dental disease to be common. Such studies include those of Colyer,[36] Voss,[37] Honma *et al.*,[38] Baker,[39] Wafa[40] and more recently Brigham and Duncanson.[41] On examining 365 abattoir skulls, Honma *et al.* found that 100 per cent identified as being 12 years or older had dental caries.[38] Examining 218 abattoir skulls, Baker recorded an incidence of 60 per cent periodontal disease and 79 per cent infundibular caries in horses aged over 15 years.[39] In 355 abattoir skulls, Wafa found 12.6 per cent of cases to have abnormalities of development or eruption; 16.7 per cent to have wear abnormalities; 34.9 per cent with periodontal disease; 29.3 per cent to have caries and 6.5 per cent to have dental pulp exposure.[40] He concluded this latter disorder was 'of greatest clinical significance' and that 'all of the periapical infections in this study were attributed to exposure of the dental pulp.'

This contrasts with the survey by Dixon *et al.* of 400 referral dental cases of which 162 were referred with primary apical abscessation (i.e. of unknown etiology).[18] Although pulpar exposure was identified in a small number of these cases (of primary apical abscessation), many more apical abscesses were seen to have arisen from predisposing concurrent dental conditions, such as deep periodontal pocketing following dental displacement, that were defined as secondary apical abscesses. Dixon *et al.* detailed the incidence of gross pathological dental disorders in a referral population, most being of a severe nature, typical of referral dental cases.[18,42–44] Brigham and Duncanson's study of 50 abattoir skulls found most dental disorders to be in CT, with 20 per cent having CT diastema; 26 per cent with focal overgrowths (hooks); 56 per cent with sharp enamel overgrowths; 20 per cent with missing adult teeth; 8 per cent with 'wavemouth' and 12 per cent with caries. This last figure of 12 per cent incidence in caries is relatively low compared with other studies,

considering that 62 per cent of skulls examined were aged at over 18 years old.

Developmental dental disorders

(see chapter 6)

Although a number of different dental abnormalities are often present concurrently and may be interrelated in their pathogenesis, it is useful to classify them as being either developmental or acquired in origin. Those termed 'developmental' are present at time of tooth eruption, while 'acquired' disorders develop subsequently. The presence of developmental dental disorders may often predispose acquired disorders to cause more severe clinical disease.

Developmental conditions arising prior to eruption

Polyodontia (supernumerary teeth) are thought to arise from the splitting of dental buds during dentogenesis, and in horses commonly results in a seventh (caudal) maxillary CT, which will often be connated (one or more joined teeth – often near the roots). Polyodontia may result in dental displacement of the supernumerary or adjacent teeth, and focal overgrowths will eventually develop if the additional tooth is unopposed occlusally. Although they may follow the same general endodontic arrangement of the tooth type as their position would indicate, supernumerary teeth are usually irregularly shaped and do not fit well in the CT row, predisposing to food pocketing between themselves and the adjacent teeth. This usually results in progressive periodontal disease that can lead to apical abscess formation or oromaxillary fistulas in advanced cases.

In humans the congenital absence of teeth may be referred to as hypodontia, anodontia or oligodontia, and may be partial or total.[45] In horses, the term hypodontia has not been widely used. The term anodontia refers to complete absence of dental development, which does not appear to have been reported in horses. Oligodontia, referring to the congenital absence of one or more teeth, has been reported in horses by at least five authors. It arises from failure of a dental bud (or buds) to develop for ensuing dentogenesis. Oligodontia may be considered physiological (i.e. normal) when horses fail to develop the first premolar (Triadan 05s; 'wolf teeth'), or when mares do not develop canine teeth. Reported cases of pathological equine oligodontia have involved absence of multiple teeth, with dental dysplasia (abnormal growth or development) being present in some or all teeth that are present. Oligodontia may also be associated with other epidermal defects such as poor development of coat and hooves.[46]

Dysplasia of dental tissues may also result in an irregularly shaped tooth that does not fit well in the dental row, with a potential for food pocketing and periodontal disease. To date this disorder appears to be rarely diagnosed in horses, although this may be due to poor recognition of the condition. Abnormal enamel morphology has been observed in mandibular CT, with branching in the regions of pulp horns 1–2 (Fig. 9.15). As enamel acts as the 'scaffolding' and template for subsequent deposition of dentine and cementum, these latter tissues will follow this abnormal pattern and consequently all three calcified tissues are dysplastic.

Hypohidriotic ectodermal dysplasia is the most common form of human ectodermal dysplasia and is characterized by varying degrees of oligodontia and dental dysplasia, together with decreased sweating (hypohidrosis) and decreased hair development (hypotrichosis).[47] Coat changes have been reported in cases of both equine oligodontia and dental dysplasia, suggesting similarities between these two conditions.[46] Hypohidriotic ectodermal dysplasia in man is an X-linked, recessive trait and carrier females may be asymptomatic. It is unknown if these two congenital dental disorders reported in horses are hereditary.

Hypoplasia of equine dental tissues is most commonly recorded in infundibular cementum. Central infundibular cemental hypoplasia is the most common true developmental disorder.[24] It is often not possible to establish its presence by examining the occlusal surface of a young tooth. Baker reported 43 per cent of infundibula in erupted teeth to contain connective tissue remnants at their apex.[48] This condition has been incorrectly referred to as infundibular necrosis and infundibular caries.

All incisors and maxillary CT have some degree of infundibular cemental hypoplasia that may predispose to endodontic disease.[49] Recent evidence may suggest that additional vascular supplies to cementum exist at the base of at least some infundibulum (J Easley, personal communication); however, for the majority of maxillary CT, premature eruption, premature removal of protective deciduous teeth ('caps'), or an otherwise compromised infundibular blood supply (e.g. excessive deposition of infundibular cementum near the occlusal surface constricting the blood supply to cementocytes deeper within the infundibulum) may all be reasons for inadequate cementum deposition. The resultant 'hypoplastic' infundibular region is then prone to

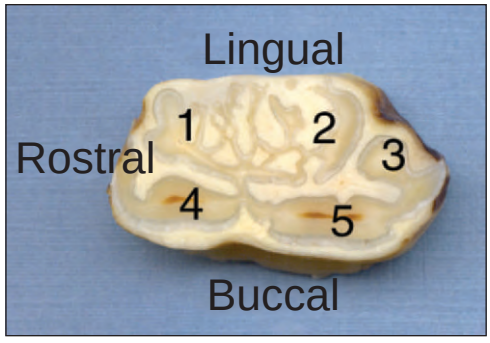


Figure 9.15. Mandibular cheek tooth (Triadan 307) with enamel dysplasia of pulp horns 1 and 2, both of which have an abnormal branched appearance. Carious cementum is also present, i.e. the darker stained region of peripheral cementum near pulp horn 3, which resulted from local periodontal food pocketing.

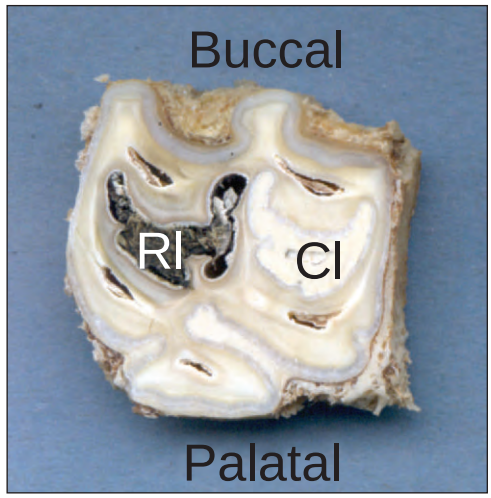


Figure 9.16. Maxillary cheek tooth (Triadan 210) with a normal level of cementum deposition in the caudal infundibulum (CI) but with marked infundibular cemental hypoplasia of the rostral infundibulum (RI). Both infundibula were intact at the occlusal surface and organic matter within the RI was mainly blood vessel remnants. No caries of the infundibular cementum or adjacent infundibular enamel were present.

packing with food material from the oral cavity (Fig. 9.16). Central infundibular cemental hypoplasia is often found to affect a single infundibulum within the tooth, and this is usually the rostral infundibulum, as seen in the CT computer-assisted tomography (CAT) scan in Fig. 9.17.

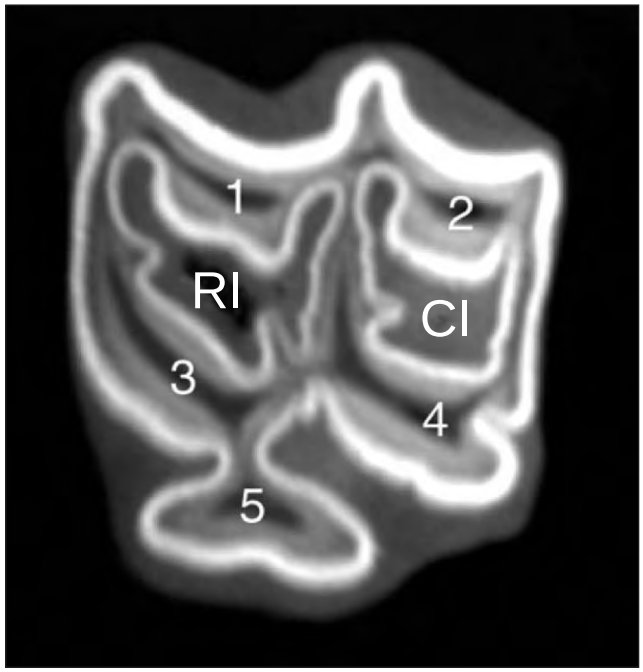


Figure 9.17. Computer-assisted tomograph (CAT scan) of a maxillary CT with infundibular cemental hypoplasia of its rostral infundibulum (RI). The caudal infundibulum (CI) appears radiographically normal. By using a CAT scan we can determine if the pulp horns (1–2, 3–4 or 3–5) connect at this level. Similarly, we may non-invasively determine if there is any possible communication between the infundibulum and dentin or pulp.

As with dentine and odontoblasts, cementum can be deposited by cementocytes throughout the life of the tooth while it remains within the dental alveolus. Cementocytes can respond quickly to harmful stimuli with further rapid deposition of cement when appropriately stimulated.[50] The process whereby infundibular cemental hypoplasia progresses to result in an apical abscess is discussed under infundibular caries.

A recent study by Dacre noted peripheral cemental hypoplasia in 41 per cent of mandibular CT extracted with associated apical infection.[20] The degree of hypoplasia varied from zones of hypoplastic cementum within the rostral groove to almost complete cemental aplasia on the lingual aspect of one tooth. Where large areas of the peripheral cementum are hypoplastic, this can affect the ability of the periodontal ligament to support the tooth within the alveolus and may predispose to periodontal disease. Peripheral cementum is nourished by the vasculature of the periodontium and gingiva, and once this contact is lost, for example following eruption above the gingiva to become clinical crown, or with advanced periodontal disease, peripheral cementum may be considered an inert tissue.

Enamel hypoplasia has been histologically identified both within the infundibulum of maxillary CT (Fig. 9.18) and the peripheral enamel of mandibular CT (Fig. 9.19). Imaging calcified dental tissues with CAT enabled the area of interest to be identified prior to sectioning. This author has observed enamel hypoplasia in both normal CT and in CT extracted because of apical abscessation. Embryological enamel may be thought of as the tooth's developmental skeleton and template, with any defects in its morphology being directly transferred to the tissues (either dentine or cementum) surrounding it. Consequently in teeth with hypoplastic enamel, the adjacent peripheral tissues will often be reduced

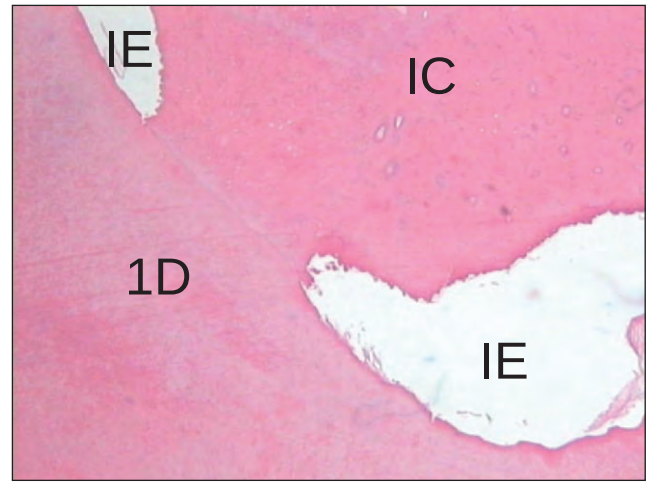


Figure 9.18. This longitudinal section of the apical aspect of a maxillary CT infundibulum shows where infundibular cementum (IC) is in direct contact with primary dentin (1D), the infundibular enamel (IE) having failed to develop in this local region, i.e. localized enamel hypoplasia. As this is a decalcified histological section, the region previously filled with (highly mineralized) infundibular enamel is empty (H and E).

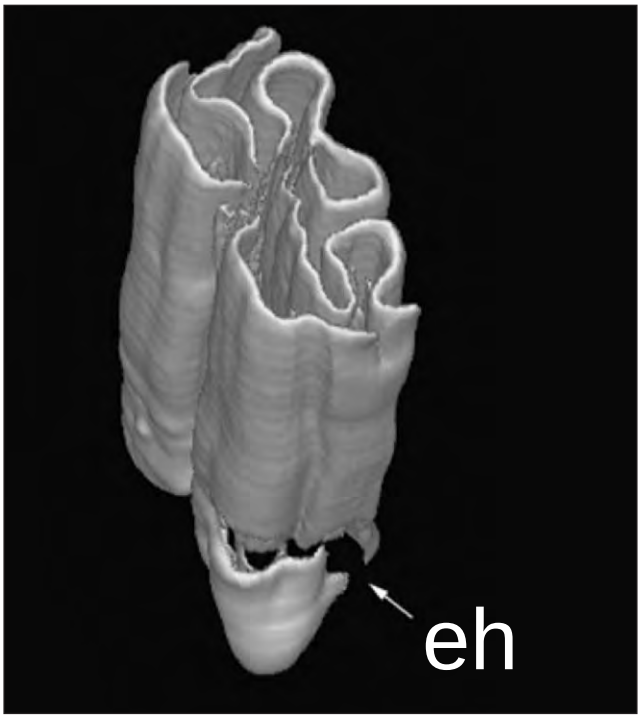


Figure 9.19. A computer tomograph scan of a mandibular CT with enamel hypoplasia (eh), the enamel having failed to develop on the caudal aspect, *circa* one-third from the enamel's apical limit. Decalcified histology confirmed the enamel to be absent in this area.

(hypoplastic) or dysplastic. This appears to be more evident in cementum than dentine and when occurring in peripheral cementum will result in reduced periodontal attachment, with the tooth becoming unstable. When enamel is so dysplastic that the overall shape of the tooth is altered and it no longer sits correctly in the tooth arcade or row, this allows rapid progression of food pocketing and periodontal disease with subsequent apical abscess formation. It has yet to be established in horses whether the use of drugs, such as tetracyclines, during periods of amelogenesis (enamel formation) affects enamel formation as recorded in other species.

Developmental conditions arising at eruption

It is believed that when there is excessive rostrocaudal compression within a cheek tooth row, a permanent tooth may become vertically impacted between the two adjacent (deciduous or permanent) CT. This most frequently involves the Triadan 07s and 08s, being the last permanent CT to erupt.[42,51] The potential for deciduous teeth to become trapped between adjacent teeth in the row (i.e. retained caps) is also only possible in these two cheek teeth positions. Mild compression of either the erupting permanent or retained deciduous teeth remnants may result in 'eruption cysts' (3- or 4-year-old bumps) bilaterally on the ventral mandible or rostrodorsal maxilla. If crowding is severe and a tooth becomes further impacted, it may result in a more pronounced and often unilateral swelling and may be associated with heat and pain. During this time, the tooth and associated region are hyperemic, which may predispose to an anachoretic pulpitis. This is when bacteria, carried in either the blood stream or local lymphatic system, lodge within an area of pulp that is already compromised by being mildly inflamed or oedematous.[52,53] Such a disorder may then develop into a microabscess within the pulp before progressing to become an apical abscess. Impaction of incisors may also occur, notably when a deciduous tooth has become wedged between its neighbors. Treatment of impactions is discussed in Chapter 16.

A diastema (pl. diastemata) may be thought of as the opposite of an impaction, being a space between two adjacent teeth. Developmental diastema result from teeth either having insufficient angulation to achieve good compression between adjacent teeth, or because the embryonic teeth developed too far apart. Voss noted that horse teeth taper toward their apices, and as they continue to erupt the occlusal surface reduces in size, with potential acquired diastema resulting.[37] Diastema have been functionally termed as either 'open,' when they do not trap food, and closed (or valve) diastema when they do.[54] Having trapped food they are likely to predispose to local periodontal disease.

Teeth may also become rotated or otherwise displaced due to developmental malpositioning of dental buds, or due to overcrowding before, during or following eruption. Dixon *et al.* reported 70 per cent of cases of CT displacements were developmental in origin due to overcrowding of the CT row at time of eruption, and frequently found displaced teeth to be bilateral.[42] The remaining cases of developmental CT displacement were caused by abnormal tooth positioning. This may result in the non-eruption of the displaced teeth if they are sufficiently horizontal as reported by Becker[55] and Edwards.[56] When displaced teeth are associated with apical abscessation, it is usually attributed to concurrent advanced periodontal disease, which is in turn caused by diastema between the displaced and adjacent tooth.

Acquired conditions

Pulpar exposure

Being hypsodont, equine teeth have a prolonged eruption throughout most of their life.[29] This is an evolutionary adaptation to compensate for wear at the tooth's occlusal surface (at a rate of approximately 2–3 mm per year) due to the prolonged mastication (up to 20 hours per day) of forage which may contain abrasive silicates or phytoliths.[57] To avoid pulpal exposure on the occlusal surface, dentine is continually laid down by odontoblasts throughout the life of the tooth.[19] Excessive attrition of dentine with resultant pulpar exposure has been proposed as a route of infection into equine apical tissues.[23,40,58] When dental attrition exceeds the rate of secondary dentine deposition, the pulp will eventually become exposed. The rate of dentine formation

is dependent on the health of the layer of odontoblasts present at the periphery of the pulp.[59] If this layer becomes compromised, either directly (e.g. from trauma or from thermal damage during dental treatments), or indirectly (e.g. from decreased or total loss of vascular supply) the resultant decreased dentine production may be insufficient to prevent pulpar exposure. Where there has been complete necrosis of the dental pulp, no further dentine deposition is possible, and with continued attrition at the occlusal surface, the pulp will eventually become exposed to the oral cavity. In all cases of pulpar exposure, the outcome is dependent on the tooth's ability to withstand prolonged bacterial invasion.

'Spontaneous' pulpar exposure on the occlusal surface of equine CT was reported in three mandibular and one maxillary cheek tooth by Becker,[55] who also cited three previous authors as recording an incidence of 2.8–3.5 per cent of this disorder in equine CT. In a survey of 355 abattoir skulls in Ireland, Wafa found 6.5 per cent to have dental pulp exposed.[40] Dixon *et al.* also clinically recognized pulpar exposure in some cases of apical infection, but commented that such lesions may not be detected unless teeth are specifically examined for them.[18] This was in fact the case, as on subsequent examination of extracted apically infected CT from that study, many were found to have occlusal pulpar exposure of non-traumatic origin that was previously undetected clinically. Multiple pulpar exposure was found in 44 per cent and 41 per cent of apically infected maxillary and mandibular CT respectively.[20] Examples of these are seen in Fig. 9.20. Equine practitioners should be aware of the potential significance of such lesions and should look for such (often subtle) lesions on the occlusal surface, including using a modified dental pick explorer.

In brachydont teeth that develop very fine vasculature at the apical foramen, bacterial invasion following occlusal pulpar exposure in adults is likely to result in pulpitis with resultant compression of its apical blood vessels leading to pulp ischemia and probable pulpar death. Young equine hypsodont teeth may often resist such pulpar infection and inflammation following pulpar exposure. This is due to the relatively large apical foramen and large vasculature present, which allow pulpitis to occur without the resultant edema occluding blood supply with consequent ischemic necrosis.[60]

Pulp stones

Pulp stones, also termed denticles (i.e. concentric areas of calcified dentine), have been observed in equine teeth both within viable pulp (free stones) and in areas replaced with secondary dentine (Fig. 9.21A,B). Their presence is usually stated to be evidence of pulpar irritation or inflammation.[28] Such inflammation may arise from focal areas of anachoretic pulpitis, or from other noxious stimuli including chemical, bacterial, vibrational (mechanical) or thermal stimuli.[61] Their formation creates a corresponding decrease in functional size of the pulp chamber that may compromise pulpar microcirculation. This in turn may affect the rate of dentine production. Their frequent occurrence in grossly normal teeth may indicate that some pulp stones can exist without compromising pulp vitality.[14]

Apical abscesses

The terms 'apical abscess,' 'apical infection,' 'periapical abscess,' 'dento-alveolar infection,' 'dental sepsis' and 'tooth root abscess' have been used interchangeably because none describes the disorder(s) fully.[62] The term 'apical abscess' more correctly refers to those abscesses resulting from acute ischemic pulpar necrosis and subsequent abscess formation following vascular occlusion in the apical foramina of brachydont teeth. Use of 'periapical' to describe the condition excludes infection of endodontic tissues, which may be inaccurate. 'Dento-alveoar' infection implies that both the tooth and the alveolar socket are infected, which may not be true in early cases. It does, however, describe the extent of the tissues infected in advanced cases, when the supporting bones become involved as frequently occurs in hypsodont teeth.[51] 'Dental sepsis' can mean tooth infection, which might also include carious attack. 'Tooth root abscess' is also inaccurate because in horses apical abscesses may occur prior to the development of any true roots (i.e. apical areas without enamel), especially in mandibular CT.

Aside from their frequency of occurrence in horses,[3,18,40,63] apical abscesses are of major clinical significance for three

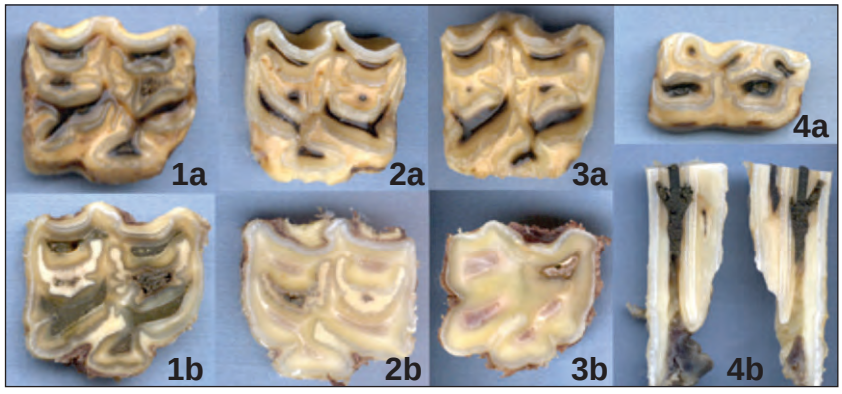


Figure 9.20. Pulpar exposure on the occlusal surface is difficult to detect on clinical examination. 1a shows the occlusal surface of a maxillary CT with five pulp horns exposed, with food deeply impacted down all pulp horns as shown in 1b a transverse section of the same tooth 4 cm below the occlusal surface. 2a is a normal tooth with a moderate degree in staining of its secondary dentin. 2b shows how translucent normal pulp appears. 3a has exposure of pulp horn 2, the consequences of which are seen image 3b. 4a shows a mandibular tooth with all five pulps exposed. A longitudinal section (4b) of this tooth reveals the extent of food impaction and caries within pulp horn 4.

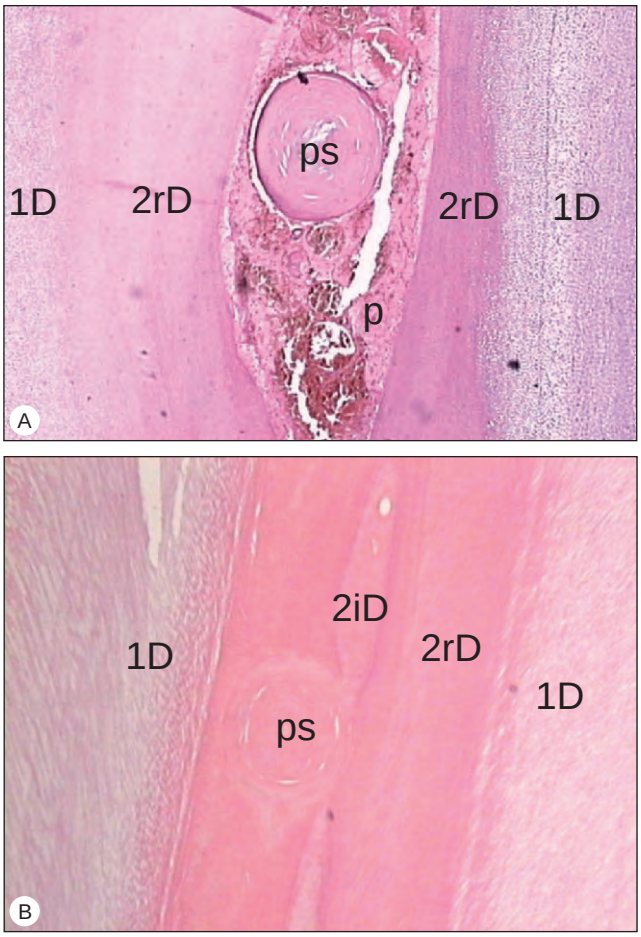


Figure 9.21. (A) A single pulp stone (ps) has formed in the middle of this partially autolyzed dental pulp. As it remains entirely within the pulp it is termed a 'free' pulp stone (denticles). Regular secondary (2rD) and primary (1D) dentin are present surrounding the pulp (H and E). (B) This pulp stone (ps) has become surrounded by regular (2rD) and irregular (2iD) dentin. The sigmoid curvature of dentinal tubules (as they head from the pulp to the amelodentinal junction) is partially visible in the primary dentin (1D) (H and E).

main reasons. Firstly, the condition most frequently affects younger animals (e.g. mean ages of 5 and 7 years for mandibular and maxillary apical abscesses, respectively in one study).[18] Secondly, the disease causes infection of the dental alveolus and supporting facial bones (possibly including the paranasal sinuses), and thirdly, to date the most common treatment for the condition involves removal of the tooth (either by oral extraction, buccotomy or repulsion) often with prolonged postoperative sequellae and commits the animal to increased prophylactic dental care for the rest of its life.[18]

In addition to classification of apical abscesses being 'primary' (i.e. of unknown etiology) or 'secondary' to some other disorder, the latter may be further classified as being either 'developmental' or 'acquired' in origin. Those classified as developmental include: secondary infections arising from polyodontia; dental dysplasia or hypoplasia; dental displacement or rotation. Apical abscesses arising from acquired conditions include post-traumatic or idiopathic fracture (affecting dental tissue, supporting bones, or both); caries; wear disorders (including acquired displacements); primary periodontal disease and following anachoretic pulpitis. This recent classification is a reflection of the numerous potential etiologies for equine apical abscesses. With continued research into this area, apical abscesses may eventually be classified as being of primary origin when they result from anachoretic pulpitis, and secondary by all other etiopathogenic means.

As noted, pulpar exposure has been proposed as a major etiopathological factor for the development of apical abscesses.[21,40,58] Many apical infections, especially of mandibular CT, occur soon after eruption when eruption cysts and an anachoretic pulpitis are believed to be present. In these young teeth very little secondary dentine has been laid down and consequently the pulp is much nearer to the occlusal surface than in mature teeth. The relatively high incidence of pulpal exposure found in teeth originally classified with primary apical abscesses (i.e. an abscess being present with no other obvious dental pathology present) raises the issue as to whether the pulpar exposure is the cause or effect of apical abscesses. A study by the author showed 44 per cent of maxillary CT orally extracted with primary apical abscesses had multiple pulpar exposure.[14] As no teeth were found with a single pulp exposed, it is likely the pulpar exposure was the result rather than the cause of the apical abscessation. When dentine production is either delayed or disrupted by pulpar infection (or other types of pulpitis), dentine production is unable to meet the rate of occlusal dental attrition with subsequent exposure of the pulp cavities. This could explain why sudden dental pain, which might be expected if a pulp were suddenly exposed, is often not recorded in horses suffering from a dental apical abscess. Pulpar exposure may also arise through idiopathic and traumatic dental fractures. Traumatically induced apical infection may be caused by dental fractures (Fig. 9.22), fractures of supporting bones exposing an apical route for infection, or by iatrogenic pulp exposure during dental treatment.

The presence of food within hypoplastic infundibular cementum will promote some degree of infundibular caries, which, depending on bacteria present, amount and type of food, and time present, has been proposed as a cause of apical infection.[48,64] When caries penetrates through the residual infundibular cementum, infundibular enamel, and into the dentinopulpal complex, a bacterial pulpitis can result. Alternatively, the tooth may be structurally weakened by destruction of the infundibular cement and enamel. If lesions in both infundibula coalesce, this implies that the carious process has invaded and destroyed not only infundibular cementum and enamel, but also the dentine present between the two infundibula. Subsequently midline sagittal fractures can occur, exposing deeper apical tissues.

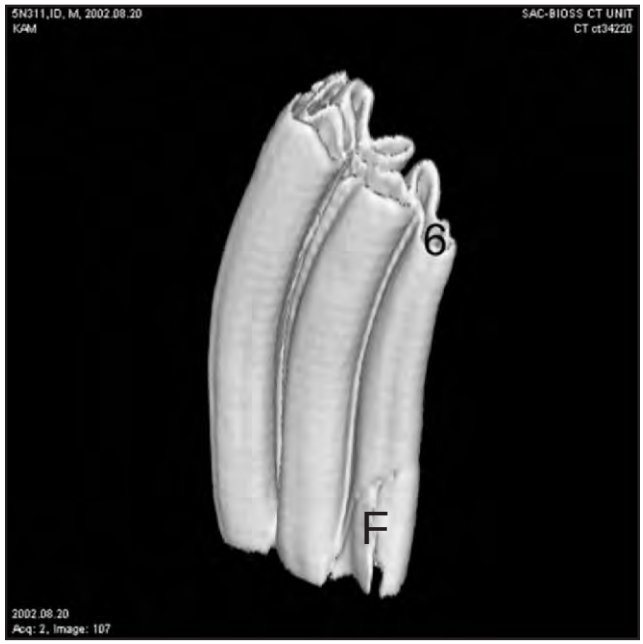


Figure 9.22. Having removed the imaged dentin and cementum from this CAT scan of an intact whole mandibular (Triadan 311) CT, a fracture (F) is clearly identifiable in the apical enamel. It is most likely this fracture arose at time of extraction. Note the curvature of this sixth CT and presence of the additional (6) caudal pulp horn.

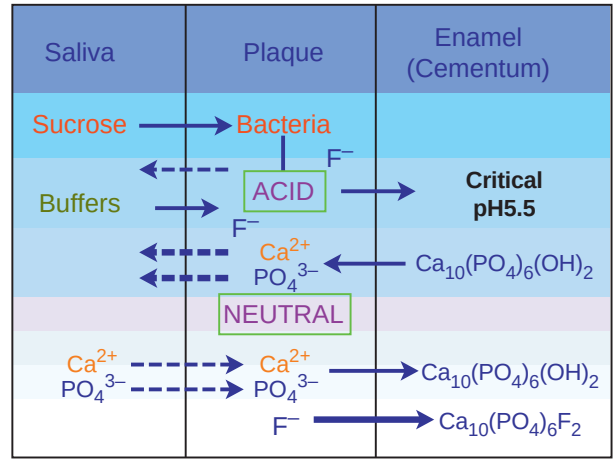


Figure 9.23. Diagrammatic representation of principle biochemical aspects of factors influencing caries under acidic (pH < 5.5) conditions (favoring caries) following degradation of carbohydrates (e.g. sucrose) and at a neutral pH, e.g. following buffering by saliva.

In either case, apical abscessation will follow. Despite many maxillary CT having caries visible on the occlusal surface of their infundibulum, it is rare for these to progress to become apical abscesses, unless the tooth fractures through this weakened area.[20]

Caries

Caries is defined as a disease of the calcified dental tissues resulting from the action of micro-organisms on carbohydrates and is characterized by both demineralization of the inorganic part, and subsequent destruction of the organic part of the tooth.[61] It is a complex and dynamic physiochemical process involving the movements of ions across the dental–oral cavity interface (Fig. 9.23), as well as biological processes between bacteria and host defence mechanisms.[65]

The generally accepted etiology of caries has remained largely unchanged since it was first postulated by WD Miller in 1889. His acidogenic theory proposed that acid, resulting from the fermentation of dietary carbohydrates by oral bacteria, led to progressive decalcification of tooth substance with subsequent destruction of the organic matrix. Oral bacteria make up the bulk of dental plaque, suspended in an amorphous matrix incorporating salivary mucoids and extracellular bacterial polysaccharides. Clean human enamel is covered within a few seconds by an adsorbed glycoprotein layer (the pellicle) from the saliva to which bacteria from the oral cavity adhere. *Streptococci* bacteria are the first to colonize the newly deposited pellicle (within hours) and remain the dominant group of organisms in human plaque for around 7 days if left undisturbed; subsequently anerobic bacteria begin to compete for nutrients, becoming the dominant group by 14 days.[65] A thorough bacteriological cataloging of both commensal and pathogenic species in the equine oral cavity has yet to be undertaken.

Dietary sugars are metabolized by bacteria to lactic acid, although acetic and propionic acids may also be produced. Plaque pH may fall by 2 units within 10 minutes of sugar ingestion by humans. At around pH 5.5 mineral ions are released from calcium hydroxyapatite crystals in mineralized dental tissues, which then diffuse into the adjacent plaque, and thereafter the oral cavity. With saliva aiding the tooth in buffering this acidic attack, the plaque will eventually return to a neutral pH (around 7.0). At this point the plaque may be supersaturated with mineral ions, some of which will reprecipitate back onto the tooth surface. Fluoride ions (usually present in higher concentration in plaque than saliva) aid in this mineral reprecipitation by forming fluorapatite when substituted for the more soluble hydroxyl ions (Fig. 9.23). Mineral loss principally occurs from teeth during prolonged acidic attacks. Baker demonstrated the presence of plaque on equine teeth using a disclosing dye.[63] There has been little work done on the role of plaque in equine dental pathology.

Early studies in horses reported incidences of infundibular caries to be as high as 79–100 per cent in certain age populations.[38,39] It is not certain, however, if infundibular hypoplasia was incorrectly identified as infundibular caries in these studies. This may be one reason why Brigham and Duncanson reported a much lower incidence of 12 per cent.[41] Infundibular caries is much more common in maxillary CT than incisors and grossly appears as a darkly stained region within the infundibular cemental lakes (Fig. 9.24). This dark staining may extend beyond the infundibular cementum into both the enamel and even adjacent dentine. Honma (1962) described a grading system that is still

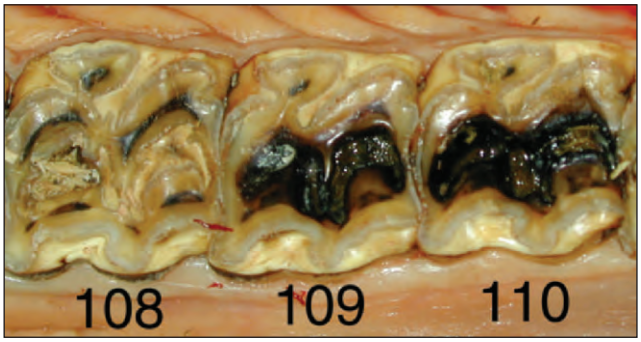


Figure 9.24. Infundibular caries is present in both rostral and caudal infundibula of maxillary CT 109 and 110. The infundibula have not coalesced at this stage but both the infundibular enamel and dentin between the two infundibula are carious.

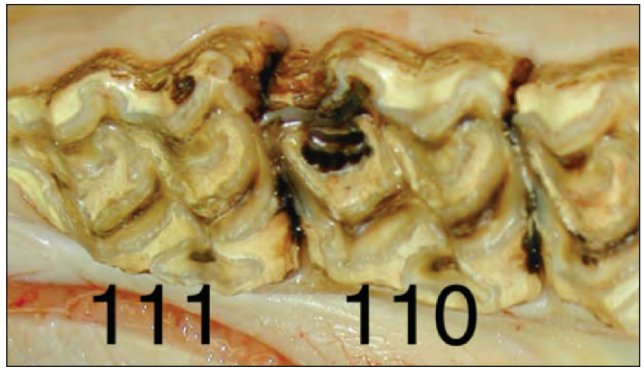


Figure 9.25. Caries is present around pulp horn 2 of tooth 110. Enamel and cementum have been lost buccally to this area, either through caries itself or because the weak carious area has fractured during mastication and broken away. There is also a small area of peripheral cement caries adjacent to pulp horn 1 of tooth 111. Additionally there are small carious regions of cementum between 109–110 and 110–111 (arrow).

used today.[38] Recently, however, with the advent of treatments for infundibular caries, a more detailed classification system has been required, as detailed below (modified Honma classification):

- zero-degree caries: no evidence of caries on a macroscopic level, but may include hypoplastic tissue (i.e. central infundibular cemental hypoplasia)
- first-degree caries: caries only affecting cementum – this may vary considerably from small darkly pitting superficial spots, to extensive destruction and loss of cementum. On this basis it may be divided into class 1 and 2 respectively
- second-degree caries: extends beyond cementum to affect adjacent enamel
- third-degree caries: extends beyond cementum to affect enamel and dentine
- fourth-degree caries: where caries has progressed to affect the integrity of the entire tooth, i.e. development of an apical abscess or tooth fracture
- [fifth-degree caries: caries resulting in tooth loss – this category is somewhat redundant in contemporary work.]

The above classification system is equally suitable for carious lesions found in peripheral calcified tissues, notably peripheral cementum (Figs 9.25–9.27). To date the pathological significance of peripheral cemental caries is unknown. By weakening and removing occlusal cementum it may contribute to an increased rate of occlusal wear, or even to the development of diastema or periodontal disease. Removal of peripheral cementum also makes proud areas of brittle enamel on the occlusal surface more prone to fracture.

Caries of dentine is commonly seen in CT with pulpar exposure (Figs 9.20, 9.28 and 9.29), grade 4 infundibular caries (using the modified Honma classification system), and dental fractures (Fig. 9.30). It is rarely found on exposed occlusal dentine, with this being mostly sclerotic; however, pioneer organisms (bacteria) have been seen under both

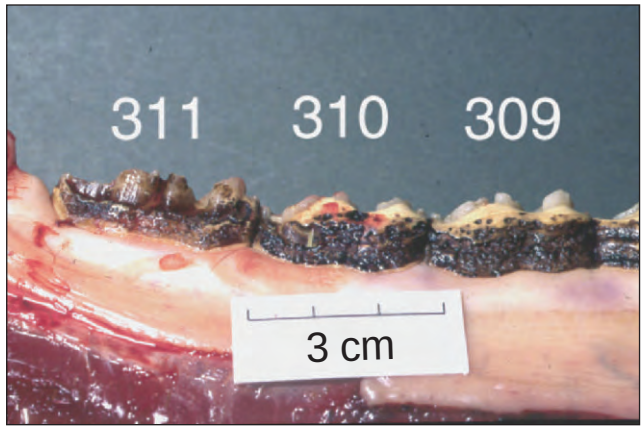


Figure 9.26. Widespread peripheral cemental caries of mandibular CT 309–311. Note how the caries has removed much of the supporting lingual cementum from the clinical crown of 311, leaving its discolored enamel.

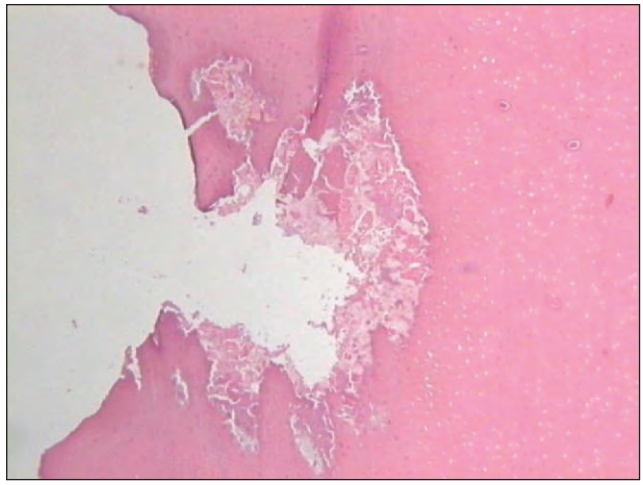


Figure 9.27. Caries of peripheral cementum in a decalcified section from a mandibular CT. This shows the characteristic pitting nature of these lesions (H and E).

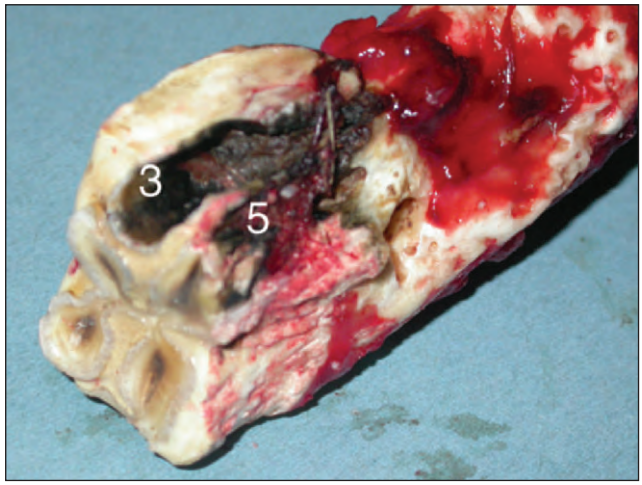


Figure 9.28. Advanced caries of pulp horn 3 (3) which extends into pulp horn 5 (5) in this mandibular CT. This tooth was extracted orally and crush marks from the extraction forceps are seen on the buccal face of its clinical crown. Note the hyperplastic peripheral cementum on the reserve crown that has been laid down in response to the apical abscess present.

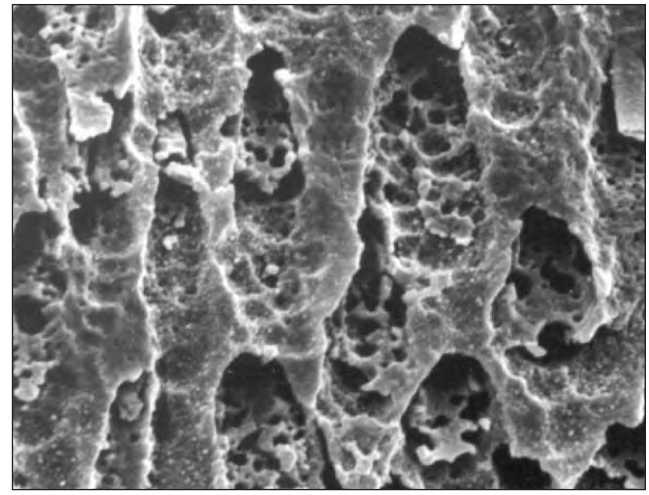


Figure 9.30. A scanning electron micrograph of carious primary dentin present on a mandibular CT following a dental fracture. Note the fenestrated appearance of the carious dentinal tubules.

light microscopy and transmission electron microscopy advancing down dentinal tubules in both clinically normal and diseased CT (Fig. 9.31). Those in normal teeth have had their advance down the dentinal tubule stopped a few microns from their point of entry into the tubule, while those in diseased teeth have been found in dentine adjacent to dental pulp.[14]

Fractures

Apart from numerous articles on the surgical repair of displaced and fractured teeth, there is a paucity of published information on the incidence, histology, ultrastructural anatomy and pathogenesis of equine dental fractures. Becker reported an incidence of fractures in all equine teeth to be 0.71 per cent in 30,000 cavalry horses, while Geres found radiographic evidence of fractures in 3.5 per cent of 500 skulls, although this higher incidence may have been caused by post-mortem fractures.[55]

A study by Dixon *et al.* of 400 cases referred with dental disorders found complicated traumatic fractures (i.e. with pulpal exposure) of the incisors and/or their supporting bones in 25 per cent of incisor cases and 'idiopathic' incisor fractures in 6.8 per cent of cases.[43,44] Incisors were usually fractured in a transverse plane in contrast to CT fractures, which were reported to be usually sagittal in nature. The incidence of CT fracture in cases was 7.5 per cent and 6.9 per cent for traumatic fractures and idiopathic fractures

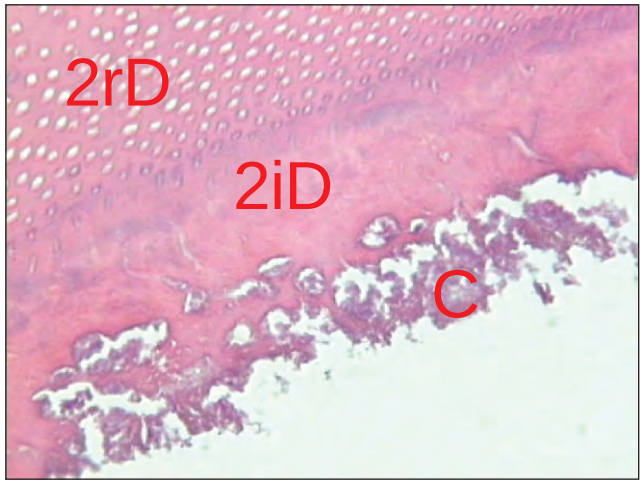


Figure 9.29. A decalcified transverse histological section of a CT following pulpar exposure, when food and bacteria invaded, destroying any remaining pulp before progressing to attack secondary irregular dentin (2iD) at the carious pulp margin (C). Secondary regular dentin (2rD) will often be attacked subsequently (H and E).

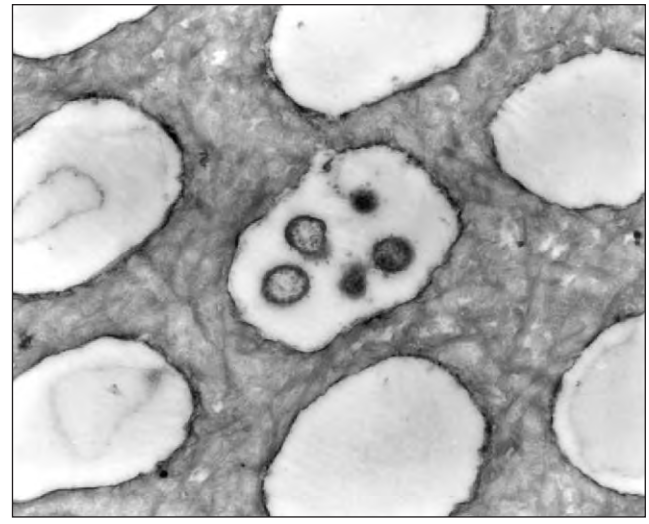


Figure 9.31. Pioneer organisms (bacteria) are present within the central dentinal tubule in this transmission electron micrograph. Such micro-organisms may invade down exposed dentinal tubules and infect the pulp if the host defence mechanisms are inadequate.

respectively. Dental trauma is common in horses, usually resulting from kicks, biting inanimate objects, cribbing, and high-speed encounters with immobile objects.[66] Of those categorized by Dixon as having suffered CT dental trauma, 71 per cent were mandibular and 29 per cent maxillary; 11 of the 18 (61 per cent) cases of mandibular CT trauma resulted from external trauma (kicks most frequently), compared with three of the eight (38 per cent) maxillary CT cases.[44] The two other mechanisms of trauma identified were bit trauma and iatrogenic trauma during dental treatment. It was noted that iatrogenic fractures were more likely to occur in younger (age <8 years) teeth when molar cutters were used to remove overgrowths; at this age, little secondary dentine has been laid down to protect the brittle enamel.

'Idiopathic' dental fractures were classified when no definite etiology could be identified.[44] Of the 24 cases (27 CT) with iatrogenically fractured CT teeth, 22 (81.5 per cent) were maxillary and 5 (18.5 per cent) were mandibular, with maxillary 09s being the most commonly affected tooth (12/27, 44 per cent). Gross caries with subsequent midline sagittal fracture was recognized in eight (30 per cent) maxillary CT, with lateral 'slab' fractures in 14 CT (52 per cent) and medial slab fractures in a further three CT (11 per cent). It was reported that these slab fractures seldom involved the pulp cavity, involving less than 20 per cent of the total tooth width. It was also noted that only 24 per cent of teeth with slab fractures developed apical infections, compared with 100 per cent of the midline sagittally fractured teeth. The remaining two teeth (7 per cent) had lost oblique aspects to the occlusal aspect of the clinical crown.

There are several classification systems for fractured teeth in human dentistry. One widely adopted is that of Ellis,[67] who divided traumatized teeth into nine classes:

1. simple fracture of crown enamel, involving little or no dentine
2. extensive fracture of crown, involving considerable dentine but not the dental pulp
3. extensive fracture of the crown, involving considerable dentine and exposing the dental pulp
4. the traumatized tooth becomes non-vital, with or without loss of crown structure
5. tooth lost as a result of trauma
6. fracture of the root, with or without loss of crown structure
7. displacement of the tooth, without fracture of crown or root
8. fracture of the crown *en masse* and its replacement
9. traumatic injuries to deciduous teeth

The above classification system is a good starting point to classify equine dental fractures; however, the complex nature of equine CT requires further details to be recorded. In a recent study, dental fracture planes were examined in 20 equine CT with idiopathic fractures.[68] These fracture planes tended to run through lines of normal anatomical weaknesses, and in contrast to Dixon *et al.*'s assumptions,[44] included exposure of pulp in 18/20 cases examined (Figs 9.32 and 9.33). Fifty per cent of iatrogenically fractured maxillary CT were lateral slab fractures running through pulp horns 1 and 2 (Fig. 9.32). Twenty per cent of maxillary cases had gross caries present resulting in midline sagittal fracture with no pulpar exposure. Both teeth had coalesced rostral and caudal infundibula and their fracture resulted in oral exposure of the alveolar and apical regions. The remaining three cases were medial slab fractures, with all three having differing fracture planes running through pulp horns 3–4, 3–5 and 4.

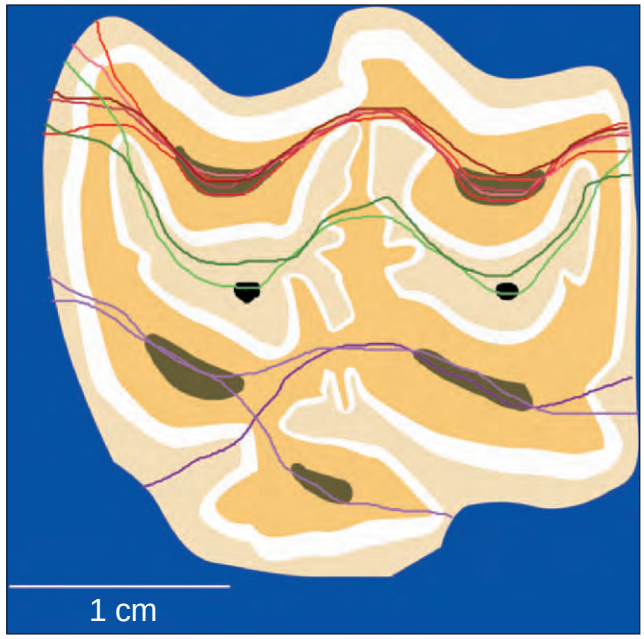


Figure 9.32. Diagrammatic representation of fracture planes through 10 maxillary CT – five through pulp horns 1 and 2 (red), two through both infundibula (green) and three through palatal pulp horns 3, 4 or 5 (purple).

In 10 cases of mandibular iatrogenic CT fracture examined in the above study, nine had a fracture plane running through buccal pulp horns 4 and 5 (Fig. 9.33). The fracture in the remaining case ran obliquely through pulp horns 1, 4 and 5. Such a high proportion of lateral slab fractures in mandibular CT is attributable to two key factors. Firstly, the movement of the mandible during the so-called 'power-stroke' when mastication is in a dorsomedial direction, with a massive force being repeatedly transferred to the lateral aspects of the tooth on each grinding stroke. Secondly, the amount of calcified tissue supporting the lateral aspect of the mandibular CT at their rostral and caudal aspects is lowest where the fourth and fifth pulp horns are present (Fig. 9.34). This is particularly so in younger teeth, when these two areas of future pulp horns are still part of a common pulp chamber.

It was found that most maxillary and mandibular idiopathic CT fractures involved all three mineralized dental tissues and that the fracture planes were normally perpendicular

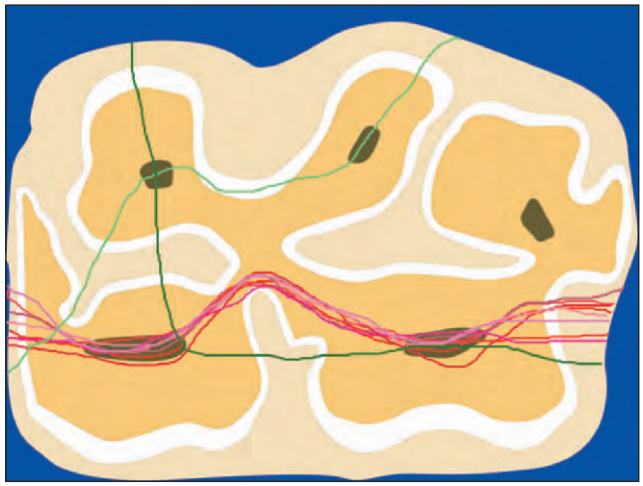


Figure 9.33. Diagrammatic representation of fracture planes through 10 mandibular CT – eight through pulp horns 4 and 5 (red), one passing through pulp horns 1, 4 and 5 (dark green) and one through pulp horns 1 and 2 (light green). A magnified SEM image of the area within the rectangle is seen in Fig. 9.34.

to equine type 1 enamel plates (Fig. 9.34). The fracture sites involved were points of anatomical weakness where these mineralized dental tissues are usually at their thinnest, the exception being midline saggital fractures seen in maxillary CT with advanced infundibular caries, where much of the calcified dental tissues had already been lost. No slab fractures involved the full length of the tooth, with most fractures ending at the level of the alveolar crest. This is in contrast to midline sagittal fractures where the teeth usually split into two halves over their full length. Although slab fractures usually result in pulpar exposure, apical infection in this category is much lower (24 per cent) than observed for midline sagittal fractures (100 per cent). This indicates that different treatment options are appropriate for different types of fractures.

The incidence of pulpar exposure in CT with apical abscesses differs from the incidence in CT with idiopathic

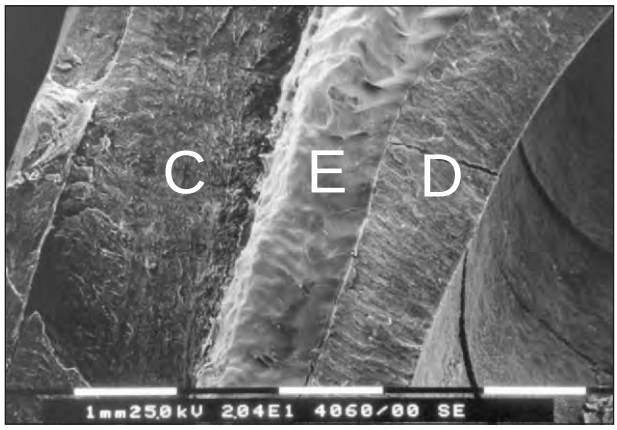


Figure 9.34. Scanning electron micrograph of a mandibular buccal 'slab' fracture showing that cementum (C), enamel (E) and dentin (D) are all involved in the fracture plane. Note the irregular surface of the fractured enamel.

fractures.[20,68] Histological examination of sectioned fractured teeth often shows no evidence of pre-existing pulpar exposure.[14] These findings indicate pulpar exposure is not a precursor to idiopathic cheek teeth fracture, but rather these fractures occur as previously suggested, at sites of anatomical weakness.

Exposed fractured dentine surfaces often show an intricately fenestrated lattice of dentinal tubules – the result of carious attack following fracture (Fig. 9.30), or may be worn smooth with no evidence of caries. Odontoblast processes may also be observed on exposed fracture surfaces and these usually appear vital in the initial post-fracture period. This contrasts to the odontoblast processes observed on the occlusal surface of normal teeth by Kilic *et al.*, who postulated that they may be just their calcified remnants.[23]

Periodontal disease (see chapter 10)

In 1906 Colyer described periodontal disease as the 'scourge of the horse', finding this disease present in approximately one-third of 484 heads examined. Baker recorded an incidence of 60 per cent periodontal disease in horses aged over 15 years[39] and Wafa (1988) found periodontal disease to be present in 37 per cent of 355 abattoir skulls examined, recording a higher incidence of periodontal disease in older animals (60 per cent in those over 20 years of age).[40]

Gingivitis is used to designate inflammatory lesions confined to the marginal gingiva. It is generally accepted that with time some cases of gingivitis will progress to periodontitis. Periodontitis (also termed, alveolar osteitis, or periostitis) is present once lesions have extended beyond the gingiva to include destruction of the underlying connective tissue and loss of alveolar bone (by resorption).[69] It is a complex disease process involving genetic, microbial, immunological and environmental factors that determine the risk of acquiring and progression of the disease. As with caries, formation of dental plaque is considered to be the main etiological factor for periodontal disease in brachydont species. If unresolved, periodontal disease may lead to marked loss of alveolar bone, invasion of periapical tissue, septic pulpitis, and eventual tooth loss.[70] In severe cases (e.g. some cases of severe diastema) the disease may progress to widespread osteomyelitis and even necessitate euthanasia on welfare grounds. Maintenance of a healthy periodontium is reliant on the normal forces of mastication acting on the periodontal ligament. Wear disorders, ranging from sharp points causing buccal ulceration to severe shear mouth, will cause unevenly distributed shear forces to act on the periodontium. Additionally, oral pain may increase food stagnation and decrease saliva flow around gingivae, allowing bacteria to flourish. This is more apparent in the caudal regions of the mouth where reduced movement of ingesta results in feed stagnation.

In humans, periodontal disease is of major importance, with 50 per cent of adults aged 55–64 in one survey having severe (>4 mm periodontal attachment loss) periodontal disease.[71] In humans, cats, and dogs periodontal disease

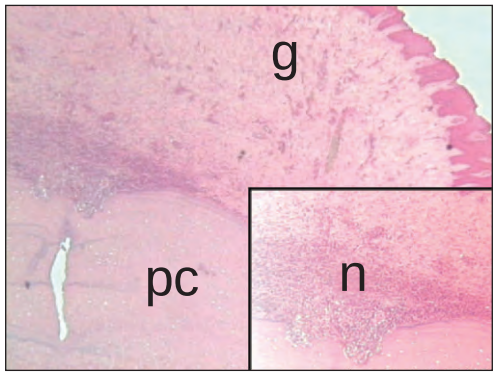


Figure 9.35. Localized periodontitis where a neutrophilic infiltration (n – inset lower right) is present within the gingiva (g) and the peripheral cementum (pc) has already receded through carious attack (H and E).

begins with a focus of gingivitis usually between adjacent teeth and proceeds to the formation of a triangular-shaped pouch that fills with food from the oral cavity. As bacteria multiply and more feed substrate is impacted into the defect, a vicious cycle is perpetuated, resulting in progressive periodontium breakdown (Fig. 9.35). When this pocket extends as far as the apical region, bacteria may directly invade the pulp chambers via apical foramen.

Primary periodontal disease is uncommon in the horse (except transiently during dental eruption); however, the prevalence of secondary periodontal disease has been reported from 10–60 per cent, with higher levels in older populations.[18,48] Apical infection caused by deep periodontal disease is uncommon in young teeth that may have a reserve crown in excess of 80 mm. In contrast, apical abscesses resulting from periodontal disease are far more common in older animals with shorter reserve crowns, with these infections frequently draining *per os* instead of fistulating through the supporting bones.

Iatrogenic dental disease

In recent years there has been increased awareness of equine dental disease and also an increase in the equipment to treat such conditions,[72] including equine adapted or specific motorized dental tools.[73–75] Pulpal insults can be caused by heat, pressure, vibration, desiccation, chemical exposure and bacterial infection.[61] Heating of the pulp can cause various histopathological changes, such as burn reactions at the periphery of the pulp, including formation of 'blisters,' protoplasm coagulation, and expansion of liquid contained in the dentinal tubules and pulp with resultant increased outward liquid flow from tubules.[76] These reactions can lead to vascular pulpar injuries with subsequent tissue necrosis.[77]

Zach and Cohen showed that pulpar temperature increases of 5.6°C following use of air turbine drills resulted in 15 per cent irreversible cellular pulp damage within the pulps of individual monkey teeth, 60 per cent for temperature elevations of 11°C, and 100 per cent for temperature elevations of 16.6°C.[78] A recent study recorded pulpar temperature changes in extracted equine cheek teeth that were being ground for 120 s, using one of three motorized dental tools, and in a second group of CT that were ground for 30 s using a fourth motorized tool.[79] Mean occlusal pulp temperature elevations of 12.7°C were noted for teeth ground for 120 s and 6.5°C for teeth ground for 30 s. These temperature increases are sufficient to cause pulpar damage.

Aside from the potential risks associated with thermal pulpar insult, aggressive dental reductions (that do not expose a pulp cavity) e.g. full 'bit-seating,' have the potential to expose sensitive dentine to the oral cavity.[27] In normal circumstances dentinee in contact with the oral cavity is sclerotic, i.e. the dentinal tubules have been occluded either by calcification of the odontoblast process, or by the tubule being filled by a 'smear' layer (ground dental dust). If this sclerotic layer is removed completely, sensitive odontoblast processes may be exposed or even removed from their dentinal tubules. This may not only be painful for the horse, but may also initiate a pulpitis. Many equine practitioners will be aware of horses that have developed anorexia or are quidding badly following major dental reductions and where pulp has not been obviously exposed. This is usually attributed without any evidence to temporomandibular joint pain, when in fact it is more likely to be due to dental pain. Such cases may benefit from antibiotic therapy to prevent bacterial pulpitis as well as non-steroidal anti-inflammatory agents.

Use of both aggressive solid carbide blades and motorized equipment allows the practitioner to remove large amounts of calcified dental material quickly and in a controlled manner. This is preferable to previously used molar shears or guillotines; however, great care should still be taken in their use while potential iatrogenic damage caused may not be readily observed at the time of the procedure, it may nonetheless have arisen at a microscopic level.

ACKNOWLEDGMENTS

The Home of Rest for Horses
Professor PM Dixon

REFERENCES

1. BEVA (1965) British Equine Veterinary Association survey of equine disease, 1962–63. *Veterinary Record,* 77, 528–538.
2. Traub-Dargatz JL, Salman MD and Voss JL (1991) Medical problems of adult horses, as ranked by equine practitioners. *Journal of the American Veterinary Medical Association,* 198, 1745–1747.
3. Uhlinger C (1987) Survey of selected dental abnormalities in 233 horses. *Proceedings of the 33rd Annual Convention of the Association of Equine Practitioners,* 33, 577–583.
4. Kertesz P (1993) In search of Mr Bishop. *Veterinary Record,* 133, 608–610.
5. Wyn-Jones G (1985) Interpreting radiographs 6: Radiology of the equine head (Part 2). *Equine Veterinary Journal,* 17, 417–425.

6. Lane JG, Gibbs C, Meynink SE and Steele FC (1987) Radiographic examination of the facial, nasal and paranasal sinus regions of the horse: I. Indications and procedures in 235 cases. *Equine Veterinary Journal,* 19, 466–473.
7. Gibbs, C (1999) Dental imaging. In: *Equine Dentistry*, eds. GJ Baker and J Easley, WB Saunders, London, pp. 139–169.
8. Dixon PM and Copeland AN (1993) The radiological appearance of mandibular cheek teeth in ponies of different ages. *Equine Veterinary Education,* 5, 317–323.
9. Barakzai SZ and Dixon PM (2003) A study of open-mouthed oblique radiographic projections for evaluating lesions of the erupted (clinical) crown. *Equine Veterinary Education,* 15, 143–148.
10. Tietje S, Becker M and Bockenhoff G (1996) Computed tomographic evaluation of head diseases in the horse: 15 cases. *Equine Veterinary Journal,* 28, 98–105.
11. Weller R *et al.* (2001) Comparison of radiography and scintigraphy in the diagnosis of dental disorders in the horse. *Equine Veterinary Journal,* 33, 49–58.
12. Tucker RL and Farrell E (2001) Computed tomography and magnetic resonance imaging of the equine head. *Veterinary Clinics in North America: Equine Practice,* 17, 131–44, vii.
13. Simhofer H (2003) Pathological findings in cheek teeth using rigid endoscopy. *Equine Veterinary Journal,* in press.
14. Dacre IT (2003) Unpublished data.
15. Muylle S, Simoens P and Lauwers H (2000) The dentinal structure of equine incisors: a light and scanning electron-microscopic study. *Cells Tissues Organs,* 167, 273–284.
16. Bender IB (2000) Pulpal pain diagnosis – a review. *Journal of Endodontics,* 26, 175–179.
17. Lynch CD and McConnell RJ (2002) The cracked tooth syndrome. *Journal of the Canadian Dental Association,* 68, 470–475.
18. Dixon PM *et al.* (2000) Equine dental disease part 4: a long-term study of 400 cases: apical infections of cheek teeth. *Equine Veterinary Journal,* 32, 182–194.
19. Ten Cate AR (1998) *Oral Histology*, ed. AR Ten Cate, Mosby, St Louis, pp. 128–149.
20. Dacre IT and Dixon PM (2002) Aetiology and pathogenesis of apical abscesses in equine cheek teeth. *Veterinary Dental Forum,* Savannah, pp. 227–231.
21. Kilic S, Dixon PM and Kempson SA. (1997) A light microscopic and ultrastructural examination of calcified dental tissues of horses: 1. The occlusal surface and enamel thickness. *Equine Veterinary Journal,* 29, 190–197.
22. Kilic S, Dixon PM and Kempson SA (1997) A light microscopic and ultrastructural examination of calcified dental tissues of horses: 2. Ultrastructural enamel findings. *Equine Veterinary Journal,* 29, 198–205.
23. Kilic S, Dixon PM and Kempson SA (1997) A light microscopic and ultrastructural examination of calcified dental tissues of horses: 3. Dentine. *Equine Veterinary Journal,* 29, 206–212.
24. Kilic S, Dixon PM and Kempson SA (1997) A light microscopic and ultrastructural examination of calcified dental tissues on horses: 4. Cement and the amelocemental junction. *Equine Veterinary Journal,* 29, 213–219.
25. Muylle S, Simoens P and Lauwers H (2001) The distribution of intratubular dentine in equine incisors: a scanning electron microscopic study. *Equine Veterinary Journal,* 33, 65–69.
26. Lowder MQ and Mueller PO (1998) Dental embryology, anatomy, development, and aging. *Veterinary Clinics in North America: Equine Practice,* 14, 227–45, v.
27. Kempson SA, Davidson M and Dacre IT (2003) The effect of three types of rasps on the occlusal surface of equine cheek teeth: a scanning electron microscopic study. *Journal of Veterinary Dentistry,* 20, 19–27.
28. Torneck CD (1998) In: *Oral Histology,* ed. AR Ten Cate, Mosby, St Louis, pp. 150–196.
29. Kirkland KD, Baker GJ, Manfra MS, Eurell JA and Losonsky JM (1996) Effects of aging on the endodontic system, reserve crown, and roots of equine mandibular cheek teeth. *American Journal of Veterinary Research,* 57, 31–38.
30. Kierdorf H and Kierdorf U (1992) A scanning electron microscopic study on the distribution of peritubular dentine in cheek teeth of Cervidae and Suidae (Mammalia, Artiodactyla). *Anatomica Embryologica (Berlin),* 186, 319–326.
31. Ten Cate AR (1998) In: *Oral Histology,* ed. AR Ten Cate, Mosby, St Louis, pp. 69–77.
32. Ten Cate AR (1998) In: *Oral Histology,* ed. AR Ten Cate, Mosby, St Louis, pp. 408–423.
33. Kilic S (1995) A light and electron microscopic study of calcified dental tissues in normal horses. Thesis, University of Edinburgh, pp. 1–193.
34. Paphangkorakit J and Osborn JW (2000) The effect of normal occlusal forces on fluid movement through human dentine in vitro. *Archives of Oral Biology,* 45, 1033–1041.
35. Dick W (1862) *Manual of Veterinary Science.* Adam and Charles Black, Edinburgh.
36. Colyer F (1931) Abnormal conditions of the teeth of animals in their relationship to similar conditions in men. The Dental Board of the United Kingdom.
37. Voss HJ (1937) *Periodontal Disease in The Horse.* Enke, Stuttgart.
38. Honma K, Yamakawa M, Yamauchi S and Hosoya S (1962) Statistical study on the occurrence of dental caries of domestic animals. *Japanese Journal of Veterinary Research,* 10, 31–37.
39. Baker GJ (1970) Some aspects of equine dental disease. *Equine Veterinary Journal,* 2, 105–110.
40. Wafa NS (1988) A study of dental disease in the horse. Thesis, National University of Ireland, Faculty of Veterinary Medicine, University College Dublin, pp. 1–205.
41. Brigham EJ and Duncanson G (2000) An equine postmortem study: 50 cases. *Equine Veterinary Education,* 12, 59–62.
42. Dixon PM *et al.* (1999) Equine dental disease part 2: a long-term study of 400 cases: disorders of development and eruption and variations in position of the cheek teeth. *Equine Veterinary Journal,* 31, 519–528.
43. Dixon PM *et al.* (1999) Equine dental disease part 1: a long-term study of 400 cases: disorders of incisor, canine and first premolar teeth. *Equine Veterinary Journal,* 31, 369–377.
44. Dixon PM *et al.* (2000) Equine dental disease. Part 3: A long-term study of 400 cases: disorders of wear, traumatic damage and idiopathic fractures, tumours and miscellaneous disorders of the cheek teeth. *Equine Veterinary Journal,* 32, 9–18.
45. Soames JV and Southam JC (1993) In: *Oral Pathology,* eds. JV Soames and JC Southam, Oxford University Press, Oxford, pp. 1–18.
46. Ramzan PH, Dixon PM, Kempson SA and Rossdale PD (2001) Dental dysplasia and oligodontia in a thoroughbred colt. *Equine Veterinary Journal,* 33, 99–104.
47. Tape MW and Tye E (1995) Ectodermal dysplasia: literature review and a case report. *Compendium of Continuing Education in Dentistry,* 16, 524–528.
48. Baker GJ (1974) Some aspects of equine dental decay. *Equine Veterinary Journal,* 6, 127–130.
49. Baker GJ (1999) In: *Equine Dentistry,* eds. GJ Baker and J Easley, WB Saunders, Philadelphia, pp. 49–59.
50. Jones SJ (1981) *Dental Anatomy and Embryology,* eds. JW Osborn, Blackwell Scientific Publications, Oxford, pp. 193–209.
51. Colyer N (1990) *Colyer's Variations and Diseases of the Teeth of Animals*. Cambridge University Press, Cambridge.
52. Gutman JL and Harrison JW (1991) *Surgical Endodontics.* Blackwell Scientific Publications, Boston.
53. Soames JV and Southam JC (1993) In: *Oral Pathology,* eds. JV Soames and JC Southam, Oxford Medical Publications, Oxford, pp. 49–55.

54. Carmalt JL (2003) Understanding the equine diastema. *Equine Veterinary Education,* 15, 34–35.
55. Becker E (1962) *Zahne.* Verlag Paul Parey, Berlin.
56. Edwards GB (1993) Retention of permanent cheek teeth in three horses. *Equine Veterinary Education,* 5, 299–302.
57. Bennett D (1992) In: *Horse Breeding and Management,* ed. JW Evans, Elsevier, Amsterdam, pp. 1–37.
58. Lane JG (1994) A review of dental disorders of the horse, their treatment and possible fresh approaches to management. *Equine Veterinary Education,* 6, 13–21.
59. Ten Cate, AR (1998) *Oral Histology,* eds. AR Ten Cate, Mosby, St Louis.
60. Dixon PM (1999) In: *Equine Dentistry*. eds. GJ Baker and J Easley, WB Saunders, London, pp. 3–28.
61. Shafer WG, Hine MK and Levy BM (1983) *A Textbook of Oral Pathology*. Saunders, Philadelphia.
62. Mueller PO and Lowder MQ (1998) Dental sepsis. *Veterinary Clinics in North America: Equine Practice,* 14, 349–63, vii.
63. Baker GJ (1979) A study of dental disease in the horse. Thesis, Glasgow University, pp. 1–96.
64. Schumacher J and Honnas CM (1993) Dental Surgery. *Veterinary Clinics in North America: Equine Practice,* 9, 133–152.
65. Soames JV and Southam JC (1993) In: *Oral Pathology*, eds. JV Soames and JC Southam, Oxford University Press, Oxford, pp. 19–33.
66. Hague BA and Honnas CM (1998) Traumatic dental disease and soft tissue injuries of the oral cavity. *Veterinary Clinics in North America: Equine Practice,* 14, 333–347.
67. Ellis RG and Davey KW (1970) *The Classification and Treatment of Injuries to the Teeth of Children.* Year Book Publishers, Chicago.
68. Dacre IT and Dixon PM (2002) The histopathology of cheek teeth fractures. *Veterinary Dental Forum Proceedings*, 16, 232–237.
69. Soames JV and Southam JC (1993) In: *Oral Pathology,* eds. JV Soames and JC Southam, Oxford University Press, Oxford, pp. 91–111.
70. Crabill MR and Schumacher J (1998) Pathophysiology of acquired dental disease of the horse. *Veterinary Clinics of North America: Equine Practice*, 14(2), 291–307.
71. National Institute of Dental and Craniofacial Research (2003). Periodontal diseases: microbial and host genomics/proteomics. Internet communication, 24 April.
72. Scrutchfield WL and Schumacher J (1993) Examination of the oral cavity and routine dental care. *Veterinary Clinics of North America: Equine Practice*, 9, 123–131.
73. Scrutchfield WL (1999) In: *Equine Dentistry*, eds. GJ Baker and J Easley, WB Saunders, London, pp. 173–184.
74. Dacre KJ, Dacre IT and Dixon PM (2002) Motorised equine dental equipment. *Equine Veterinary Education,* 14, 263–266.
75. Baker GJ and Allen ML (2002) The use of power equipment in equine dentistry. *AAEP Proceedings,* 48, 438–441.
76. Castelnuovo J and Tjan AH (1997) Temperature rise in pulpal chamber during fabrication of provisional resinous crowns. *Journal of Prosthetic Dentistry,* 78, 441–446.
77. Raab WH (1992) Temperature related changes in pulpal microcirculation. *Proceedings of the Finnish Dental Society,* 88 (Suppl 1), 469–479.
78. Zach L and Cohen C (1965) Pulp response to externally applied heat. *Oral Surgery Oral Medicine Oral Patholoogy* 19, 515–530.
79. Dacre IT, Uttley L and Dixon PM (2003) Thermal Changes in Equine Dental Pulp Associated with Mechanical Grinding. In press.

10 Abnormalities of Wear and Periodontal Disease

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Introduction

In reviewing the chewing cycle of the horse, it was shown that the structure and function of the teeth, support structures, bones, joints and muscles produce a coordinated, efficient 'machine' that initiates the digestive process. It was also suggested that the efficiency of this process could be changed by minor alterations in occlusal contact pattern. In this chapter the recognition of occlusal surface abnormalities, the induced pathological changes and their clinical effects and management will be described.

Definitions

A range of descriptive and architectural terms are used to describe occlusal surface irregularities that develop in horses' teeth as a sequel to abnormalities of wear. It is clear that any irregularities that develop will progress, i.e. become worse, as the teeth continue to erupt.

Incisor teeth irregularities result in changes of the 'bite plane' – the normal horizontal plane that is made by contact of the upper and lower incisors. In parrot-mouthed and monkey-mouthed horses (overjet/overbite and underjet/underbite) there may be no incisor contact and so the bite plane, in its definitive sense, does not exist. A subject that has arisen in recent discussions on dental prophylactic techniques in the horse has been the exact relationship, from an etiological point of view, between incisor and cheek teeth contact. It has been suggested that as horses age and the frontal plane of incisor contact becomes more horizontal, there arises a condition in which the incisors may 'lock up' and thereby change cheek teeth occlusal contact patterns. More data and studies are required to evaluate this suggested hypothesis, i.e. what is the chicken and what is the egg? Contact irregularities will be discussed further in this chapter.

When viewed from the side, the cheek teeth arcades conform to a series of interlocking, regular irregularities. These surfaces afford a completely efficient machine to chew food. It must be noted that the cheek teeth erupt in a series of arcs – they can be seen radiating in a series from the temporomandibular joint. In general as horses age, and to some extent depending upon the nature of the feeds consumed, these occlusal surfaces ('blades') are maintained throughout life. It is normal for the edges to be slightly reduced, i.e. the surfaces get a little smoother with age. There are some breed differences that are seen in the plane of the cheek teeth arcades. Smaller breeds of horses, ponies and miniature horses have cheek teeth arcades that are not truly horizontal and this slight dishing, i.e. more crown rostrally and caudally, becomes more obvious with age.

In a study of age-related morphology of equine mandibular cheek teeth,[1] it was shown that even though erupted crown is lost constantly as a result of masticatory wear, an overall increase in tooth length occurred during the first year following eruption, with no change in length from 1–2 years following eruption (Fig. 10.1). This apparent discrepancy is a result of the fact that the rate of increase in root length during the first 2 years following tooth eruption exceeds the rate of loss of erupted crown (Fig. 10.2). This resulted in an overall increase in the first year and lack of change in the second year in overall tooth length, even though occlusal loss due to wear was occurring. Three years following eruption the loss of erupted crown, due to wear, exceeded root length formation and a net loss of overall tooth length occurred (Fig. 10.3).

While the crown and reserve crown length decreased each year from 0–8 years following eruption, the rate of tooth loss was not constant. The rate of crown loss increased each year from 0–5 years and then decreased from 5–7 years following eruption. Similarly, the rate of root formation was highest from the time of eruption until 5 years after eruption and then decreased from 5–7 years of tooth age (Fig. 10.2). These results suggest that equine cheek teeth undergo an increased rate of attrition, an increased rate of eruption or both during the period of rapid root formation in the first 5 years following eruption. The results of this investigation indicated that crown loss due to masticatory wear was not constant, as has previously been reported, but was variable. This variable rate of crown loss may be influenced by the rate of root formation, the rate of eruption of reserve crown, and a variance in tooth hardness or the amount of chewing.

The increased rate of control of coronal loss in cheek teeth during the first 5 years post eruption may contribute to the rapid reformation of enamel points following removal. Due to the staggered eruption times of the cheek teeth of the horse, individual horses from 1– 9 years of age possess cheek

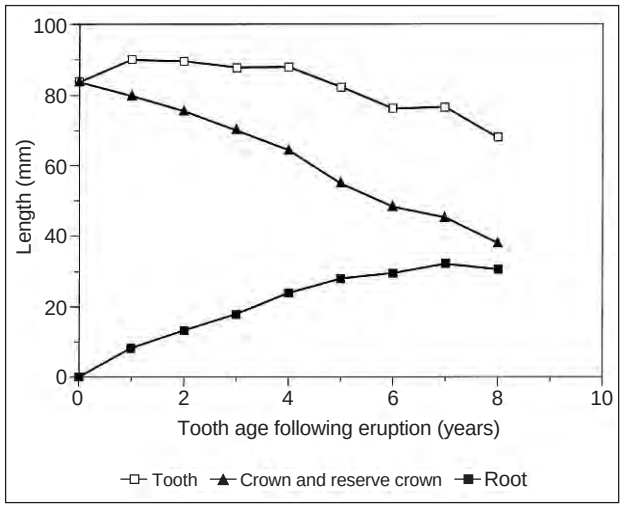


Figure 10.1. Mean tooth, crown and reserve crown, and root length (from Kirkland 1994,[1] with permission).

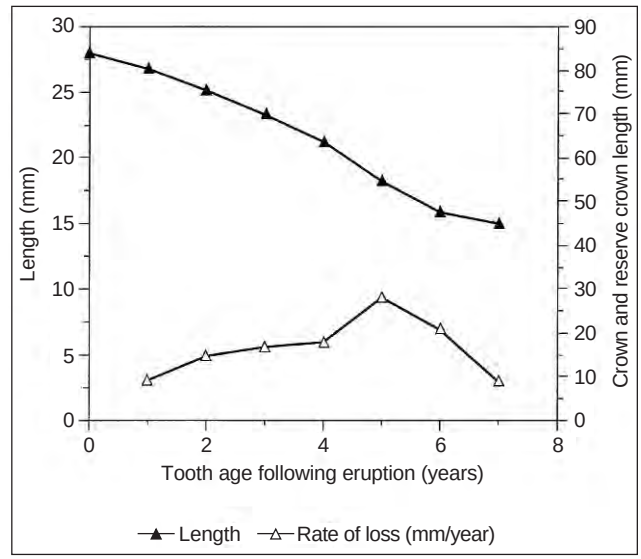


Figure 10.3. Crown length and rate of crown loss (from Kirkland,[1] with permission).

teeth that are, in the first 5 years, relatively longer following eruption. Horses under the age of 9 years may, therefore, require more frequent oral examinations and dental flotations to remove enamel points.

Mean occlusal length decreased at a constant rate in cheek teeth from 0–8 years of age (Fig. 10.4). This trend corresponds to the shape of equine cheek teeth, which taper in circumference from the crown to the tooth end. As occlusal crown is lost due to wear, the tooth erupts a narrower reserve crown. This decrease in crown length over time shortens the mesial-to-distal length of the cheek teeth arcade and results in a shortened masticatory surface. This decreased surface area may be an important factor to consider in geriatric patients suffering weight loss and/or poor thrift. The eruption of intermittently narrower cheek tooth crowns may also be a factor in the increased incidence of periodontal disease in older horses.[2] As teeth of variable mesial to distal crown lengths make up the cheek tooth arcade of older horses, the integrity of the once tightly packed battery of teeth may be lost. The interproximal spaces (spaces between adjacent teeth) increase in size and may allow the areas to trap food and predispose the cheek teeth to gingivitis and periodontal disease. It has been suggested that there is caudal- to rostral-compression of the cheek teeth during the eruption process and that these pressures result in rotation of some teeth, particularly 309 and 409. In this way the continuous interproximal contact of the rows of mandibular cheek teeth may be lost and diastemata are formed. In such cases feed material becomes trapped into these spaces and, in time, instigates periodontal inflammatory disease. Management should be based on attempts to restore normal occlusal

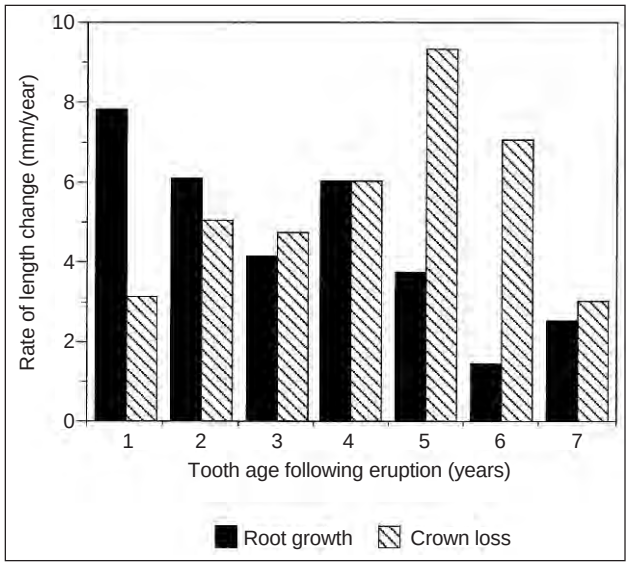


Figure 10.2. Rate of root formation and crown loss (from Kirkland,[1] with permission).

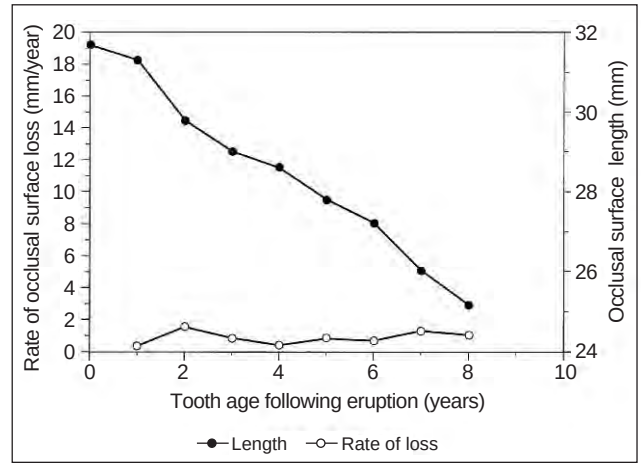


Figure 10.4. Occlusal length (308 mesial to distal) and rate of reduction post eruption (from Kirkland,[1] with permission).

contacts. Dental overgrowths should be removed, rotated teeth may need to be extracted and oral hygiene plans of irrigation and antibiotic therapy initiated.

Specific irregularities that are frequently seen include:

- Rostral hooks (106 and 206) and caudal hooks (311 and 411) – these beak-like occlusal overgrowths develop as a result of incomplete occlusal contact (Fig. 10.5).[3] Hooks 106 and 206 may cause oral pain, bit pressure points and buccal surface calluses or ulcers. They are easily diagnosed and corrected by grinding or floating.
- Enamel points (enamel edges) – the folding of the enamel organ and subsequent secretion of enamel in the unique pattern of the maxillary and mandibular teeth results in the appearance of enamel points on the buccal edges of the maxillary (Fig. 10.6) and the lingual edges of the mandibular cheek teeth. Incomplete full occlusal contact tends to exacerbate these points and, in turn, contact with the oral mucosa may lead to ulcers and callus formation (Fig. 10.7). Such areas may be sensitive to cheek pressure on palpation and on mouth opening and may subsequently lead to further abnormalities in chewing patterns and hence a compounding of the tooth surface irregularities.
- Step mouth – absent teeth lead to mesial and distal movement of adjacent teeth and consequently changes in the conformation of the occlusal surfaces. Such overgrowth and points may result in bizarre occlusal patterns (Fig. 10.8). Large hooks on 311 and 411 may cause penetration of the palatine mucosa, ulcer formation and in rare cases, laceration of the palatine artery. If the occlusal defects persist, there may be rotation of adjacent or opposing teeth and an arcade alignment that will defy therapy short of exodontia.
- Wave mouth – this is the term that is used to describe a series of convex and concave changes of the teeth crowns and occlusal surfaces, usually with reciprocal concave and convex changes on the opposing arcade that may occur over time. It results in an inefficient grinding surface with a tendency for the slopes to become rather smooth.



Figure 10.6. Enamel points, buccal margins of left maxillary arcade, rostral hook 206.



Figure 10.5. Rostral hook 106.



Figure 10.7. Buccal trauma resulting from presence of enamel points.



Figure 10.8. Irregular dental arcades following loss of 411.

The severity, and perhaps irregularity, of the waves may be influenced by concomitant dental pathology (maleruptions, impactions or missing teeth) and also by the shape and size of the horse's head. Those animals that have a marked caudal slope along their mandibular arcades as a result of head shape will exacerbate the waves (e.g. ponies). Another contributing factor to wave formation is the presence of enamel lake cemental hypoplasia on 109 and 209 and from this, a tendency for the forces of attrition to cause these teeth to cup out.

- Ramps – this is the presence of more crown in a rising 'ramp' on 309–311 and 409–411. Both waves and ramps require careful evaluation and appropriate floating and grinding to correct the defects. In cases of abnormal wear, it may not be possible to decide the precise process that has resulted in some teeth becoming completely worn away and the opposing tooth overgrown. Does it indicate that there are changes in density or hardness of odd teeth so that a particularly 'soft' tooth is worn away while its opposing number super erupts? (Fig. 10.9.) This is unlikely but it does show that the pathophysiology of chewing is complex and that irregularities of wear have a multifactorial etiopathogenesis.
- Shear mouth – this is an extreme form of cheek tooth malocclusion with reduction of the lingual surfaces of the maxillary teeth and buccal surfaces of the mandibular teeth. There is complementary overgrowth of the buccal surfaces of the maxillary teeth and lingual surfaces of the mandibular teeth. The result is an extreme malalignment of the cheek teeth surfaces and extremely sharp and ineffective edges. This condition results in profound chewing dysfunction, loss of body condition and may lead to death. Management is not easy and requires multiple realignment and tooth reduction procedures as well as special diets. In very old horses extreme crown attrition may render the horse essentially toothless (Fig. 10.10). In such cases body condition may still be maintained with the use of custom designed feeds and dietary supplements.

It is frequently noted that there is very little accumulation of dental plaque and dental calculus on normal teeth of horses.[2,3] The exception to this observation is the presence of both plaque and calculus on the canine teeth. It is concluded that the normal frictional forces of chewing are extremely effective in keeping the surrounding tissues of the teeth of the horse healthy. Invariably, abnormal occlusal contact results in the formation of dental plaque and calculus. In a long-term study of 400 cases of equine dental disease, there were 44 cases of gross abnormalities of wear of the cheek teeth. These included shear mouths, wave mouths and step mouths. In all except four cases there were multiple disorders, but prominent overgrowths were more common on the lingual aspect of the mandibular cheek teeth than on the buccal aspect of the maxillary cheek teeth.[4] In these cases all loose teeth and overgrown teeth were removed; all other cases were subjected to tooth grinding to restore the normal integrity of the occlusal surfaces.[4,5]



Figure 10.9. Gross irregularities of wear and associated periodontal pathology.

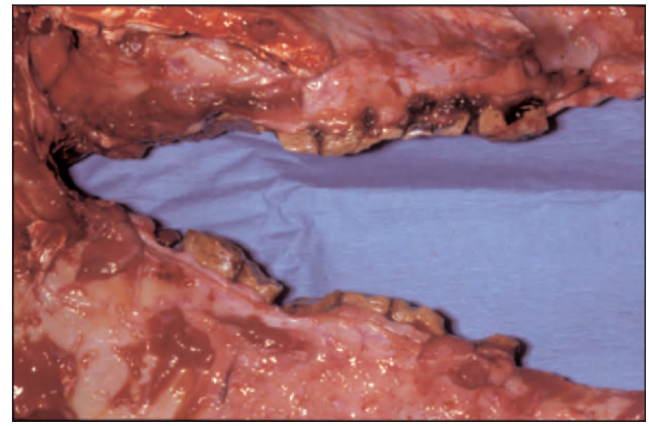


Figure 10.10. Extreme cheek teeth wear in an aged pony mare.

Diastema formation

Diastemata are detectable interdental (interproximal) spaces between the cheek teeth (CT). It has been proposed that in most cases of CT diastemata, insufficient caudal angulation of the erupted crown of the first CT and/or insufficient rostral angulation of the erupted crowns of the fifth and sixth CT are present. This results in insufficient compression of the occlusal surfaces of the sixth CT and allows interdental spaces to open.[6] There is also an occlusal to root apex narrowing or tapering of the CT so that there may be a tendency for interproximal tooth contact to be lost in older horses (Figs 10.11–10.14).

Classical descriptions of periodontal disease in man and in many animals have referred to the problem as a 'silent disease' in that it is symptom free. It has recently been suggested that diastema formation and associated periodontal disease in those cases in horses is, in fact, a painful oral condition.[6] Treatments have been aimed at improving occlusal contact by removing overgrown crowns and extracting grossly displaced or rotated teeth, and modification of the diet, using a soaked complete concentrate feed is also helpful.

Periodontal disease

Periodontal disease is the presence of disease and loss of tissue in those structures that surround the tooth or teeth. Periodontium means around the tooth and in that sense is confined to the bony socket. In clinical practice the periodontium includes the alveolus (bony socket) cement, periodontal ligament and the gingivae. Diseases of the periodontal structures have been known since antiquity. Skulls of some ancient cave dwellers show evidence of chronic periodontal disease. An acute form, now known in man as acute necrotizing gingivitis or Vincent's infection, was reported at least as early as 400 BC in soldiers of the Greek army of Xenophon.[7]



Figure 10.11. Mandible showing lateral displacement of 310 and 409 (from Dixon *et al.*,[6] with permission, *Equine Veterinary Journal*).

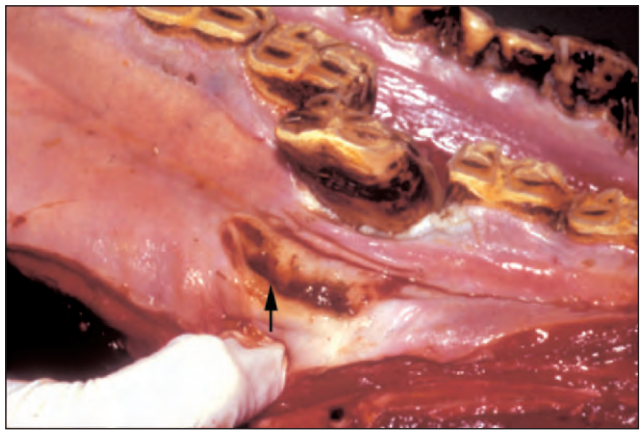


Figure 10.12. 310 from Fig. 10.11; there is extensive laceration of the adjacent buccal mucosa (arrowhead) (from Dixon *et al.*,[6] with permission, *Equine Veterinary Journal*).

Clarification of various periodontal diseases is difficult as nearly every case begins as a minor localized disturbance which, unless adequately treated, gradually worsens until the alveolar bone is resorbed and the tooth is exfoliated. This means that a variety of etiological stimuli may produce similar end-stage pathology and so the true etiopathogenesis may not be defined. In human medicine the descriptive pathological reports of John Hunter were the basis of all subsequent analyses of periodontal disease.[8]

The term parodontal disease was used in human dental pathology[8] and introduced into veterinary literature to describe gingival and dental disease processes in dogs and horses in 1939 and continued in 1948 and 1960.[9–11] It was subsequently concluded, after reviewing the literature and referring to the classifications of periodontal disease in man that the generic term periodontal disease is the most suitable term to use in veterinary dental pathology and clinics.[12]

Pathology of the periodontium may be grouped in four categories (American Academy of Periodontology, 1957):

1. inflammation (gingivitis, periodontitis)
2. dystrophy (gingivosis, periodontosis)
3. neoplastic
4. anomalies.

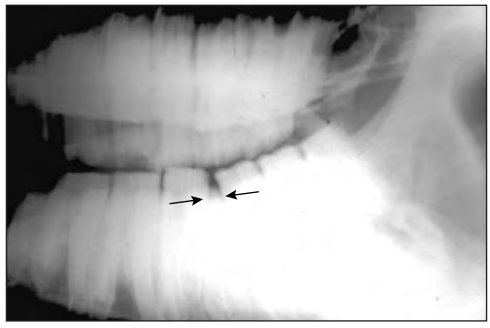


Figure 10.13. Latero-oblique radiograph obtained with the horse's mouth open demonstrating diastema (arrows) (from Dixon *et al.*,[6] with permission, *Equine Veterinary Journal*).

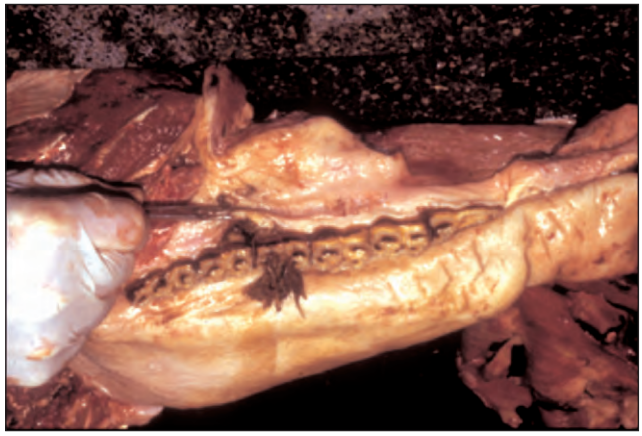


Figure 10.14. Extensive periodontal food pocketing in mandibular CT diastema. Major buccal periodontal pocketing (from Dixon *et al.*,[6] with permission, *Equine Veterinary Journal*).

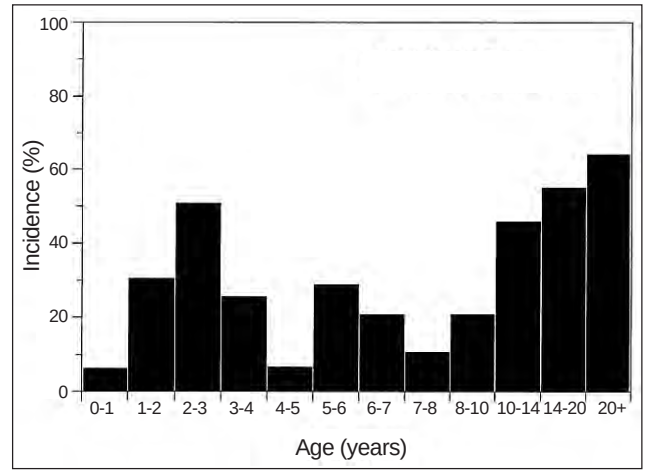


Figure 10.15. Prevalence of periodontal disease in 416 skulls related to age.

All four categories are known to occur in the horse, but most information is available for category 1, inflammatory periodontal disease. Periodontal inflammation has been recognized for years as being important in the horse. It was suggested that 'quidding' was a pathognomatic sign of periodontal disease or alveolar periostitis.[13] In those observations, it was noted that the lesions start primarily in the interproximal areas of the teeth and the caudal mandibular spaces were most affected. In an examination of 50 equine skulls in the 1930s, 30 per cent were found to be affected with periodontal disease.[8] In that study it was concluded that the disease was initiated by gingival trauma caused by the feeding of coarse chaff. Repeated observations in the 1970s and 1990s in the UK and USA have shown similar high prevalence levels of the disease.[3,12] It was found that in an examination of the teeth and gums of 218 and 446 skulls, the incidence of periodontal disease changed with age. There was a 40 per cent prevalence in horses 3–5 years of age; this fell in horses 5–10 years of age, and then increased to 60 per cent in horses over the age of 15 years (Fig. 10.15).

Work in other species, dogs in particular, has shown that dental work is necessary for the maintenance of the health of the gingival mucosa. Studies in ferrets[14] and dogs[15,16] concluded that the frictional forces associated with chewing hard substances were sufficient to keep the teeth free of dental scale and the gums in a healthy state.

The physiological process of eruption of the permanent dentition is responsible for the frequency of periodontal disease seen in younger horses. It suggests that at least some levels of periodontal pathology will resolve once normal occlusion is achieved by the permanent dentition, thereby reinforcing the concept that the detection and correction of abnormalities of wear is a key factor in the prevention of periodontal disease.

In man at least there is an increasing body of evidence linking the presence of periodontal disease to numerous other serious diseases. In the UK it was found that there was an association between poor oral health and overall mortality in that there was an increase in risk (mortality) of 2.6 per cent in patients with poor oral health (periodontal disease) when compared with those with good oral health.[17] Consequently, it is common in periodontic practice in man to supplement antibiotic coverage as a prophylaxis against cerebral and myocardial infarctions during periodontal treatment and periodontal surgery.[18]

Numerous studies have attempted to show similar correlations in other animals without definitive proof. One study involving blood cultures in horses undergoing tooth extractions and periodontal curettage failed to prove that periodontal surgery resulted in bacteremias.[3]

Periodontal anatomy and function

Each tooth is independently and firmly attached to the bony structure (alveolus) within the bones of the head. The teeth are attached by bundles of connective tissue fibers referred to as the periodontal membrane or ligament (Fig. 10.16A,B).

The arrangement of fibers in the periodontal ligament is complex. Dense bundles of collagen run in various directions from the bone of the socket wall to the cement covering the reserve crowns and the tooth roots. The embedded portions of the collagen bundles are referred to as Sharpey's fibers. In general, the fibers are arranged in such a fashion that occlusal forces and pressures, are translated into longitudinal forces on the fibers to provide resistance. The periodontal ligament contains blood vessels and nerves. The arrangement of the collagen fibers protects the vessels from occlusal pressure that might result in ischemia. In this way the tooth is suspended firmly within the alveolus and at the same time it is permitted some slight movement.

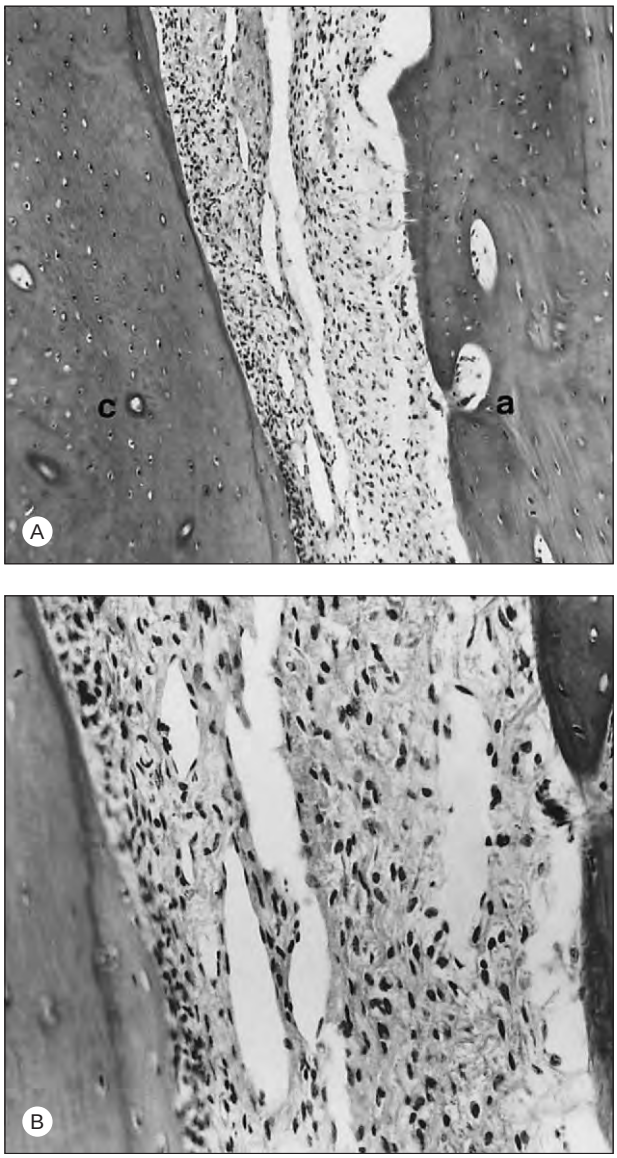


Figure 10.16. (A) Periodontal ligament: c = cementum, a = alveolar bone, decalcified H and E × 110. (B) Periodontal ligament, H and E × 250.

Clinical signs of periodontal disease in the horse

Gingival hyperemia, edema, ulceration, deepening periodontal pockets and packing of feed material into these spaces are the classic pictures of periodontal disease (Figs 10.17–10.19). It is necessary, of course, to carry out a thorough examination of the mouth to detect such conditions (see Chapter 13). There will be both supra- and subgingival plaque and calculus deposits associated with these lesions. Periodontal disease in the horse has been divided into four categories[3] based on evaluation of the severity of the lesion:

1. local gingivitis with hyperemia and edema
2. erosion of gingival margin 5 mm, periodontal pocket
3. periodontitis with loss of gum
4. gross periodontal pocketing, lysis of alveolar bone, loosening of bone support (Fig. 10.20).

Horses with grades 1 and 2 periodontal disease may not show overt signs of oral discomfort. The careful owner may notice some excess salivation and a sensitivity to cold water. Halitosis is the pathognomic sign for severe periodontal disease in the horse and for this reason the use of disposable gloves is recommended when examining the oral cavity of the horse.

Etiology of periodontal disease

The role of normal dental work in maintaining the health of periodontal structures has been documented in most mammals. It therefore is not surprising that abnormalities of wear associated with tooth eruptions in young horses and arcade irregularities in older horses are the most common initiating factors in the pathogenesis of periodontal disease in the horse.

Other factors influence the development and progression of periodontal disease and it is commonly described as being a multifactorial infection.[16] Some of these factors include plaque, oral microflora and calculus, as well as age, general

The gingiva has a mucous membrane surface with a dense internal fibrous attachment to the periosteum. Extending from the epithelial attachment of the gum to the crown of the tooth is a free margin of gum enclosing the gingival margin of the gum, the gingival sulcus (crevice). The interdental gingival tissue is referred to as the col. The col is covered with a non-keratinized epithelium. The free gingiva 'sticks' to the crown of the tooth by surface tension. It is customary to use the terms sub- and supragingival to refer to the locations below and above the gum line. If and when the gingival sulcus deepens in periodontal disease, it is referred to as a periodontal pocket (perio pocket). In examining and charting oral and dental disease, it is common practice to measure the depths of these pockets in millimeters.

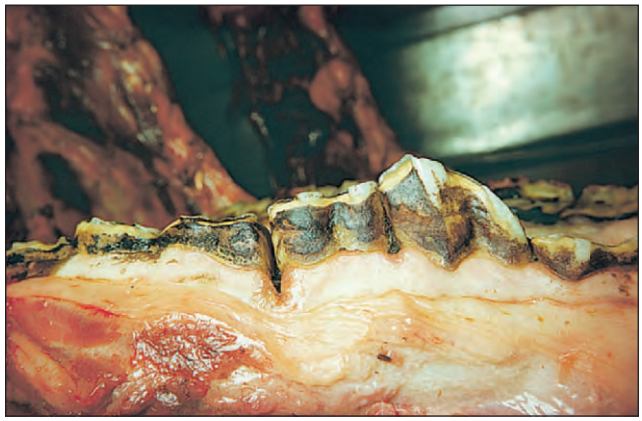


Figure 10.17. Gingival hyperemia and edema.

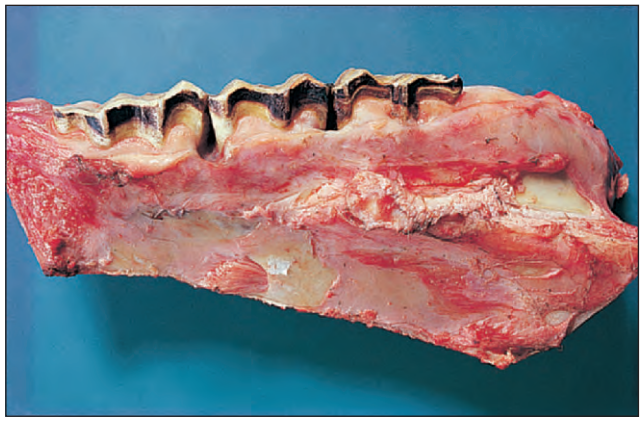


Figure 10.18. Periodontal pocketing.



Figure 10.20. Grade 4 periodontitis with loss of alveolar bone – radiological appearance.

health, chewing patterns, breed, immune status and local irritants (e.g. grass awns).

Plaque is an organic matrix made up of salivary glycoproteins and contains oral bacteria as well as inorganic material derived from feed materials. Bacterial fermentation within this layer releases free radicals and material that results in injury to the gingiva and within the gingival sulcus. Aerobic bacteria are more prevalent in supragingival plaque and facultative anaerobes live in the sulcus. Bacterial numbers and types change in the presence of initial gingival inflammation and a 'domino' cycle is initiated:

inflammation → plaque build up → hyperemia
↑ ↓
loss of tooth
↑
bone loss ← loss of support tissue ← edema

In advanced disease, there is significant loss of alveolar bone. Periodontal inflammation may result in attempted repair with the production of excess cementum over the surface of the reserve crown – in some cases this may progress to a form of hypercementosis and the production of nodules of cementum.



Figure 10.19. Gross pocketing with food impactions.

In advanced cases of periodontal disease, with tooth loosening or rotation, extraction of the affected teeth may be the only course of action. In human and canine dentistry newer techniques of irrigation of the periodontal pockets, resection of gingival tissue and the implantation of perioceutics within the inflamed gingival pockets have been used with some success. A technique has been developed for use in the horse that produces a water and medical-grade baking-soda slush that removes feed material and debris from interproximal spaces and periodontal pockets. This procedure can be supplemented by the use of dental impression material to create a barrier over an implanted pharmaceutical (Doxyrobe Gel, Pharmacia and UpJohn, 7000 Portage Road, Kalamazoo, MI).[19]

Treatment

The horse is no exception to the general rule of periodontologists that states that prevention is better than treatment. Once gum recession and loss has occurred, it is not possible to undertake a treatment regime that will result in reattachment of gum and a reduction of gingival pocket size in the horse. Consequently, the equine clinician's role is to eliminate irregularities of wear, oral ulcers and other conditions that may initiate the progressive process of periodontal pathology (see Chapter 16).

In horses with major irregularities of wear and advanced periodontal pocketing, treatment is aimed at restoring, as closely as possible, normal or near normal occlusion. Loose teeth should be extracted, periodontal pockets irrigated and opened where possible, i.e. enlarged so as to discourage food impactions in these areas.

Summary

Irregularities of wear and periodontal disease have been described as the scourge of the horse in the era of horse transport and farming.[8] These observations of the early

nineteenth century have been re-examined and it is clear that although there has been an improvement in the general health of the horse in the past 60 years, irregularities of wear and periodontal disease are still extremely common.

REFERENCES

1. Kirkland KD (1994) The morphology of the endodontic system reserve crown and roots of equine mandibular cheek teeth. MS Thesis, University of Illinois, pp. 24–102.
2. Baker GJ (1979) A study of dental disease in the horse. PhD Thesis, University of Glasgow, pp. 42–56.
3. Becker E (1962) Dental disease in the horse. In: *Handbook of Special Pathology of Domestic Animals,* ed. E Joest, Paul Parey, Berlin, pp. 642–651.
4. Dixon PM, Tremaine WH, Pickles K *et al.* (2000) Equine dental disease Part 3: a long term study of 400 cases: disorders of wear, traumatic damage and idiopathic fractures, tumours and miscellaneous disorders of the cheek teeth. *Equine Veterinary Journal,* 32, 9–18.
5. Dixon PM (2000) Removal of equine dental overgrowths. *Equine Veterinary Education,* 12, 68–81.
6. Dixon PM, Tremaine WH, Pickles K *et al.* (1999) Equine dental disease Part 2: disorders of development and eruption and variations in position of the cheek teeth. *Equine Veterinary Journal,* 31, 519–528.
7. Shafer WG, Hine MK and Levy BM (1963) *A Textbook of Oral Pathology* (2nd edn). WB Saunders, Philadelphia.
8. Colyer F (1931) *Abnormal Conditions of the Teeth of Animals in Their Relationship to Similar Conditions in Man.* The Dental Board of the United Kingdom, London, pp. 38–43.
9. Wright JG (1939) Some observations on dental disease in the dog. *Veterinary Record,* 51, 409–421.
10. Shuttleworth AC (1948) Dental disease of horses. *Veterinary Record,* 60, 563–567.
11. Hofmeyer CFB (1960) Comparative dental pathology (with particular reference to caries and parodontal disease in the horse and dog). *Journal of the South African Veterinary Medical Association,* 29, 417–480.
12. Baker GJ (1970) Some aspects of dental disease. *Equine Veterinary Journal,* 2, 105–110.
13. Little WM (1913) Periodontal disease in the horse. *Journal of Comparative Pathology and Therapeutics,* 24, 240–249.
14. King JD (1947) Experimental investigations of parodontal disease. *British Dental Journal,* 82, 61–69.
15. Brown MG and Park JF (1968) Control of dental calculus in experimental beagles. *Laboratory Animal Care,* 18, 527–535.
16. Lindhe J, Hamp SE and Loe H (1973) Experimental periodontitis in the beagle dog. *Journal of Periodontal Research,* 3, 1.
17. DeStefano F, Andra RF, Kahn HS *et al.* (1993) Dental disease and risk of coronary heart disease and mortality. *British Medicine,* 306, 688.
18. Wiggs RB and Lobprise HB (1997) Periodontology. In: *Veterinary Dentistry Principles and Practice,* eds. RB Wiggs and HB Lobprise, Lippincott-Raven, Philadelphia, pp. 186–231.
19. Greene SK and Basile TP (2002) Recognition and treatment of equine periodontal disease. *Proceedings AAEP,* 48, 463–466.

11 Dental Decay and Endodontic Disease

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Introduction

In many species and particularly in man, dental caries is the most common cause of dental decay. Caries is a disease of the calcified tissues of the teeth characterized by a demineralization of the inorganic, and a destruction of the organic, substance of the tooth. In ancient times, dental caries was associated with the idea of worms in or around the teeth. It was believed that the gastric juice from a pig would expel worms – as large as earthworms – from a decayed tooth. Gallen believed that dental caries was produced by an abnormal condition of the blood that affected the internal structure of the tooth.[1]

Dental caries does occur in the horse, but the term has been subjected to various interpretations based on etiopathogenesis.[2–10] In this chapter dental decay, as it occurs in the horse, will be reviewed and the pathophysiology of endodontic disease (e.g. pulpitis) illustrated and analyzed.

Dental caries

Working in Koch's laboratory in 1889, Miller found that organisms from carious dentin produced lactic acid when cultured with starch or with sugar.[11] From these observations, he formulated the 'chemico-parasitic' theory of dental caries: 'Dental decay is a chemico-parasitic process consisting of two stages, the decalcification of enamel, which results in its total destruction, and the decalcification of dentin as a preliminary stage, followed by dissolution of the softened residue. The acid which affects this primary decalcification is derived from the fermentation of starches and sugar lodged in the retaining centers of the teeth.'

In the past 100 years, many, many studies have detailed this process, giving us the role of specific bacteria, the microscopic, transmission and electron microscopic appearance and the prophylaxis of dental caries in man.[12]

The bulk of scientific evidence does implicate carbohydrates, oral micro-organisms and acids in dental caries. A proven relationship was made in human caries between prevalence and the acid-producing bacterium *Lactobacillus acidophilus*.[13] Under laboratory conditions, dental caries in hamsters and rats was considered to be an infectious and transmissible disease. Pure cultures of streptococci isolated from hamster caries would induce the typical picture of active caries in other hamsters.[14] The transmissible and infectious nature was confirmed in gnotobiotic rats. In a study in which gnotobiotic rats were fed on a coarse-particle, high-sugar diet which would produce dental caries in normal animals, the subjects (gnotobiotes) did not develop caries. However, a single strain of oral streptococcus isolated from a control rat on the same diet and introduced into the gnotobiotic animals resulted in caries development.[15]

Cariogenic acid is produced beneath the macrobacterial layer which forms on the surface of the tooth, plaque. There is a significant difference in the pH of plaque in carious teeth and non-carious teeth (5.5 and 7.1 respectively).[16] It has been proposed that there may be two forms of caries pathogenesis. In one type, micro-organisms invade the enamel lamellae, attach to the enamel and involve the dentin before there is clinical evidence of caries. In the other, bacteria in the dental plaque form acids which decalcify the enamel prior to invasion by micro-organisms.

In apparent conflict with acid production and lowering of pH, it was also noted that by proteolysis-chelation, demineralization of enamel could occur at a neutral or even alkaline pH.[17] This process is brought about by an initial breakdown of the protein and other components of the enamel, chiefly keratin, by keratinolytic micro-organisms. In this way, soluble chelates are formed with the mineralized component of the tooth and decalcification results. It can, therefore, be concluded that it is possible for the initial attack to be on both the organic and inorganic portions of the tooth simultaneously.

Horse caries (dissolution of the calcified tissues of the teeth) occurs under a number of circumstances. In some environments, and under some feeding programs, foods that are cariogenic (i.e. caries forming) may adhere to the tooth crowns. The use of sweet foods (e.g. molasses or the by-products of sweet-potato-processing plants) produces a cariogenic diet. Consequently there is surface erosion particularly of the incisor teeth labial surfaces – that is directly caused by bacterial fermentation and acid production (pH below 7.1) within the adherent dental plaque. The surfaces become mottled and may be discolored with exposed dentin (Fig. 11.1).

The process of cement formation – both peripheral and within the incisor and maxillary cheek teeth infundibula – has been discussed in Chapter 3. It has been frequently



Figure 11.1. Carious lesions of deciduous incisors.

misinterpreted that infundibular cemental hypoplasia is in fact caries. Histopathological examination, however, reveals that this is not the case. It has been suggested that 'an open infundibulum' is the cause of the dental decay.[18] Clearly this concept is at fault in that all maxillary and incisor teeth have open infundibula – the channel that represents the vascular pathway to cementogenesis. The belief that cemental hypoplasia is of major pathological significance has even encouraged people to 'fill' these defects.[19] Despite the confusion over the incidence and significance of maxillary cheek teeth cemental hypoplasia, there is no doubt that the presence of feed material impacted into areas of hypoplastic cement does create the potential for bacterial fermentation, acid production and caries of cement to be initiated. No data are available as to the prevalence of caries of cement as distinct from cemental hypoplasia in the horse. Perhaps the interesting question is why we do not see a greater incidence of carious pathology associated with cemental hypoplasia in the horse. It has been suggested that although conditions exist for acid production at this location, it may not occur because of the hostile microclimate that exists in this region during life.[5] More research work needs to be directed toward this problem.

The complexities of pathologic bacterial fermentation within the periodontal structures of horses with periodontal disease may result in dental caries. This analysis leads to the concept of four forms of dental caries in the horse:

1. caries of cement from the occlusal surface
2. caries of peripheral cement
3. caries of root cement originating from purulent periodontitis
4. caries from an open pulp cavity.[5]

It is also clear that conditions that interfere with the normal 'cleansing' action of the frictional forces of the chewing cycle will create tooth surface conditions that predispose to carious demineralization. It is, therefore, not surprising that there is a complex and interdependent relationship between dental and oral pathology in the horse, for example, malformation, maleruption, fracture, irregularities of wear, plaque deposition, periodontal disease and dental decay.

Endodontic disease

The pulp cavities of teeth have a multicellular morphology and a vital role in the formation of the teeth and the maintenance of the health of the teeth. Dental pulp is seen to develop as a condensation of connective tissues under the inner enamel epithelium at the bell stage of tooth embryology (dental papilla). At an extremely early stage, odontoblasts are differentiated from cells in the pulp and they, in turn, begin to secrete a collagenous matrix. This matrix is known as predentin or uncalcified dentin or dentinoid, and represents the beginning of hard tissue formation of the tooth.[20] Tissues within the dental papilla continue to elaborate and the definitive structure of the dental pulp is established. The dental pulp contains odontoblasts, collagen and elastic fibers, and as they form, nerve tissue and vascular channels. Fibroblasts, histiocytes, mast cells, polymorphonuclear leukocytes, lymphocytes, plasma cells and eosinophils are also found in dental pulp – the majority of these latter cells are present under conditions of inflammation. It appears that a low-grade inflammatory response is a normal histologic feature of dental pulp and reflects the physiologic response of dental tissues.

The pulp chambers change in size and shape with age and also in response to crown attrition and tooth root formation.[21] In the incisors and canines, the pulp chamber is a simple cone with a single root apex. Mandibular and maxillary teeth have more complex pulp chamber shapes. Mandibular teeth have, initially, a common pulp chamber with five coronal extensions and two large root openings. As the horse ages there is, eventually, separation of the two root systems, but it is not yet defined how consistent these separations are related to age. One study suggests that, for at least 3 years post eruption, there is still a connection between the pulp chamber as related to the mesial and distal roots.[21] Transverse sections of the reserve crowns of the maxillary cheek teeth also show five coronal extensions of the pulp chamber. When casts of the pulp chamber are made, these coronal extensions give the appearance of tentacles arising from a pre-root common chamber and the apical extensions of the true roots (Figs 11.2–11.5). Maxillary teeth have three roots. Details of root apex structure are not available for the horse. In most species, the pulp chamber within the root apices is complex and is referred to as the apical delta. Standard patterns include two or three side branches, giving the appearance of a branching river delta.

As horses age, we recognize a number of changes within the pulp chamber. These can be summarized as a decrease in the cellular components, a tendency for dentinal sclerosis and a decrease in the number and quality of blood vessels and nerves. The most important change, however, is a

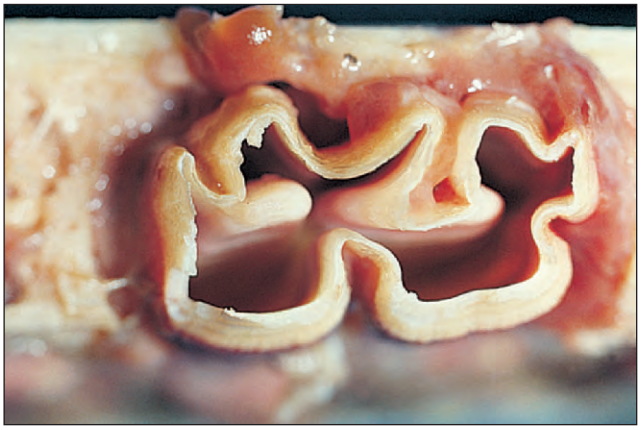


Figure 11.2. Developing root ends over 2 years. Note changes in pulp chamber shape and root formation.

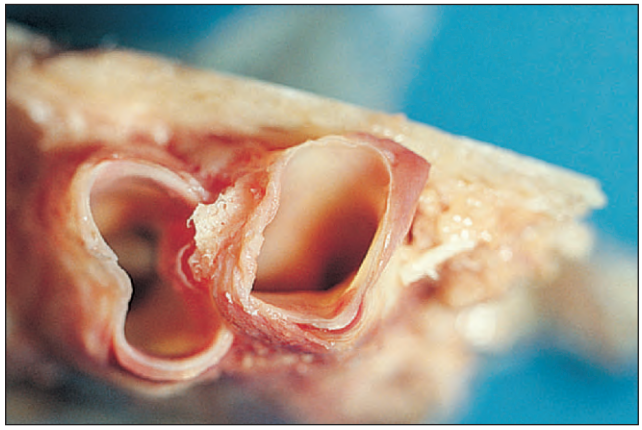


Figure 11.4. Developing root ends over 2 years. Note changes in pulp chamber shape and root formation.

reduction in size and volume of the pulp chamber as a result of the deposition of secondary, and in some cases reparative, dentin. In hypsodont teeth, where there is a pattern of occlusal attrition and, under normal conditions, a matching continuous eruption of reserve crown, it should be clear that without formation of secondary tissue there would be exposure of the pulp cavity. This tissue is secondary dentin and it is formed circumferentially throughout the life of a vital tooth. The mechanism that results in the formation of secondary dentin is complex and its production is initiated by signals transmitted to the pulp through the dentinal tubules. Dentinal sclerosis occurs when the tubules become filled with mineral deposits.[22] This is part of the aging process of the tooth and explains why rates of tooth attrition (the 'wear away' rate) decrease with age, i.e. teeth become more dense with age. Signals that stimulate physiologic secondary dentin production include pressure, temperature and chemical signals.

In the presence of inflammation, the pulp defense mechanism includes the mobilization of histiocytes and their conversion to defensive macrophages. Inflammatory changes result in the release of agents that change capillary and venule blood flow within the pulp. There may be an increase in arteriovenous anastomosis blood flow so that pulp ischemic changes develop, i.e. there appears to be an increase of blood flow within the tooth but the flow bypasses vital tissues of the pulp and ischemia and pulp necrosis results.[23] Because the pulp has no collateral circulation it is particularly vulnerable to this type of injury – as are the lamellae of the foot and hoof. Under such conditions there is again stimulation of pulp odontoblasts, and as a result the production of reparative dentin (this may, in some teeth, be referred to as secondary dentin, irritation dentin or tertiary dentin). Reparative dentin has a variable quality depending on the severity of the inflammatory insult.

Pulpitis in the horse may be caused by a number of factors. Trauma to the mouth and teeth may result in overt and covert tooth fractures which in turn will create periodontal lysis and periapical pathology. Tooth crown fractures and dental procedures, such as the cutting of overgrown teeth, may also penetrate the pulp chambers. It can be stated that under normal occlusal contact and wear, the pulp chambers of the horse do not reach above the gum line. However, care must be taken when working on overgrown teeth because

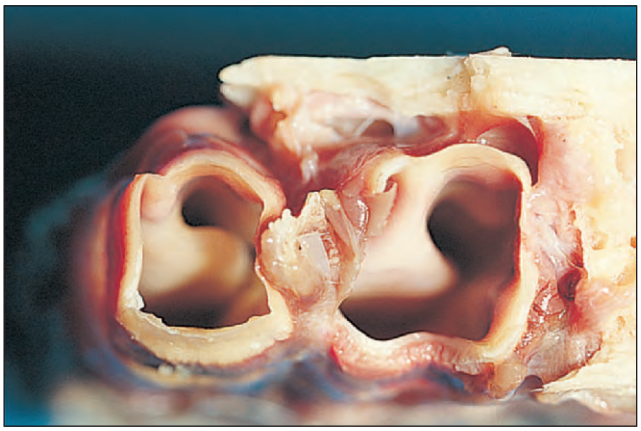


Figure 11.3. Developing root ends over 2 years. Note changes in pulp chamber shape and root formation.

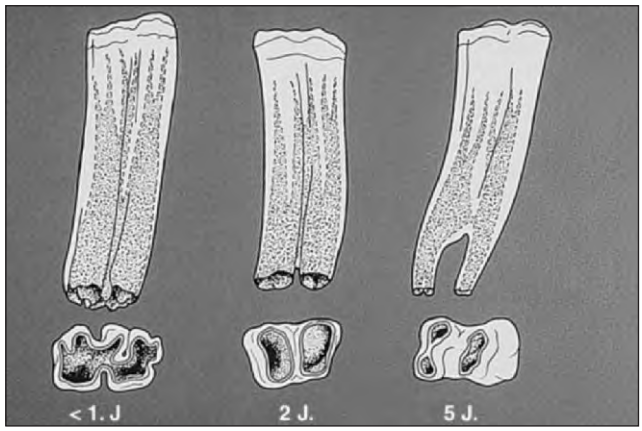


Figure 11.5. Representation of the maturation changes that occur in mandibular pulp chambers.

there is a reduction in the rate of production of secondary dentin and consequently accidental pulp exposure can be a problem. When acute pulp exposure happens, particular techniques are needed to retain pulp vitality (see Chapter 19). It is also clear that other forms of dental work affect the dental pulp. It has been suggested that some human dental procedures are in fact 'cooking the pulp in its own juices.'[23] In recent years there has been an increase in the development and availability of power tools for dental work in the horse. It is important that we recognize the consequences of the thermal effects of power equipment. It should always be accompanied by the use of some coolant, for example an attached, continuous water spray. It is interesting to note that in those species in which pulp responses to thermal effects have been documented there is no safe speed and, in fact, at low rpm rates (under 50,000) there are major changes in odontoblastic effect.

In an *in vitro* study of the power equipment induced thermal changes at the dentinal-pulp chamber junction it has been demonstrated that 2 minutes of contact time, during crown reduction procedures, produced temperature increases above 22°C.[24] It was also demonstrated that water cooling during power reduction precluded the chance of dentistogenic thermal pulp necrosis.

Figure 11.6 is a schematic representation of the possible sequelae of pulpitis in the horse.[5] It can be appreciated that the end result of pulpitis, for example dental fistula, facial swelling and sinus empyema, is based not on the specifics of the pulp disease, but more on the location of the tooth. It is then the local response to the periapical disease that results in the specifics of individual cases and the particular clinical signs that are seen. A constant feature of pulpitis is dental pain. Pain receptors in the pulp are stimulated either directly when exposed, or indirectly via the odontoblast process. Pain is reduced in chronic disease such that in teeth affected by pulpitis, apical osteitis and draining mandibular fistulas may not demonstrate a painful reaction when stimulated.

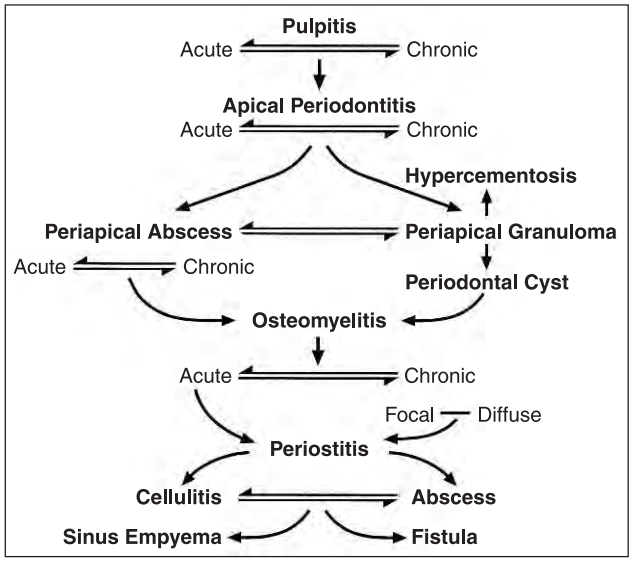


Figure 11.6. Representation of the potential sequelae of pulpitis in the horse.

In case studies on dental extractions for dental decay, it is noted that the teeth most frequently affected are the 8s (i.e. 108, 208, 308, 408).[5] It has been inferred that the effect of delayed and impacted eruptions accounts for major vascular changes in the pulp and subsequent anachoretic pulpitis.[25] In this condition, bacteria are attracted to the inflamed pulp through the dentinal tubules.

As part of a long-term study of 400 cases of equine dental disease in a referral series, 162 horses suffered from primary apical infections of their cheek teeth (CT).[26] There were 92 with maxillary CT infections and 70 with mandibular CT infections. In total there were 193 diseased teeth. In the 84 mandibular CT, 55 involved the second and third teeth (307, 407, 308 and 408) with only 17 found in the 9s, 10s and 11s. There were 12 found in 306 and 406. In the maxillary series (109 teeth), a similar tendency for the rostral teeth to be more frequently diseased than the caudal was found. There were 11 involving 106 or 206 and only 12 involving 110, 210 and 111, 211. Eighty-six teeth were the 7s, 8s and 9s. Ponies were over-represented in the mandibular CT series. Mandibular CT apical infections mainly occurred in younger horses (median age 5 years). Maxillary CT apical infections had a median age of 7 years with a range of 2–24 years. Gross infundibular caries was recognized in 15 of the 92 cases.

Diagnosis of pulpitis in the horse is relatively simple when there is traumatic exposure of the pulp. In most cases, however, the veterinarian does not see the horse until there are fairly advanced clinical signs such as facial or mandibular swelling, fistula formation and loosening of teeth. Radiology is of major importance in documenting the presence of pathological changes resulting from pulpitis and periradicular (around the root) disease (see Chapter 14). The clinician may, however, be misled in the appearance of certain radiographic phenomena in that they are susceptible to multiple interpretations including apical radiolucency. In one study, three endodontists made radiographic interpretations of some 250 films and re-examined the same films 6–8 weeks later. They agreed with their own interpretation only 72 per cent of the time.[27] In an earlier study, six endodontists agreed with each other less than 50 per cent of the time.[28]

Techniques that are available in other species to test for pulp sensitivity and viability such as thermal (hot and cold) pressure and electrical testing have not been used in the horse. There are, however, some indications that these tests will be used in the future as endodontic therapies become more widely adopted.

Summary

Dental decay in the horse is a consequence of the loss of pulpal blood supply, either from direct trauma, usually acute, or from the inflammatory disease processes associated

with pulpitis. The clinical signs depend upon both the chronicity of the disease process and the location of the tooth. In teeth with reserve crowns and roots that are not within the maxillary sinuses the signs relate to apical osteitis and drainage through the surrounding bone and skin, e.g. teeth 308 and 408 and the development of mandibular dental fistulas. In some cases there may also be drainage into the oral cavity. Commonly, dental decay of teeth with reserve crowns and roots within the maxillary sinuses is associated with sinus empyema.

A thorough understanding of the biological behavior of pulp, its response to physical and chemical trauma and the production of secondary dentin, is key to the diagnosis of dental decay and effective case management.

REFERENCES

1. Guerini V (1909) *A History of Dentistry*. Lea and Febiger, Philadelphia, pp. 12–48.
2. Hofmeyer CFB (1960) Comparative dental pathology (with particular reference to caries and paradontal disease in the horse and dog). *Journal of the South African Veterinary Medical Association*, 29, 471–480.
3. Honma K, Yamakarvi M, Yamouchi S and Hosoya S (1962) Statistical study on the occurrence of dental caries in domestic animals. The Horse. *Japanese Journal of Veterinary Research*, 10, 31–36.
4. Baker GJ (1974) Some aspects of equine dental decay. *Equine Veterinary Journal*, 6, 127–130.
5. Baker GJ (1979) A study of dental disease in the horse. PhD Thesis, University of Glasgow, pp. 66–87.
6. Wafa NSW (1989) A study of dental disease in the horse. MVM Thesis, University College of Dublin, pp. 62–72.
7. Easely KJ (1991) Recognition and management of the diseased tooth. *37th American Association of Equine Practitioners Convention Proceedings*, 120–139.
8. Mueller POE (1991) Equine dental disorders: cause, diagnosis and treatment. *Compendium on Continuing Education for the Practicing Veterinarian*, 13, 1451–1461.
9. Dixon PM (1997) Dental extractions in horses: indications and pre-operative evaluation. *Compendium on Continuing Education for the Practicing Veterinarian*, 19, 366–375.
10. Dixon PM (1997) Dental extraction and endodontic techniques in horses. *Compendium on Continuing Education for the Practicing Veterinarian*, 19, 628–638.
11. Miller WD (1889) *Die Mikroorganismen des mundhole*. Liepzig, pp. 163–182.
12. Shafer WG, Hine MK and Levy BM (1963) *A Textbook Of Oral Pathology* (2nd edn). WB Saunders, Philadelphia, pp. 308–377.
13. Bunting RW (1928) Studies of the relation of *Bacillus acidophilus* to dental caries. *Journal of Dental Research*, 8, 222–229.
14. Keyes PH (1960) The infectious and transmissible nature of experimental dental caries. *Archives of Oral Biology*, 1, 304–409.
15. Fitzgerald RJ, Jordan HV and Stanley HR (1960) Experimental caries and gingival pathologic changes in the gnotobiotic rat. *Journal of Dental Research*, 39, 923–930.
16. Stephan RM (1940) Changes in H-ion concentrations on tooth substance and carious lesions. *Journal of Dental Research*, 27, 576–582.
17. Jenkins GN (1961) A critique of the proteolysis-chelation theory of caries. *British Dental Journal*, 111, 311–319.
18. Frank FR (1964) *Veterinary Surgery* (7th edn). Burgen Publishing, Minneapolis, pp. 69–74.
19. Swanstrom OG and Wallford HA (1977) Prosthetic filling of a cement defect in premolar tooth necrosis in a horse. *Veterinary Medicine/Small Animal Clinicians*, 1475–1477.
20. Seltzer S and Bender IB (1990) *The Dental Pulp Biologic Considerations in Dental Procedures* (3rd edn). Ishiyaken EuroAmerica, St Louis, pp. 1–38.
21. Kirkland KD, Baker GJ, Marretta SM, Eurell JAC and Losonsky JM (1996) Effect of aging on the endodontic system, reserve crown and roots of equine mandibular cheek teeth. *American Journal of Veterinary Research*, 57, 31–38.
22. Stanley HR *et al.* (1983) The detection and prevalence of reactive and physiologic sclerotic dentin, reparative dentin and dead tracts beneath various types of dental lesions according to tooth surface and age. *Journal of Oral Pathology*, 12, 257.
23. Kim S and Trowbridge P (1998) Pulpal reaction to caries and dental procedures. In: *Pathways of the Pulp* (7th edn), eds. S Cohen and RC Burns, Mosby, St Louis, Chapter 15, pp. 532–551.
24. Allen ML (2001) An in-vitro study of heat production in equine teeth from power reduction. MS Thesis, University of Illinois.
25. Gutmann JL and Harrison JW (1991) *Surgical Endodontics*. Blackwell Scientific Publishers, Boston, pp. 316–383.
26. Dixon PM, Tremaine WH, Pickles K *et al.* (2000) Equine dental disease Part 4: a long term study of 400 cases: apical infections of cheek teeth. *Equine Veterinary Journal*, 32, 182–194.
27. Goldman M, Pearson A and Darzenta N (1974) Reliability of radiographic interpretations. *Oral Surgery*, 34, 287.
28. Goldman M, Pearson A and Darzenta N (1972) Endodontic success – who's reading the radiograph. *Oral Surgery*, 32, 432.

12 Oral and Dental Tumors

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Introduction

Tumors of the teeth and oral cavity are relatively uncommon in the horse with most reports being of single cases. Surveys of equine neoplasia have shown that most oral tumors are very rare.[1–4] However, when they do occur they are often clinically important and can be life-threatening.[5] Furthermore, even the most astute owners may not notice oral lesions in the early stages and so many tumors are in an advanced disease state when first recognized. This makes the general diagnosis of neoplasia relatively straightforward.[6] However, the gross appearance of many neoplastic masses can be remarkably similar and definitive diagnosis depends on histologic examination.[7]

Confirmation of the exact nature of tumors in the mouth may be difficult because of concurrent long-standing infection or granulation tissue proliferation. The classification of oral or dental masses is also complicated by the existence of some tumor-like conditions that have many of the clinical features of a neoplasm. Indeed some masses have histologic features that support a diagnosis of neoplasia but are, in fact, not cancerous.[8–10] For example, fibrous metaplasia of the nasal region and hard palate have been described[11,12] and benign neoplastic growths are seen occasionally in association with abnormal germinal tissue of tooth roots.[13]

Some non-neoplastic conditions such as epulides, gingival hyperplasia, granulation tissue and hamartoma can give the clinical suspicion of neoplasia. The variable classification of oral lesions also makes initial assessment of tumors difficult, and biopsy is often the only way of establishing the true nature of the mass. There are also some cystic dentigerous conditions that may easily be mistaken for neoplasia. There may also be difficulties, as some tumors fall into the undifferentiated or unclassifiable myxoma/spindle-cell tumor group,[14] which have ill-defined histologic characteristics and variable clinical features. The variation in classification of equine tumors makes the specific diagnosis of many clinically obviously neoplastic diseases difficult and is affected by the variable practice of different pathologists. It has to be recognized, however, that oral and dental tumors are relatively uncommon. This means that individual pathologists are unlikely to have an extensive database of experience at this anatomic site.

Although there have been some advances in therapeutic options the low incidence of these oral conditions makes it difficult to define the best approach to a particular tumor, and we lack comparative efficacy studies for the various modalities. Treatment options may also be affected by the delayed detection of tumors. Many have a benign character but their size may make them impossible to treat by any currently available means. Clinicians frequently have to make compromises from the ideal treatment options.

The prognosis for a particular case is often the primary objective of the clinician. Owners of horses with neoplastic disease are generally more concerned with the prognosis than with the disease itself but some will expect treatment to be successful in every case. As most of these conditions are rare it is often difficult to provide realistic and objective prognosis, with or without treatment. Their course is rarely predictable and so prognosis is frequently equivocal. It is often impossible for a pathologist to provide an accurate prognosis when there are few recorded cases of individual lesions. However, more frequent reporting has improved the understanding of most equine neoplastic disease.

In common with other neoplastic disease, oral and dental tumors can be classified according to their origins[15] (dental, bone, soft tissue) and their behavior (benign/malignant, invasive, proliferative or ulcerative).

Some of the oral neoplastic conditions are very destructive and so there may be extensive secondary changes that are more obvious than the underlying condition. Furthermore, neoplastic tissue is more susceptible to infection and trauma and so the signs may be more severe than the tumor itself warrants. These factors have a considerable bearing on the ability to diagnose the oral lesions simply from clinical supposition and experience.

The presenting signs, which are often subtle in the early stages, include:

1. gross appearance of an abnormal mass of tissue or secondary anatomic alterations due to proliferation or destruction of tissue

2. oral bleeding (hemosalivation/melena)
3. dysphagia/dysmasesis (dysmastication)
4. weight loss
5. secondary changes in other adjacent organs.

For example, oral carcinoma can result in loss of buccal sensation and so the horse may suffer from severe self-trauma to the soft tissues of the mouth. A destructive oral carcinoma involving the palate will produce an oronasal fistula that will probably be recognized first by the presence of a nasal discharge.

While in a few cases clinical judgement can be valuable in the diagnosis, general investigative procedures for oral neoplasia are limited but are nevertheless important. The most important aspects include a detailed clinical examination to establish both the clinical nature and extent of the lesion, the identification of the structures involved and whether these are primary or secondary. A relatively small lesion in the mouth or adjacent structures may be secondary to a much more extensive lesion elsewhere. A good example is lymphosarcoma - the oral or pharyngeal lesions may be clinically insignificant, yet there can be extensive internal organ involvement. Biopsy of any suspected neoplastic lesion is the mainstay of investigation. In many cases it is useful to consult with a pathologist before biopsy to ensure that the best diagnostic specimens are obtained and also so that the pathologist can orientate the specimens correctly in the context of precise anatomic location.

Prognosis will vary markedly with the specific characteristics of the tumor. In some cases these may not be the same as the classical description. For example histopathology may suggest high malignancy but the tumor shows no evidence of this behavior. The converse situation can also arise.

Investigation of a suspected mass should begin with a detailed clinical history and a thorough clinical examination. While biopsy of a suspect mass is frequently performed it should be preceded by investigations that help to establish the extent and possible nature of the condition. For example, it may be very important to know if bone or other structures are involved and this may influence both the site of biopsy and the method required to obtain diagnostic material.

Further diagnostic tests include:

1. Radiography/computed tomography/magnetic resonance imaging:
   (a) These modalities have limited applications for superficial soft-tissue tumors but are very useful for those involving bony structures or for those that are difficult or impossible to palpate or inspect (such as tumors within the paranasal sinuses). It is helpful to establish the presence or absence of any secondary changes in the bone and other structures.
   (b) Radiographic positioning is a critical aspect of diagnostic radiology and in many cases can be achieved or at least assisted, by proper thought about the projections most likely to help.
   (c) Contrast angiography can be a useful aid to surgical and possibly medical therapy, especially in aggressive tumors with large blood supplies.
2. Gamma scintigraphy:
   (a) Gamma scintigraphy can currently be used in a non-specific way to identify small foci of tissue inflammation; the detection of a focus in either soft tissue or bone phase scans presently has low specificity.
   (b) With increasing interest in monoclonal technology using radiolabeled antibody, it is entirely reasonable to expect that the method may be added to the investigative list.
3. Ultrasonography:
   (a) Ultrasonography is becoming increasingly valuable as more sophisticated equipment is developed. The detail of soft-tissue masses can be remarkable.
   (b) Clearly there are limitations in the head region relating mainly to the superficial bones but structures such as the tongue and the orbit can be usefully examined.
   (c) In some cases the nature of the tumor/mass can be identified, e.g., soft-tissue masses can easily be differentiated from those involving bone, and some tumors such as melanoma have a strongly suggestive ultrasonographic structure.
   (d) The blood supply to the mass can sometimes be identified and this may provide useful therapeutic information.
4. Endoscopy:
   (a) Oral examination by a direct light or loupe is helpful in many cases but the most caudal teeth and the oropharynx may be obscured, especially if there are significant tissue distortions either directly or secondarily due to the tumor.
   (b) Oral endoscopy is a risky procedure in the conscious standing horse – even when sedated. There may be logical reasons to perform this under general anesthesia (both to protect the equipment and to ensure a thorough examination). If a lesion is seen a biopsy can be taken. However, specimens that are obtained by endoscope biopsy instruments are seldom diagnostic; the size of specimens coupled with the inevitable artefactual distortion of tissues is such that a reliable diagnosis may be frustrated. Nasal endoscopy can be very helpful in identifying tumors that involve both the mouth and the nasal cavity or paranasal sinuses. Percutaneous sinusoscopy is a useful and simple procedure that can be performed via a small trephine in the lateral wall of the relevant sinus.
5. Hematology and biochemistry:
   (a) Hematological findings are seldom specific but anemia (deriving from chronic inflammation or

paraneoplastic syndromes) and alterations in leukograms may confirm that there are significant secondary effects. Anemia is a common feature of the paraneoplastic syndrome in horses but most primary oral tumors have little or no effect on the major body systems.

(b) There are no specific circulating tumor markers identified for horses yet, but it is likely that these will be identified in time. Meantime non-specific changes including hyperfibrinogenemia, hypoalbuminemia and occasional evidence of the paraneoplastic syndrome (including hypercalcemia and organ failure) are variable hematologic features.

6. Biopsy:

Biopsy is probably the most reliable method of establishing a definitive diagnosis. Biopsy of a suspected neoplastic mass can be achieved by:

(a) *Excisional biopsy.* The whole lesion is removed and examined. There are risks in this process but also benefits: in the event that total excision is achieved the results should be excellent. However, failure to remove all the tumor or contamination of the wound site with tumor cells during surgery can be potentially serious. For example, the equine sarcoid may be removed safely in a few cases but total excision is seldom achieved, and seeding of the wound with tumor cells during the surgery can result in numerous new satellite lesions at the original site.

(b) *Wedge or sectional biopsy.* A small portion of the tumor is removed solely to establish the diagnosis. A rational approach can then be made to treatment selection. It is important to try to select viable tumor tissue and to avoid areas that are ulcerated or necrotic, since such tissues are less likely to yield diagnostically useful information.

(c) *Hollow needle (Trucut) biopsy.* This method is used to obtain a core biopsy through the lesion with minimal damage to the overlying skin or mucosa. A specific location can be selected by clinical judgment or by other methods such as ultrasonography, radiography, or computed tomography.

(d) *Fine needle aspiration.* This is almost atraumatic to the tumor and so there are much reduced risks of significant tumor interference. The technique is often performed badly and the specimens are often handled badly – collection of aspirated cells must be performed with care and requires all formalin solutions to be removed completely from the environment. Bad specimen handling means that the method has a poor reputation that is not entirely justified. The gauge of needle used should be selected to allow a suitable sample to be obtained. A soft or fluid-filled mass can usually be aspirated with an 18-gauge needle (or finer). Solid masses require larger diameter needles and firmer suction. The selected needle is inserted into the tumor mass and a 10 ml syringe attached to it. Repeated short but intense aspirations are made before the needle is withdrawn. The point of the needle is then directed at a clean, grease-free glass microscope slide, and the cells 'jetted' onto the slide by repeated ejection of air from the syringe. Thick preparations should be smeared rapidly before being air-dried or fixed according to the requirements of the pathologist. This method requires interpretation by a skilled cytopathologist because low numbers of tumor cells may not be easily recognized among normal ones. Tumors vary in the ease with which they are aspirated, so the yield of cells may not always support a firm diagnosis. Again it is important to avoid areas that are clearly necrotic or inflamed or infected, since the cytology of such areas is not diagnostically critical. An important practical consideration for cytology is to ensure that smears are not exposed to formalin fumes since the latter spoils cellular preparations for subsequent staining with Romanowsky cytological stains.

Notwithstanding the specific tests that can be applied in the investigation of oral and dental tumors, most of the commoner conditions are fairly distinctive, and a tentative diagnosis can usually be made. Problems may arise with rarer tumors and those with prominent secondary inflammation and necrosis.

Oral tumors are conveniently divided into:

- primary tumors of dental, soft tissue or bone origin
- secondary tumors of non-oral tissues.

The latter are rare but must be considered when investigating an oral mass.

Classically, primary oral neoplasms are divided into three groups according to the tissue of origin:

1. teeth (odontogenic) tumors
2. bone (osteogenic) tumors
3. soft-tissue tumors.

The lack of reports of extensive series of individual oral tumors and tumor-like masses testifies to the fact that most of these conditions are uncommon[16–19] and that no serious multicenter attempt has been made to classify them and to quantify their prevalence. The specific difficulties that are presented by the tumors and their profound effects (whether benign or malignant) means, however, that veterinarians are expected to make prognostic decisions that are inevitably based on limited experience. Recommendations for treatment of rare conditions cannot be made with any certainty, and pathologists are often expected to provide information that simply does not exist. The reported satisfactory or unsatisfactory treatment of a single reported case does not entitle pathologists or clinicians to refer to 'common' treatment or 'usual' tumor behavior.

Table 12.1. Equine Dental Tumors Derived from Odontogenic Epithelium (E) or Mesenchyme (M)

Histologic designation	Synonyms
Ameloblastoma (E)	keratinizing ameloblastoma adamantinoma
Cementoma (M)	
Complex odontoma (E)	
Cementifying fibroma (M)	
Ameloblastic fibroma (E)/(M)	ameloblastic fibro-odontoma

Tumors of dental-tissue origin (odontogenic tumors)

Tumors in this category are all rare, although it has been suggested that they are commoner in horses than in other species.[17] Odontogenic tumors are classified according to the inductive effect of one dental tissue on the others.[18,20] These tumors are almost always clinically benign but are frequently locally invasive and aggressive. As a general rule, dental tumors are best treated by wide surgical removal (to ensure complete ablation of tumor and abnormal tissue) at an early stage in their development when such surgery has a chance of success. In most cases, however, the masses are not recognized sufficiently early and so local recurrences are common in spite of attempts at wide surgical excision.[19] Most oral bone and dental tumors are benign but can have serious secondary effects such as nasal obstruction and dental and facial deformity, resulting in dysmasesis and weight loss.

Odontogenic tumors are of variable histologic appearance and are categorized currently on their morphologic basis[20] (Table 12.1). Their features are summarized in Table 12.2.

Ameloblastoma

Definition

These tumors are derived from odontogenic epithelium. True ameloblastoma produce no inductive changes in the connective tissue and so lack dentin and enamel.

Occurrence

These are commoner in the mandibular region of older horses[21–26] but can involve the maxilla and the medullary cavity of the mandible. Several cases have also been reported in young foals.[27,28]

Clinical features

They may be overtly tooth-like or very mixed masses with no obvious dental tissue. They often develop a central cystic region and cause bony/solid swellings and abnormalities in the associated dental arcade (Fig. 12.1A). Occasionally they can present with a discharging sinus on the side of the face.

Differential diagnosis

Ossifying fibroma and other tumors of the jaw such as invasive squamous cell carcinoma and myxomatous tumors should also be considered (although the latter tend to be destructive rather than proliferative). Osteosarcoma is singularly rare in the horse. Infections of tooth roots, radicular cysts and adjacent bone can be similar but are associated with extensive necrosis and typical radiographic features often complicated by maxillary reactive bone proliferation with obvious facial swelling. Jaw fractures and other dental abnormalities, including malerupting and supernumerary cheek teeth, should also be considered.

Diagnostic confirmation

Biopsy and radiographic findings are typical but can be similar to other tumor masses. Ameloblastomas usually have a rubbery consistency and have a roughly spherical or multilocular shape with a cystic radiographic appearance (Fig. 12.1B). Odontomas are radiolucent or partially mineralized with foci of enamel tissue mixed throughout.

Table 12.2. Summary of Features of Odontogenic Tumors Based on Published Characteristics[20,36]

Tumor type	Age group affected (yrs)	Clinical behavior	Best treatment option	Prognosis
Ameloblastoma	wide range	benign/locally invasive	surgical excision/ hemimandibulectomy ± radiation	fair–good; eating difficulties may be severe
Ameloblastic odontoma	<3	benign/locally invasive	surgical excision/ hemimandibulectomy ± radiation	fair–good; eating difficulties may be severe
Cementoma	onset uncertain	benign	surgical removal	good
Complex odontoma Compound odontoma	<5	benign malformation	surgical removal	fair (if removal feasible)
Cysts/hamartoma	various	benign	surgical removal	fair (if removal feasible)

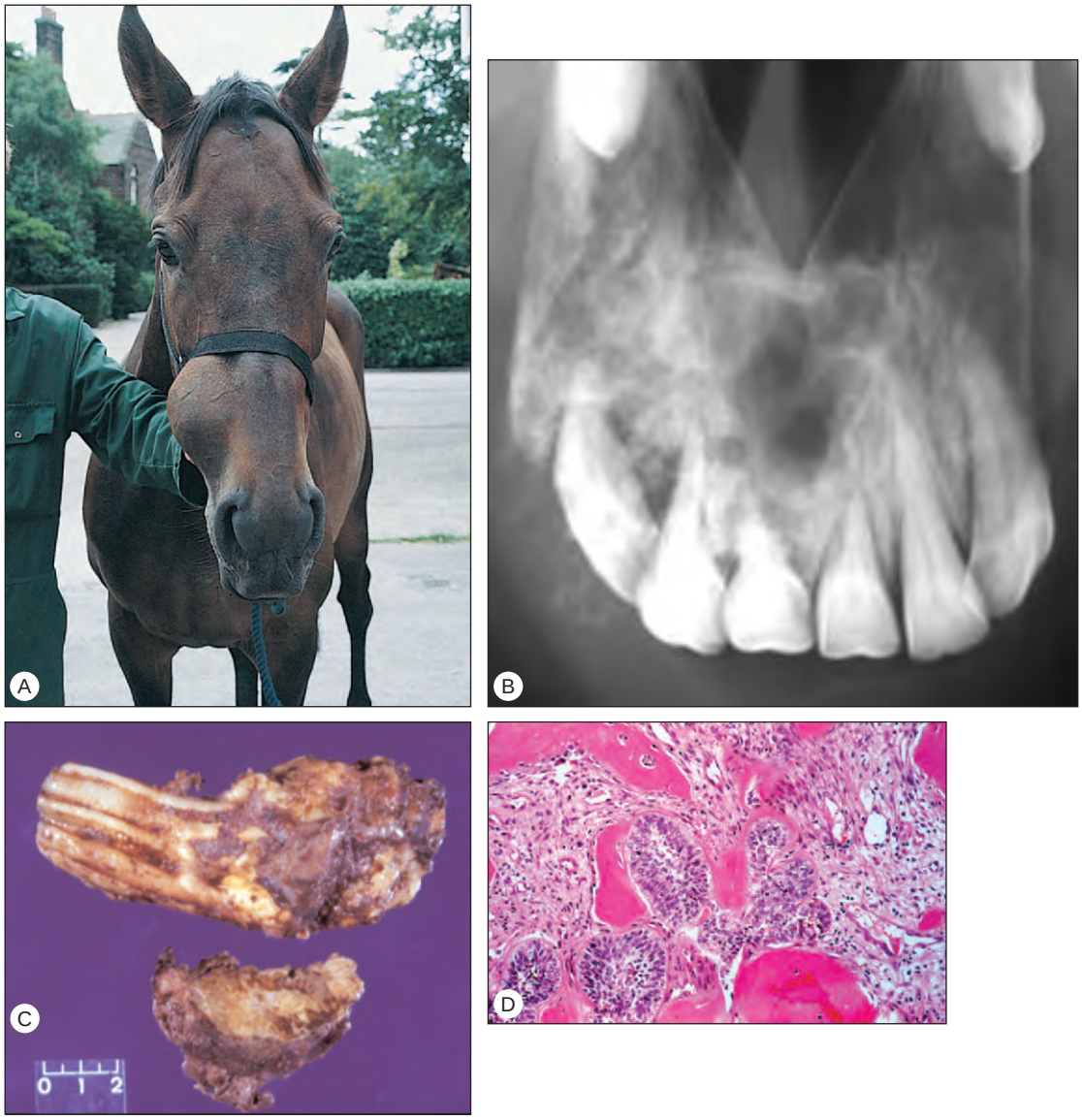


Figure 12.1. (A) Facial swelling caused by an ameloblastoma. The tissue contained no obvious dental tissue remnants. This differentiates it from an ameloblastic odontoma. (B) Radiographic appearance of an ameloblastoma in a 2-year-old thoroughbred colt showing the characteristic multiloculated nature with radiodense fragments throughout the mass (reproduced with the permission of Dr Bruce Bladon). (C) An ameloblastoma excised from a 2-year-old thoroughbred colt showing the relationship to the tooth and the expansive mass at and around its root. (D) Histologic section of an ameloblastoma showing clusters of orderly ameloblasts (arrows) separated by connective tissue and spicules of hard dental material.

Even when there is extensive ulceration there should be little confusion between this and carcinomas or sarcomas, which are much more destructive than tumors of dental origin.

Pathology

These lesions are characterized grossly by swelling of the affected jaw and osteolytic changes within the jaw outline of the lesion (Fig. 12.1C). They can be solid or cystic and are usually discrete. The major characteristic histologic feature is the presence of odontogenic epithelium (Fig. 12.1D). If there is marked epithelial keratin formation the lesion is termed keratinizing ameloblastoma. The lesion may be well circumscribed or there may be local infiltration by odontogenic epithelium.

Treatment

Surgical removal can be curative if treatment is initiated early and wide excision can be performed. Radiation is probably the best option and has been used successfully.[29]

Prognosis

The expansile nature of these tumors and their late recognition (particularly in foals and young horses) make the outlook poor. Many horses are euthanased soon after they

are diagnosed with the tumor, although the rate of growth may be slow and some useful quality of life may be possible.

Cementoma

Definition

Rare; an unusual benign or reactive change derived from mesenchymal tissue without epithelial components. It typically occurs in the root region of the developing tooth. There are few published reports of this tumor but one such lesion affected an incisor tooth (DCK, unpublished). It is possible that some of the features of this condition could be found in abnormal or supernumerary cheek teeth where extensive distortion of the dental structures is encountered.

Clinical features

The location of these tumors (at the base of the tooth) makes their early recognition and diagnosis unlikely. Only when there is overt swelling can the possibility be explored (Fig. 12.2). Radiographically they have a distinctive very dense appearance and the tissue contains sheets of cementum-like material. Secondary alveolar changes involving either infection or reactive bone proliferation may however, make them harder to recognize. Alterations in the crown structure are unusual but will make the condition easily recognizable.

Pathology

This lesion presents as a mass in the jaw or as a mass that involves the nasal cavity or maxillary sinus. The lesion may be secondary to traumatic tooth fracture, dental impaction or periodontitis. It is characterized histologically by mosaic-like basophilic cement lines and by anchoring of Sharpey's fibers into cement matrix. With reactive cementoma there is additional inflammation and fibrosis. Cementifying fibroma is a rare lesion that is analogous to ossifying fibroma, but the tumor matrix includes the complex basophilic lines of typical cementum.

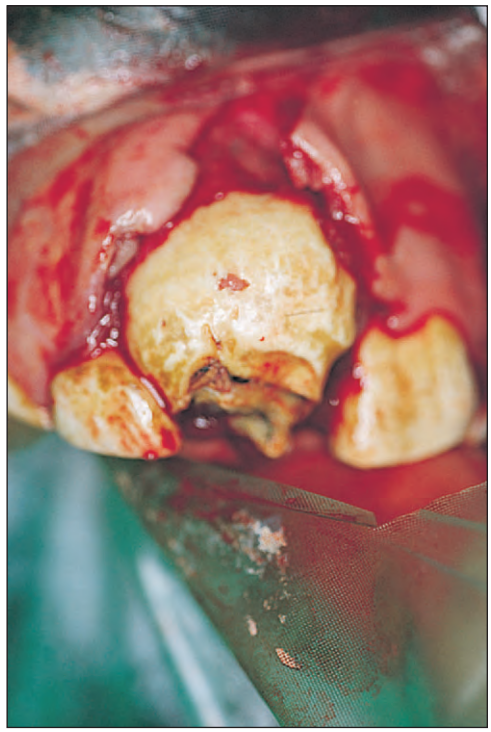


Figure 12.2. Cementoma of the crown of an incisor tooth in a 3-year-old Hanovarian mare.

Treatment

Removal of the tooth in its entirety is feasible but may be hindered by the ball-like aggregation of hard tissue at the tooth root.

Prognosis

The lesion is benign and removal is curative. The empty socket simply fills with dense fibrous tissue.

Complex/compound odontoma

Definition

Irregular tumor-like mass of dental tissues in well-differentiated form. There is a marked inductive effect on mesenchymal tissue in both forms. There is some justification for considering some of these lesions to be hamartomas of dental tissue rather than tumors. Complex odontoma contains all the elements of a normal tooth but the structure is chaotic. Compound odontoma is similar except that the tissue is organized into a recognizable tooth-like structures (denticles), although they may be grossly distorted.

Occurrence

Both young and older horses may be affected with a greater prevalence in younger animals.

Clinical features

Firm painless swellings over the root regions of the maxillary cheek teeth or the premaxilla are characteristic.[30] Swelling may not be obvious if the more caudal four cheek teeth are involved as the expansion would be contained in the maxillary sinuses (Fig. 12.3A).

Differential diagnosis

Dental disease with new bone formation and lysis. Sinus cysts may be related to these in some cases.

Diagnostic confirmation

The radiographic appearance is characteristic – multiple small lobulated masses within a well-defined cyst-like structure at the root of a maxillary tooth are typical.

Pathology

Complex odontoma presents as a radiodense lesion within the jaw of young horses. Grossly they are very hard and difficult to cut. Cut surfaces reveal variegated cementum, dentin and mineralized enamel (Fig. 12.3B).[31] The gross features are confirmed histologically and there can be variable amounts of odontogenic epithelium. In the horse there is plentiful cementum.



Figure 12.3. (A) The gross distortion of the maxilla due to a compound odontoma. (B) Undecalcified thick section of a complex odontoma in a 2-year-old Morgan colt. Well-differentiated but disorganized components of tooth formation are shown (D = dentin; C = cementum; E = enamel). (Fig. 12.3B reproduced with the kind permission of Dr RR Dubielzig and Iowa State Press.)

Compound odontoma presents as a similar lesion but radiography shows several abnormal tooth-like structures (denticles) within the mass. Histologic features are reminiscent of normal tooth development. In older lesions epithelial tissue may be sparse.

Treatment

Surgical removal may be feasible and curative. Surgical removal and cryosurgery combined can be used but some masses require more than one treatment.

Prognosis

Full surgical removal should resolve the problem but repeated surgery may be needed. There are insufficient reports to establish a definitive prognosis but the few published cases do suggest that the outlook is reasonable or even good.

Incidental tumor-like masses

This group includes radicular cysts and temporal teratoma. Temporal teratoma is a rare curiosity in the horse, in which dental tissue (which may be instantly recognizable as such) is located at sites away from the jaws (Fig. 12.4A,B). The commonest site is in the temporal region where a sinus tract discharges glairy, milky material from a discrete opening on the leading edge of the pinna. The cystic structure may be situated some way from the ear itself but sometimes there is an obvious dental structure located against or attached to the temporal bone of the calvarium. Sometimes the structure has no obvious dental tissue and comprises a smooth cystic lining lying below the ear.

Radiographs are used to establish the presence or absence and the location of the dental tissue.

Pathology

These are rare lesions that present as open masses or cystic cavities of the temporal region inferior to the ear canal. They are lined by stratified squamous epithelium and contain abnormal dental structures.[32–35]

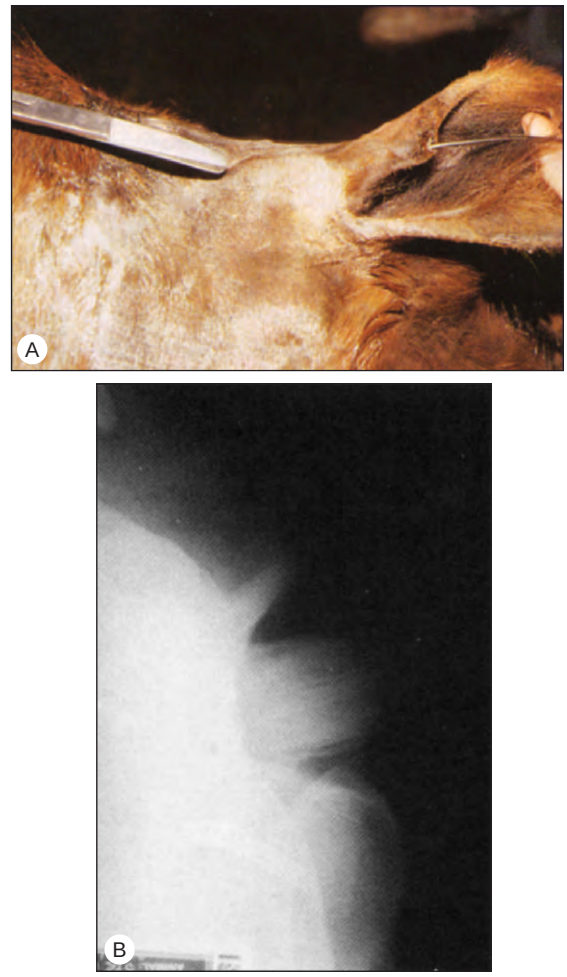


Figure 12.4. (A) A discharging sinus had been present on the anterior margin of the pinna (probe inserted) for 2 years. The tract leads to an obvious solid non-painful mass just rostral to the base of the ear. This is the typical clinical appearance of a dentigerous cyst. (B) Oblique radiograph of the temporal region of the same horse as in (A). An obvious tooth-like structure with an associated 'alveolus' is present and is typical of many cases of dentigerous cysts. In the absence of obvious tooth-like material contrast radiography will identify a distinct (or occasionally) a poorly defined cystic structure at this or a neighboring site.

Tumors of bone (osteogenic tumors)

Osteosarcoma, osteoblastoma, chondrosarcoma and fibrosarcoma have been described as arising in bone. They are all very rare in horses but there have been several reports of these in the regions of the jaws and the mandible in particular.[37] The histologic characteristics of bone-derived tumors have been described[38] and the classification of this group of tumors is based on these features.

Osteoma

Osteomas are usually slow-growing solitary, well-differentiated masses of bone enclosing marrow and fat, and many pathologists regard them as a developmental anomaly or hamartoma. They are benign but may cause some local difficulty because their growth compromises adjacent soft tissues. They cause disfigurement, obstruct the nasal passages and interfere with mastication and swallowing. Common sites include the mandible, maxilla and paranasal sinuses. The osteoma may reach a large size and have a distinctive discrete radiodense outline. They are reported in all ages of horse, with most being in the head region and in the mandible in particular.

They consist macroscopically of dense bone. The histologic features are of orderly cancellous bone; intertrabecular fibrous connective tissue may include adipocytes and hemopoietic cells.

Osteosarcoma

Definition

A malignant mesenchymal tumor in which the neoplastic cells produce modified or distinctive osteoid or bone matrix in haphazard arrangement.

Occurrence

These are very rare tumors in the horse: in this species over 80 per cent of reported osteosarcomas involve the head, and the majority are reported in the mandible.[39,40] There is a report of an osteosarcoma in the mandible of a 6-month-old quarterhorse colt, which suggests that age is probably not a significant factor,[41] although, typically, younger horses appear to be more prone to oral or dental neoplasia than older ones.

Clinical features

Painful, hot progressive swelling of the mandible with a characteristic 'sun-burst' appearance of bone lysis and irregular deposition of trabecular reactive new bone[42] (see also Chapter 14).

Differential diagnosis

Infection resulting in osteitis or osteomyelitis can be very destructive. Various cystic structures such as ameloblastoma, ossifying fibroma and fibrous dysplasia can be similar but usually have characteristic radiographic differences.

Diagnostic confirmation

The radiographic appearance is highly suggestive but biopsy provides the only definitive diagnosis. Bone biopsies should be examined in the context of preoperative radiographs so that the anatomic site of the biopsy sample is clearly defined. It is easy to miss tumor tissue in small bone biopsies and florid, non-neoplastic reactive bone or fracture callus can easily be mistaken histologically for neoplasia.

Pathology

Several histologic types are recognized in species where the incidence is higher but it is so rare in horses that it is probably unwise to extrapolate from these. The tumor tissue is, however, usually not densely cellular with formation of fibrillar stroma, bone or osteoid tissue. The cells have a high mitotic index and an atypical irregular morphology. Trauma is implicated as a risk factor for later osteosarcoma in other species such as the cat but there is no convincing evidence for this in horses.

Since this tumor is so rare in the horse, the expected histologic appearance is a speculative one based on experience of the lesion in other species. Osteosarcomas are characterized by painful bony swellings with variable degrees of bone lysis, tumor bone formation and reactive periosteal bone proliferation. The histologic features are of neoplastic osteoblasts with variable numbers of osteoclasts. The extent of formation of tumor osteoid and/or bone is variable. The tumor bone may, therefore, be hard or soft and hemorrhagic.

Treatment

Radiation offers the only hope of success but the tumors are likely to be highly malignant and so treatment is usually not contemplated. However, some progress relatively slowly and are therefore at least tolerable for limited periods. Euthanasia is the only realistic option.

Prognosis

There is insufficient data for reliable prognosis. However, the highly aggressive nature and rapid course probably justify a hopeless prognosis.

Tumors of soft-tissue origin

Clinical features of some of the equine oral soft-tissue tumors are summarized in Table 12.3.

Squamous cell carcinoma

Definition

A malignant neoplasm of stratified squamous epithelium.

Occurrence

Squamous cell carcinoma (SCC) is probably the commonest oral neoplasm but is nonetheless rare. Although mucocutaneous junctions are commonly involved outside the mouth

Table 12.3. Summary of the Clinical Features of Some of the Equine Oral Soft-tissue Tumors

Tumor type	Age group most often involved	Clinical behavior	Treatment options	Prognosis
Squamous cell carcinoma	5–14 y	benign but can be very destructive	radiation cisplatin	guarded
Sarcoid	all	locally invasive; only malignant form can be very aggressive	radiation cisplatin surgery (nodular type only)	very guarded
Melanoma	>7 (gray horses)	low	benign neglect cisplatin cimetidine per os	fair a few are malignant
Oral papilloma	<2–3 y	benign	leave alone surgical excision	good but some persist
Epulis	>10 y	benign	dental hygiene/descaling	good
Salivary adenocarcinoma	>10 y	malignant	radiation possible	hopeless
Equine juvenile ossifying fibroma	2 mo–2 y	benign	surgical excision	fair–good
Myxoma/myxosarcoma	n/k	variable	n/k	poor–hopeless

n/k = not known/insufficient reports to make a judgment.

where there is an apparent correlation with non-pigmented skin and high levels of ultraviolet light, many of the most severe and aggressive tumors occur within the mouth.[43] The role of ultraviolet light in the pathogenesis of facial and lip carcinoma is uncertain but the Clydesdale breed and horses with non-pigmented skin of the face and lips are more often affected than other breeds and colors.

There is often a suggestion that the primary tumor develops in the paranasal sinuses or nasal cavity and the destructive tissue involves the hard palate but it may be difficult to identify whether the primary lesion is in the sinus or the palate.[44] SCC possibly arises in chronically irritated hyperplastic alveolar epithelium in cases of chronic periodontitis.

Clinical features

The tumors are characteristically slow growing. They can be proliferative but are usually very destructive, ulcerative and infiltrate widely into local tissues of the mouth including the lips, buccal mucosa, hard palate and tongue. Metastases to local lymph nodes can occur (see Fig. 12.5H) and, in theory at least, may disseminate to the lungs and elsewhere. However, this behavior is rare in oral forms of SCC.[45]

Oral SCC may involve the lips (Fig. 12.5A), hard palate[46] (Fig. 12.5B), tongue[46,47] (Fig. 12.5D,F) or oral mucosa. It is also quite common for oral SCC to invade the nasal cavity and the paranasal sinuses (often to the point of gross distortion or obstruction to airflow).[48] Some involve the pharynx (Fig. 12.5E) and can physically affect its function. In nasal SCC there is altered airflow (or even complete obstruction of the ipsilateral nostril). In the latter case the patient may be presented with dysphagia of progressive, insidious onset. In both cases weight loss and poor general health are common.

More extensive spread may involve the orbit and the cranial cavity with secondary involvement of the eye or rostral brain. It is also quite common for nasal SCC to invade the hard palate forming an oronasal fistula. In all cases there may be extensive soft-tissue disruption and consequent loosening/shedding of the teeth (Fig. 12.5B,C).

The location of SCC often means that tumors are detected late when a large invasive mass may be present projecting from the gum or hard palate as a grayish, ulcerated and bleeding mass. Where the tumor surrounds a tooth this may be become dislodged and in almost all cases there is a fetid odor from the mouth.

Differential diagnosis

The differential diagnosis includes other proliferative and invasive lesions of the lips, including equine sarcoid, hemangiosarcoma, and myxomatous tumors.

Diagnostic confirmation

Biopsy is characteristic. Fine needle aspirates can be used but may be misleading, since they often consist mainly of stroma and inflammatory cells.

Pathology

Histologically the tumor has distinctive characteristics with irregular cords of downward-invading neoplastic

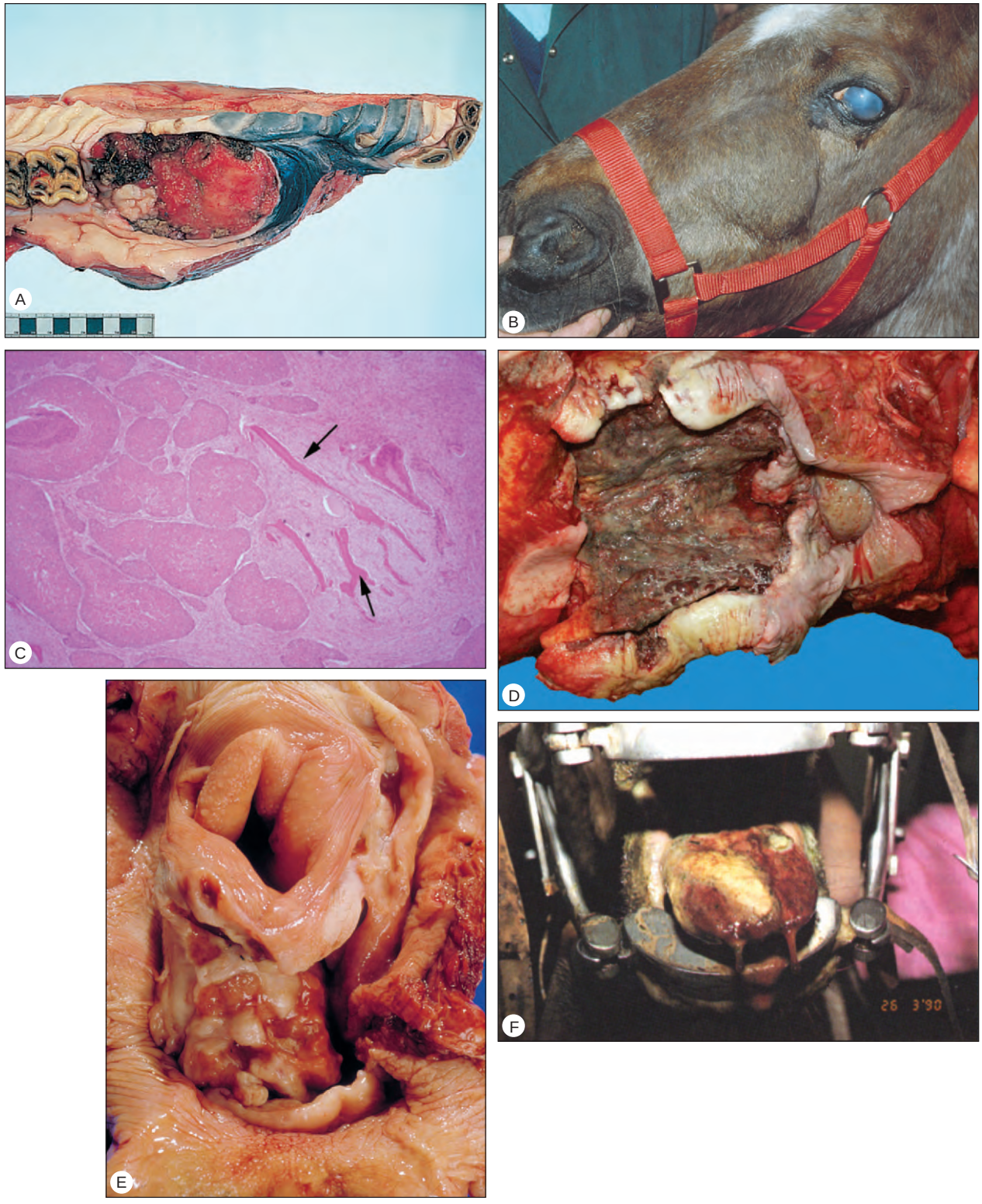


Figure 12.5. (A) A destructive carcinoma on the lip of a 23-year-old pony gelding. Treatment with intralesional cisplatin and topical 5-fluorouracil was not effective. (B) Facial distortion due to nasopalatine squamous cell carcinoma in a 12-year-old pony gelding. Note the extensive tissue destruction and the loss of the maxillary teeth. (C) A histologic section of the endonasal carcinoma shown in (B). Solid cords of squamous carcinoma are surrounded by fibrous stroma and trabeculae of non-neoplastic reactive bone (arrows). (D) This horse was presented with weight loss and dysphagia. The highly destructive and invasive carcinoma was not visible endoscopically from the pharynx and was only seen with difficulty during an oral examination. (E) Squamous cell carcinoma of the pharynx which was identified some 3 months after a lesion had been detected in the hard palate. It is possible that this developed independently or that it was an extension of the earlier lesion. (F) Carcinoma of the free portion of the tongue. The local lymph node was enlarged (see (H)) (slide courtesy of Dr RR Pascoe).

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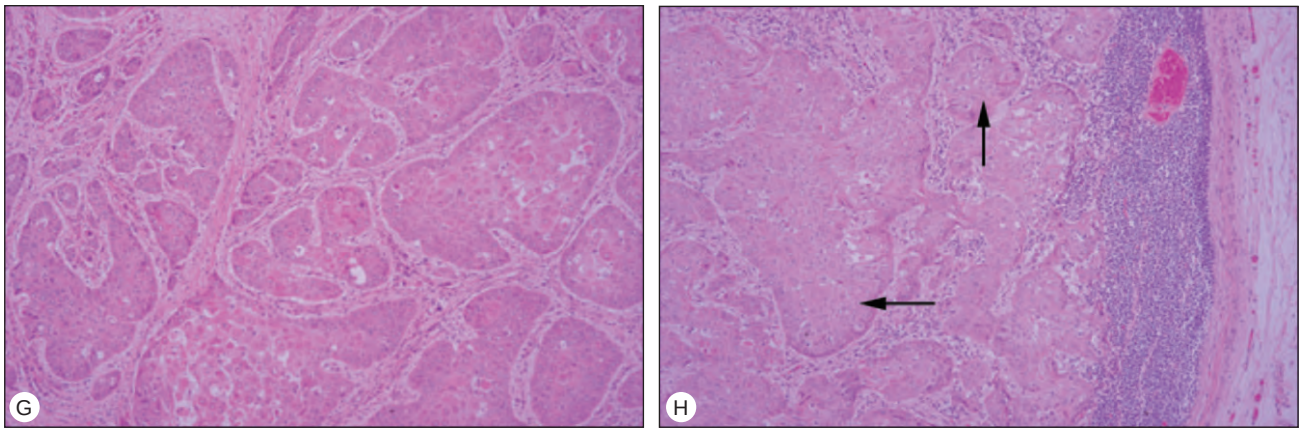


Figure 12.5, cont'd, (G) Photomicrograph of cords of solid squamous cell carcinoma with deep invasion of the tongue. (H) Metastasis of squamous cell carcinoma to regional lymph node showing cords of tumor cells (arrows) beneath cortical lymphoid tissue.

keratinocytes. The accumulation of variable amounts of keratin produces 'keratin pearls.' SCC characteristically has large amounts of non-neoplastic fibrous stroma in which inflammatory cells are plentiful. The abundant stroma results in a lesion that is characteristically tough or scirrhous when palpated or excised.

Radiographic examinations can be used to identify masses in the sinuses and the extent of bone destruction produced by invading carcinoma.

Treatment

While surgical excision of oral SCC lesions has been reported to be successful,[49] this on its own can be very difficult. There is a very high rate of recurrence following such attempts. Small discrete tumors may, however, be amenable to surgical removal, and extensive excision involving hemimandibulectomy, such as has been described for other tumors of the jaw, will also possibly resolve the condition but may leave an unacceptable cosmetic or functional deficit.

Squamous cell carcinoma appears to be relatively sensitive to gamma radiation and this offers the best prognosis with a reasonably high success rate (DCK, unpublished observations). Teletherapy is logical and can be finely controlled but repeated fractionated doses need to be used, and the horse therefore needs repeated general anesthesia. The number of centers where this can be performed is small and the procedure is necessarily very expensive. The much simpler iridium-192 interstitial brachytherapy using linear platinum-sheathed wires has been used to good effect (DCK, unpublished observations). There are serious logistic and human health risks, however, and limits to size and location of the tumors that can be treated. Placement of the sources within the highly mobile tissues of the mouth carries serious dangers if the horse were to dislodge the wires and swallow them.

Some SCCs respond to intralesional cisplatin*, either in water-soluble form with frequently repeated injections, or as an emulsion of the solution containing at least 1 mg/ml with an equal volume of sesame or almond oil.[50] The use in oral SCC has apparently not been reported but the material carries operator safety risks.

The response to immunomodulation using mycobacterial protein materials such as Bacillus Calmette Guerin (BCG) is disappointing in horses when compared to treatment of squamous cell carcinoma in other species such as cattle (DCK, unpublished observations).

Treatment of labial SCC with 5 per cent fluorouracil cream applied topically has been shown to resolve some cases and improve others[51] but cures may be difficult to effect using this alone. It is, however, a very useful adjunct to other forms of treatment and may be particularly applicable to small, ulcerated, buccal and lip lesions.

Prognosis

The tumors are always locally invasive but slow to metastasize, so while the clinical prognosis is inevitably poor many cases can survive long periods even with quite extensive involvement. Secondary complications such as facial or oral distortion, dysphagia, loosening of teeth and nasal obstruction inevitably suggest a poorer prognosis. However, oral SCC is a low-grade invasive tumor that tends not to metastasize beyond the local lymph node. However, it is probably unwise to assume that this will be the case in all affected patients.

Sarcoid

The equine sarcoid is a very common fibroblastic tumor of the skin and can involve the mouth.[52,53] The term sarcoid is used clinically to describe a spectrum of cutaneous tumors that variously involve connective tissue and epithelium with a range of clinical behavior. There are usually two distinct forms that affect the mouth itself (as opposed to the skin of the lips and cheeks). The nodular form remains subcutaneous and is most often located at the angle (commissure) of

*cis-diamminedichloroplatinum, or cis-DDP, cis-platinum.

the mouth. The malevolent/malignant form[54] also occurs in the tissues of the cheeks in particular and can ulcerate into the mouth. The verrucose form often involves the perioral skin.

Clinical features

Intracutaneous or subcutaneous nodules with an ulcerated surface are the commonest oral manifestation of sarcoid (Fig. 12.6A). The nodules frequently ulcerate either on the cutaneous surface or into the mouth. The verrucose form is also a common type in the perioral skin but does not involve the oral mucosa. Combinations of nodules within the skin and cutaneous involvement of verrucose sarcoid are also common (mixed sarcoid) (Fig. 12.6B). Nodules may extend through the cheek musculature into the oral mucosa (Fig. 12.6C). The malignant form has various combinations of the sarcoid types but is highly invasive and nodules may be linked by cords of sarcoid tissue (Fig. 12.6D). Sarcoid



Figure 12.6. (A) A localized ulcerated nodular sarcoid. (B) A mixed sarcoid with verrucose and ulcerated nodular components. (C) A deep-seated nodular sarcoid involving the facial skin and extending into the buccal mucosa. (D) A locally invasive (malignant) sarcoid in the cheek with extensive deep involvement of the muscles and oral mucosa.

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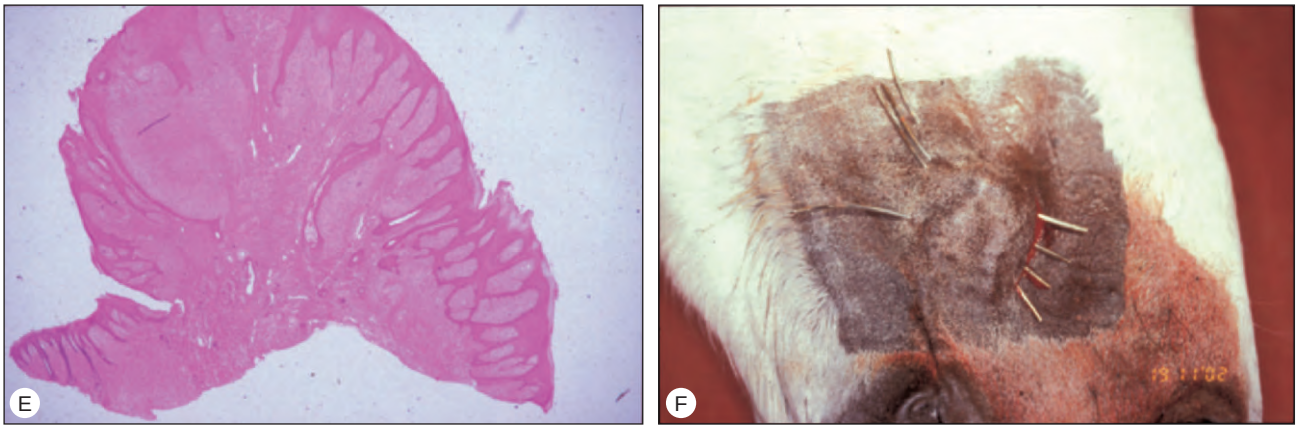


Figure 12.6, cont'd, (E) Histologic section of verrucose sarcoid showing a bulging exophytic lesion with pseudoepitheliomatous hyperplasia of the epidermis and diffuse subepithelial fibroblastic proliferation. (F) Linear iridium-192 interstitial brachytherapy successfully used to treat an invasive sarcoid in the cheek.

has not been reported on the tongue or other soft tissues of the mouth.

Differential diagnosis

The equine sarcoid can resemble some forms of viral papilloma and the nodular forms may be mistaken clinically for melanoma and for inflammatory nodules (e.g., foreign body granuloma).

Diagnosis

Because of the dangers of biopsy[55] a tentative clinical diagnosis is usually made. Horses with a single sarcoid lesion located in the mouth without any other lesion are very unusual. Horses that have the characteristic features and show lesions at other sites can usually be assumed to have sarcoid. Biopsy is not usually recommended but the histologic features are characteristic.

Pathology

The clinical term 'sarcoid' encompasses a histologic spectrum of fibroblastic tumors that may be accompanied by a variable epithelial component. The fibromatous variant is grossly well circumscribed, solitary or multiple with tough, pale fibrous cut surfaces. Adjacent epidermis is often attenuated and may be intact or ulcerated. Most of the lesion consists of randomly arranged well-differentiated fibroblasts with plentiful collagen. The malignant variant has ill-defined margins; adjacent epithelium may be ulcerated or intact. Histologic features are of randomly arranged, activated fibroblasts that form interlacing bundles and whorls. Individual tumor cells have degrees of anisokaryosis, and mitoses may be plentiful. At the histologic level it may be difficult to discern the limits of the tumor, especially in small tissue samples. The verrucose sarcoid has histologic features similar to those in the malignant form and is usually associated with marked pseudoepitheliomatous epidermal hyperplasia (Fig. 12.6E).

Treatment

The options are limited. In some cases the lesions can justifiably be left alone in view of the risks of exacerbation by incomplete excision. However, inadvertent trauma can also result in severe deterioration and so early treatment may be strongly recommended in individual cases.

Treatment of buccal forms of the disease is notoriously difficult, with radiation, cryosurgery, hyperthermia, laser excision and intralesional cisplatin carrying some chance of success. Referral to a specialist center is probably justified simply on the grounds that failure of a treatment may result in significant exacerbation of the lesion.

The best treatment is undoubtedly with radiation either as brachytherapy using interstitial linear or pelleted radioisotopes with a gamma emission capability. Radiation has a cure rate of over 95 per cent and the cosmetics of the method are impressive. The most frequent isotopes used include iridium-192 and gold-198. The former is presented in linear sources sheathed with platinum that renders the isotope effectively a total gamma emitter. Gold-198 is used as platinum pellets. The latter has a very short half-life (48 hours) and so this is logistically easier to handle (the sources do not need to be removed) but clearly this method carries much higher operator risks than the lower emissions over a longer period characteristic of iridium. Linear iridium sources are left *in situ* for the calculated period to deliver the required radiation dose and are then removed (Fig. 12.6F). During the treatment time the horse must be confined within an approved radiation unit. Complications involving wire displacement and injury or colic during the treatment period can add considerably to the logistic problems. Teletherapy is an ideal method of treatment but there are very few facilities for this at this time.

Other treatments carry a correspondingly worse prognosis with surgery being the most difficult. Intralesional cisplatin has recently gained some reputation[56] but the method carries very serious carcinogenic risks for operators and handlers

alike. The method should be restricted to specialist institutes where facilities for fecal and urine disposal ensure that risks to people are minimized.

Topical and intralesional cytotoxic chemicals such as 5-fluorouracil have also been used with very variable results. They all require repeated applications and penetration of lesions is difficult so failures are common.

Intralesional immune 'stimulants' such as mycobacterial cell wall extracts or Bacillus Calmette Guerin (BCG) can be effective in some nodular or fibroblastic forms but the prognosis is far worse than the corresponding results from treatment of periocular sarcoids of the same type.[57] Repeated injections are invariably required and each one carries the risks of causing anaphylaxis. The possibility of anaphylactic reactions can be reduced (but probably not eliminated) by premedication with flunixin meglumine and dexamethasone intravenously some 15–30 minutes before the procedure is carried out. Ensuring true intralesional injection can also reduce the risks.

Prognosis

The prognosis for any sarcoid is guarded. Recurrences are common and new lesions also develop in many sites. While the malignant form carries a very poor prognosis, it is less common than is the case with the other variants. The prognosis for oral or facial sarcoids is also related to the loss of effective use as a result of interference with tack. Lesions that occur at the angle of the mouth or in the cheeks can adversely affect the use of bits and harness. Repeated trauma from harness results in continued exacerbation and so the tumor and the horse become increasingly difficult to manage.

Melanoma

A tumor of melanocytes occurring in the skin and in other organs (including the mouth). Most melanomas are encountered in gray horses – indeed most gray horses over 5–8 years old will have melanoma at some site. Rarely other colors are also affected. The lips are a relatively common site but the gingivae and tongue may be affected.

There is a strong tendency for melanoma to develop in the parotid salivary glands and associated lymph nodes. Tumor development in these sites is usually obvious on clinical inspection. The large majority of melanomas are benign but some have an aggressive appearance and high growth rate. It is not easy to characterize the degree of malignancy in melanomas without resort to biopsy, and even then histology may not always provide a firm prognosis.

Clinical features

Oral melanomas are usually benign but can grow slowly and reach considerable size, even with histologically benign variants. Surprisingly, tumors on the lips (Fig. 12.7A) and gums are often only noticed when they are large. Their rate of growth is usually low and they have no other systemic effects (unrelated melanomas may, however, develop simultaneously in other organs). Extensive lesions can develop in the parotid and pharyngeal lymph nodes and may extend into the parotid salivary gland either directly or by contiguous spread (Fig. 12.7B). In spite of the large size of some of these lesions the clinical effects are usually minimal and relate simply to their space-occupying nature.

Differential diagnosis

Equine sarcoid and mast cell tumors should be considered in the differential diagnosis of lesions that develop in haired skin adjacent to the lips.

Diagnostic confirmation

The diagnosis of melanoma is easy to confirm from clinical features and fine-needle aspiration or hollow-needle biopsy.



Figure 12.7. (A) A large melanoma in the lip of an aged gray horse. The lesions expanded slowly but the horse remained symptom-free for years in spite of superficial ulceration. (B) Salivary gland melanoma.

Pathology

Melanomas are bulging, well-circumscribed, gray-black masses that compress adjacent soft tissues, sometimes producing ulceration of the compromised surfaces. Cut surfaces of the tumor are usually glistening, firm and uniformly black. Occasional lesions may be less homogeneous, less pigmented or even amelanotic.

Most equine melanomas are characterized histologically by the presence of myriad round-to-oval cells with plentiful densely pigmented cytoplasm. Two distinct types of cells constitute the tumor:

- melanin-producing cells (melanocytes)
- macrophages that contain phagocytosed melanin (melanophages).

Pigmented tumor cells may extend into adjacent soft tissues but this is not a reliable histologic criterion of malignant behavior. Most tumor sections have to be bleached so that the underlying cytologic features can be assessed. Nuclei are usually solitary with a large nucleolus and coarse nuclear chromatin. Mitoses are usually sparse.

There are occasional melanomas with clinical features of malignancy (invasion and metastatic spread); these have corresponding histologic features of anisocytosis and anisokaryosis, with plentiful mitoses and variable cytoplasmic pigmentation. The diagnosis of such less well-differentiated melanoma may be clarified by the use of immunostaining against cell markers such as Melan A and S100.[58]

Treatment

Surgical excision is feasible and usually effective but many melanomas are left alone without any significant problem. Prolonged daily oral administration of cimetidine at doses of up to 7.5 mg/kg bodyweight has been suggested as being effective but the results are not convincing in many cases.[59,60] Treatment of single or few oral tumors on their own probably does not warrant this approach.

Prognosis

The prognosis is usually relatively favorable as the majority have no metastatic tendency but occasional tumors are very aggressive with extensive metastatic spread and serious secondary effects.

Oral papilloma

Viral papilloma is a relatively common occurrence on the skin of the mouth and lips and in some cases they can extend into the oral cavity. Host-specific equine papilloma viruses cause them and most often affect young horses in their first year or two at grass. Less commonly they affect older horses and particularly those that have an immunocompromising disorder such as equine Cushing's disease.

Clinical features

Typically, papilloma appear as single or multiple, discrete or coalescing, verrucose, gray-pink papules on the mucosa of the mouth (Fig. 12.8). They seldom ulcerate unless they are traumatized. Most cases present with a few or many single or coalescent papillomata in or on the skin of the mouth and face. A few cases have lesions restricted to the oral mucosa and it is not known if these reflect a different manifestation of the same infection.

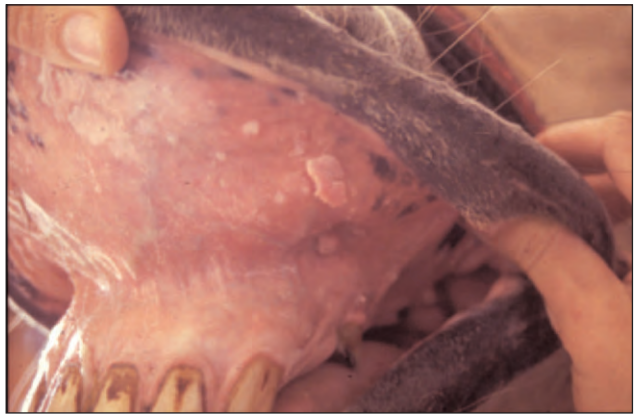


Figure 12.8. Oral papilloma lesions in an aged horse (slide courtesy of Dr Marianne Sloet).

Differential diagnosis

Viral papilloma can be mistaken for some forms of the equine sarcoid, but as the latter seldom occur in a verrucose form within the mouth, differentiation should be simple.

Diagnosis

The diagnosis is usually simply based on epidemiology and clinical appearance, but they can closely resemble the equine sarcoid. Biopsy is characteristic and most lesions resolve with age, although they may be very persistent in older horses.

Pathology

Papillomas are characteristic exophytic verrucose lesions that may be superficially ulcerated and inflamed. Histologic examination reveals filiform fronds of hyperplastic epithelium on fibrovascular cores that often contain plentiful lymphocytes and plasma cells. Intranuclear inclusions may be plentiful or sparse.

Treatment

Most papillomas resolve spontaneously over some months but individual lesions may persist, often for many years. Therapeutic measures have included autogenous vaccines prepared from wart tissues and various topical chemicals such as podophyllin. Individual lesions that prove troublesome can safely be subjected to surgical excision or cryosurgery.

Prognosis

The prognosis is excellent. The majority resolve spontaneously and those that do not have no apparent harmful effect on the horse.



Figure 12.9. Benign gingival hyperplasia (epulis) in an aged horse. The lesions were noted incidentally and were symptom-free.

Epulis

These tumor-like masses develop from the fibrous tissue of the gingiva. The term epulis is a clinical description of a smooth gingival nodule and can encompass different types of lesion such as granuloma or neoplasia. In horses they are much less common than in some other species such as the dog and cat and seldom reach significant proportions (Fig. 12.9). They can easily be overlooked but are benign and can safely be removed surgically. In many cases they arise from chronic local irritation due to persistent infection or the presence of dental tartar.

Clinical features

Usually these appear as a benign expansion of the gingival epithelium resulting in thickening and prominence of the gums particularly evident at the gingival margin where it may be 'piled up' (Fig. 12.9).

Differential diagnosis

Viral papilloma, sarcoid and squamous cell carcinoma are the main differential diagnoses; all are easily identified histologically after surgical removal.

Diagnosis

Refinement of the clinical diagnosis depends on histologic examination of resected epulides. The main differentials are easily identified histologically after surgical removal or biopsy.

Pathology

There is poor histologic characterization of the epulides, probably because they are easily recognized and are seldom treated: few are examined histologically.

Treatment

Removal of the causative factors usually results in complete resolution. Many cases will resolve spontaneously after any dental tartar or damaged dental or soft-tissue structures are removed.

Prognosis

The prognosis is excellent. There are no reports that they are precursors to more malignant tumors such as squamous cell carcinoma but one case developed a carcinoma some years after a benign epulis was removed from the base of a canine tooth (DCK, unpublished observation). It is probably unwise to draw any causative inference from this single case.

Ossifying fibroma

Definition

A poorly defined proliferative fibro-osseous, tumor-like, solitary lesion, which typically develops in the rostral mandible.

Occurrence

Most cases are reported in horses less than 1 year of age. They may, however, not be observed until the animal is handled at 12–14 months of age by which time there may be significant distortion and ulceration of the buccal mucosa.[61]

Clinical features

The majority of these tumors occur unilaterally in the rostral mandible. Lesions may rarely develop in the maxilla and in very rare cases can be bilateral. They often reach considerable size and, initially at least, are covered by a domed, smooth/normal oral mucosa. Later some ulceration is common (Fig. 12.10). Gross distortion of the lip and the associated teeth are likely. The lesion can predispose the mandible to pathologic fracture. The expanding lesion causes loosening of the teeth and consequent dysphagia. Although the lesion is usually obvious they are often only identified late probably because there is little call to examine the mouth of a very young horse.

Diagnostic confirmation

Radiographically the dense tissue is obvious with only a few showing the calcification more commonly encountered in

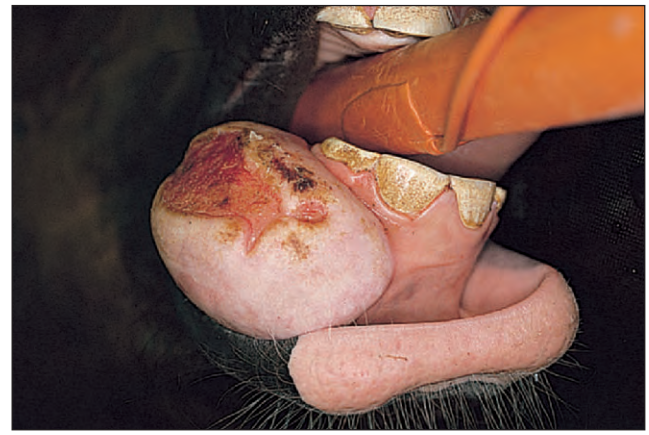


Figure 12.10. Juvenile ossifying fibroma in a 14-month-old thoroughbred gelding. Note the generally smooth outline of the firm swelling. The superficial ulceration only developed late in the condition.

other species. The more ulcerated and secondarily infected lesions may resemble other soft-tissue tumors.

Pathology

They may develop as alterations in the growth characteristics of the periodontal membrane or the developing teeth. The masses are reported to arise from a sessile base on the surface of the bone and expand to replace and displace normal structures with dense fibrous or fibro-ossous tissue. The lesion has a dense, tough, well-circumscribed appearance; it may be extensively mineralized and difficult to cut. There is a characteristically abrupt histologic transition from fibroblastic stroma to osteoblasts, which form spicules of osteoid. The dense gritty nature of the mass sometimes makes biopsy difficult.

Treatment

Surgical excision is curative provided that sufficient attention is paid to the true extent of abnormal tissue. Extensive surgical debulking followed by cobalt-60 teletherapy radiation has been successful. Radiation using cobalt-60 teletherapy alone in a standing sedated horse has also been used successfully.[62] Hemimandibulectomy or hemimaxillectomy is also an effective option.[63] Limited disabilities and acceptable cosmetic effects have been reported. Cases subjected to this surgery recovered well and were able to lead active normal lives. However, if extensive excision is required it may leave an unacceptable cosmetic and functional deficit.

Prognosis

Regrowth of tumor is common because of the difficulty of identifying the margins of the abnormal tissue. All resected tissue should therefore be submitted for histologic examination with particular attention to the surgical margins that need to be identified and marked by the surgeon.

Myxomatous tumors of the jaw

Definition

These are very rare tumors derived from embryonal connective tissue. The tumors are identified by their characteristic histologic appearance.

Occurrence

In the few reported cases, older or mature horses appear to be more often affected. The molar dental arches of the maxilla are the commonest site.

Clinical features

These tumors occur particularly in the maxillary region (and more rarely the mandibular region) and are characteristically destructive (Fig. 12.11). The combined destruction and proliferation of tumor tissue creates obvious distortion of the maxilla with secondary nasal and sinus occlusion/obstruction. Loosening of teeth and infection of alveolar bone may result. Radiographically there is an aggressive lytic appearance with a diffuse mixture of bone and soft tissues often in a partially loculated form. The cardinal radiographic signs of the more malignant forms, however, are the combined destruction of normal bone and bizarre irregular new bone formation in random arrangement.

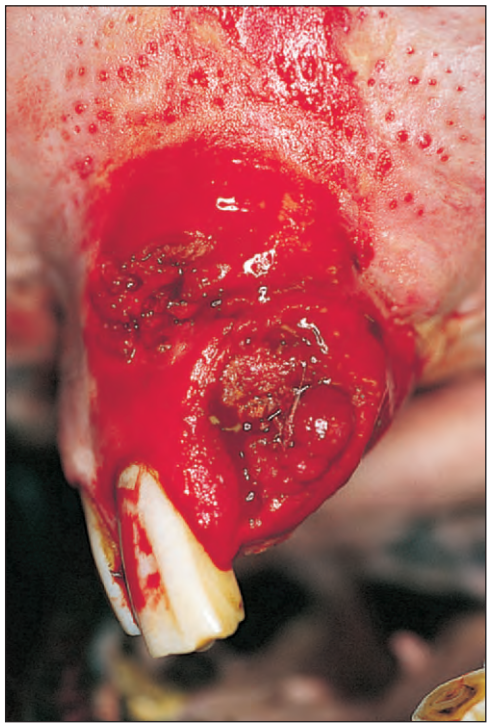


Figure 12.11. Myxomatous tumor of the premaxilla in a 14-year-old hunter gelding. Note the extensive destruction that is very similar to squamous cell carcinoma. The diagnosis can be confirmed relatively easily by biopsy.

Diagnostic confirmation

Biopsy is essential to differentiate the lesion from squamous cell carcinoma.

Pathology

This group of rare tumors includes a spectrum that extends from benign myxoma to malignant myxosarcoma. The tumors have a soft gelatinous gross appearance, and may be highly infiltrative with a tendency to metastasize. Cut surfaces of the tumor may be lobulated and slimy. Histologically the lesion contains characteristic stellate cells with abundant, amorphous extracellular matrix.

Severely ulcerated juvenile ossifying fibroma can resemble these tumors but are usually slow growing and expansive rather than destructive; ossifying fibroma has a characteristically different anatomic site, usually involving the rostral mandible.

Treatment

Treatment options are very limited – the margins of the tumor and the anatomic site make surgical excision virtually

impossible. There are no definitive reports of metastatic spread of the malignant forms of these tumors but this may reflect the short clinical duration, which inevitably results in euthanasia before secondary tumors develop elsewhere.

Prognosis

These tumors are very unpredictable in behavior: some are slow growing and relatively benign, others are highly aggressive and so carry a hopeless prognosis.

Oral hemangiosarcoma

This is a malignant neoplasm of endothelial cells that can arise in any part of the body. They are reported to metastasize early and so tumors in any locality may be primary or secondary. There are few reports of this tumor in the mouth.[64,65] Aged horses are more likely to develop them and there may be concurrent tumors at other sites such as around the eye (DCK unpublished observation).

Clinical features

A red or purple ulcerated mass in the oral mucosa (either on the sides of the tongue or on the gingiva is typical). The mass is likely to be slow growing and being subject to repeated trauma there may be some oral bleeding. The lesions may be identified incidentally during a clinical examination, but where periocular lesions occur the clinician should carefully examine all the visible mucous membranes, including the mouth, for other evidence of the tumor.

Differential diagnosis

Foreign-body reactions and ulceration arising from the attachments of *Gastrophilus* sp. may be similar.

Diagnosis

Biopsy is the only definitive way of establishing the diagnosis.

Pathology

Histologic features are of solid soft-tissue sarcoma with marked anisocytosis, anisokaryosis and myriad mitoses. There is variable formation of large or small vascular channels lined with neoplastic endothelial cells. In histologically equivocal cases immunohistochemical staining for Factor VIII-related antigen may be helpful in identifying neoplastic endothelium. Tissue margins of resected lesions should be assessed carefully to ensure that excision is complete, although the latter may be difficult for tumors of the gingiva.

Treatment

Surgical excision may be effective but there are considerable technical difficulties and risks of incomplete excision.

Prognosis

The presence of more than one lesion may not necessarily indicate that the tumor has spread metastastically, but hemangiosarcoma is a malignant tumor of horses and so this possibility must be considered.

Salivary adenocarcinoma

Salivary gland neoplasia is uncommon in horses. For example, in one study of 687 necropsies and 635 biopsies only three salivary tumors were described; two were carcinomas and one was a cystadenoma.[4] A review of 1148 equine tumors included only two salivary adenocarcinomas.[66] Six of 14 equine salivary gland tumors were adenocarcinomas; where recorded, the sites of involvement were parotid gland (three cases) and mandibular glands (one case). The age range was 7–18 years.[67]

Their recognition is important because they are reported to be highly malignant (possibly up to 33 per cent will develop pulmonary secondary tumors).

Clinical features

Recognition is difficult but clinically the affected gland becomes enlarged. Palpation is uncomfortable but not painful. Only one gland is involved and so the enlargement is asymmetrical, which differentiates it from the more usual benign parotid salivary gland swelling commonly seen in grazing horses.

Differential diagnosis

Benign swelling of parotid salivary glands occurs frequently in grazing horses. This idiopathic condition is invariably transient and self-resolving. Obstruction of the salivary ducts by sialoliths (usually in the parotid gland) occurs within the gland substance or in the more rostral part of the duct. This results in a solid obstructive lesion with generalized glandular swelling. This remains hot and painful for some days before it subsides naturally as atrophy of the gland follows.

Diagnosis

Diagnosis is reliant upon biopsy but so few cases have been described that a characteristic histologic appearance is not established. Post mortem histologic features are described.[15]

Treatment

Surgical excision is theoretically possible if diagnosis is made sufficiently early but this is unlikely in most cases.

Prognosis

The high malignancy of most salivary gland carcinomas makes the prognosis very poor.

Disorders of the jaws and teeth resembling neoplasms

Mandibular aneurysmal bone cyst

Although bone cysts are relatively common in long bones and in particular in their epiphyseal regions, mandibular

cysts are very rare. In man, they probably arise from circulatory disturbances within the bone structure[68] or traumatic alteration of the blood supply to a small area of bone.[69] While such structures might be expected in young horses they may occur in older horses, probably as a result of trauma.[10] There is no evidence to suggest that these lesions are truly neoplastic but diagnosis based on radiographic examination alone is probably unwise. While there are no reports of concurrent neoplasia in the horse, complexes of tumors and cysts are reported in other species.

Clinical features

These present as a sterile, firm, expanding swellings on the ventral mandible, resulting from progressive destruction of cortical bone accompanied by new reactive periosteal bone. Aspiration reveals small volumes of yellow or red-orange fluid. The rate of expansion may be sufficient to cause disruption and bleeding into the surrounding tissues and may easily suggest a neoplastic lesion.

Differential diagnosis

Ossifying fibroma and destructive ameloblastoma are the main differentials. Paranasal sinus cysts have a similar radiographic appearance.

Diagnosis

The radiographic appearance reveals a complex, loculated cyst-like structure containing bone fragments, cartilage and soft tissue usually with a fine rim of thin bone around the periphery. The structure closely resembles the complex of paranasal sinus cysts that may also arise congenitally or develop at later ages.

Pathology

Histologically the cystic lesion contains bone fragments, granular debris, siderophages, multinucleated giant cells and fibrovascular tissue with areas of organizing and free blood clot. Histologic examination of curetted fragments of cyst contents may often correlate poorly with the radiographic appearance.

Treatment

The only effective treatment is deep and aggressive curettage of all abnormal tissue. The defect may be filled with cancellous bone grafts collected from a remote site. Repair may be slow and the site may be cosmetically compromised but many cases can be cured effectively.

Prognosis

The prognosis is good following surgical ablation but there may be some anatomic distortion and functional disability.

Cystic lesions of bone

Cystic lesions of the jaws present as bony swellings, and may affect mastication. Radiographically the cysts are rounded with a smooth bony lining and radiolucent center. The generic term odontogenic cyst is appropriate for these non-inflammatory cysts lined by epithelium.[20] Radicular cysts occur adjacent to tooth roots. They are reactive lesions associated with oral inflammatory disease. Histologically they are lined by stratified squamous epithelium with associated inflammatory cells.

Fibrous dysplasia

Fibrous dysplasia of the bones of the skull has been reported in man.[70] A clinically similar condition is recognized in horses as a smooth contoured bone deformity arising from loss of bone structure with extensive formation of fibro-osseous matrix (Fig. 12.12). The lesion is probably not a true neoplasm and its major effects in the jaw/face region are due to the expansive space-occupying nature of the slowly expanding mass. The changes are easily recognized histologically but may be confused with neoplastic lesions both radiographically and clinically. Suspicious masses should be subjected to the full range of diagnostic tests including radiography, gamma scintigraphy, biopsy and, where feasible, computed tomography.

Pathology

Grossly normal bone is replaced by dense gritty tissue; it may be surrounded by reactive bone. Histologic features include fibrous dysplasia and the presence of 'naked' spicules of woven bone in a dense fibrous stroma. The bone appositional surfaces lack recognizable osteoblasts.



Figure 12.12. A radiograph of fibrous dysplasia of the rostral portion of the mandible of a young horse.

Other tumors that may affect the mouth and jaws

These tumors include those arising by metastatic spread from remote organs and those invading the oral cavity from neighboring areas such as the nasal cavity and paranasal sinuses. Tumors remote from the mouth and oral structures may have a serious influence on the mouth either through metastatic spread (such as in hemangiosarcoma and lymphosarcoma). Secondary involvement of the mouth is not a frequent feature of even highly malignant tumors and in any case the oral signs may be trivial compared to the other systemic involvements. The mouth may be secondarily diseased through systemic effects from functional tumors such as the pituitary adenoma-related Cushing's disease and renal tumors.

Lymphosarcoma

Multicentric (generalized) and cutaneous histiocytic lymphosarcoma may have oral manifestations (Fig. 12.13). Usually the clinical appearance is of ill-defined nodular lesions of variable size embedded in and below the mucosal surface, which probably reflect the involvement of the normally diffuse lymphoid tissue in the nasopharynx. The gingival mucosa seems to be most often affected. A similar nodular appearance is often present in the pharynx and in the sublingual tonsillar tissue where they may have some



Figure 12.13. (A) Multiple discreet dermal nodules in a pony with histiocytic lymphosarcoma. (B) Cutaneous histiocytic lymphosarcoma with involvement of the gingival tissues. Ill-defined swellings of the gingiva were due to aggregations of abnormal lymphocytes. These lesions might easily be overlooked but are a regular finding in these cases. (C) Pharyngeal nodules that can sometimes be identified endoscopically. These lesions were symptom-free but larger ones can cause dysphagia. (D) Photomicrograph of the tongue from an aged horse with oral lymphosarcoma, showing epithelium and diffuse subepithelial accumulation of neoplastic lymphoid cells. (E) Lymphosarcoma of the tongue showing neoplastic cells around and between muscle fibers.

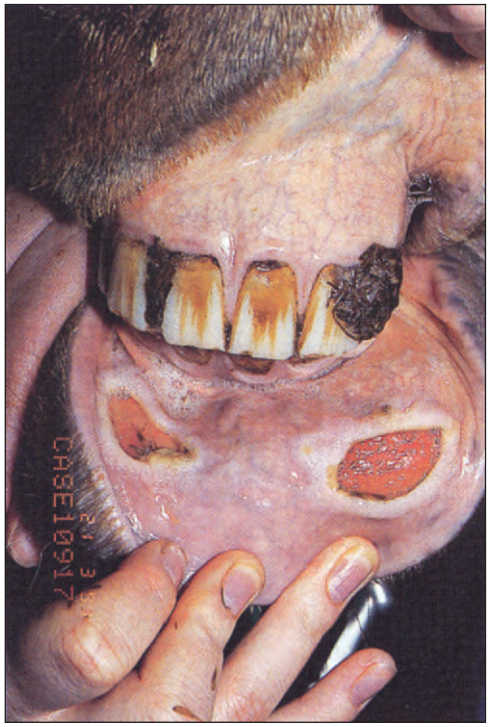


Figure 12.14. Pituitary adenoma/Cushing's disease. Oral ulceration showing a singular lack of healing is a common sign. Other dental and oral lesions may be present also.

effect on swallowing. The lesions may ulcerate or become infected. Simultaneous submandibular lymphadenopathy and other secondary effects such as anemia, lethargy and weight loss may signify serious systemic involvement.

Pituitary adenoma

Tumors of the pituitary par intermedia are responsible for secondary oral disease such as extensive non-healing ulcers (Fig. 12.14) and dental and paradontal infections. The tumors are common in older horses (the large majority are over 14–17 years). Extensive dental and alveolar disease (including periodontal disease and periapical sepsis) develop commonly. This, with advancing, age-related natural degeneration and the short residual crown of older horses predisposes the affected animals to secondary sinusitis and there may be tertiary consequences from this, including early shedding of teeth, oronasal fistulae, oral ulceration and anemia.

Note

Paraneoplastic changes in the mouth such as hypercalcemia and oral ulceration can develop as a result of neoplastic disease in other tissues (usually myeloproliferative neoplasia).[71] It is important to remember that the mouth may be one of the early sites where clinical evidence of neoplastic disease, such as anemia, icterus and azotemia, may be manifest. The lesions seen in the mouth may not be readily attributable to neoplastic disease either at a local or remote site.

REFERENCES

1. Jackson C (1936) The incidence and pathology of tumors of domestic animals in South Africa: A study of the Onderstepoort collection of neoplasms with special reference to their histopathology. *Onderstepoort Journal of Veterinary Science and Animal Industries,* 6, 1–460.
2. Hagdoost IS and Zakarian B (1985) Neoplasms of Equidae in Iran. *Equine Veterinary Journal,* 17, 237–239.
3. Pirie RS and Tremaine WH (1997) Neoplasia of the mouth and surrounding structures. In: *Current Therapy in Equine Medicine,* ed. NE Robinson. WB Saunders, Philadelphia, pp 153–155.
4. Sundberg JP, Burnstein T, Page EH, Kirkham WW and Robinson FR (1977) Neoplasms of equidae. *Journal of the American Veterinary Medical Association,* 170, 150–152.
5. Smith P (1996) *Large Animal Internal Medicine*. Mosby, Philadelphia, p 696.
6. Baker GJ (1999) Oral examination and diagnosis: Management of Oral Diseases. In: *Veterinary Dentistry,* ed. CE Harvey. WB Saunders, Philadelphia, pp. 217–220.
7. Richardson DW, Evans LH and Tulleners EP (1991) Rostral mandibulectomy in five horses. *Journal of the American Veterinary Medical Association,* 199, 1179–1182.
8. Purdy CM (1985) Mandibular aneurysmal bone cyst in a horse. *Equine Practice,* 7, 22–24.
9. Verstraete FJM and Ligthelm AJ (1988) Excessive granulation tissue of periodontal origin in a horse. *Equine Veterinary Journal,* 20, 380–382.
10. Lamb CR and Schelling SH (1989) Congenital aneurysmal bone cyst in the mandible of a foal. *Equine Veterinary Journal,* 21, 130–132.
11. Gibbs C (1974) The equine skull; its radiographic investigation. *Journal of the American Veterinary Radiology Society,* 15, 70–78.
12. Wyn-Jones G (1985) Interpreting radiographs 6: Radiology of the equine head (part 2). *Equine Veterinary Journal,* 17, 417–425.
13. McIlwraith CW (1984) Equine digestive system. In: *The Practice of Large Animal Surgery,* ed. PB Jennings. WB Saunders, Philadelphia, p. 579.
14. Moulton JE (1990) *Tumors in Domestic Animals* (3rd edn). University of California Press, Berkeley, pp. 167–168.
15. Head KW (1990) Tumors of the alimentary tract. In: *Tumors in Domestic Animals* (3rd edn), ed. JE Moulton. University of California Press, Berkeley, pp. 347–374.
16. Baker JR and Leyland A (1975) Histological survey of tumors of the horse, with particular reference to those of the skin. *Veterinary Record,* 96, 419–422.
17. Cotchin E (1977) A general survey of tumors of the horse. *Equine Veterinary Journal,* 9, 16–21.
18. Gorlin RJ, Meskin LH and Brodey R (1963) Odontogenic tumors in man and animals; Pathological classification and clinical behavior – a review. *Annals of the New York Academy of Science,* 108, 722–771.
19. Turrel JM (1995) Oncology. In: *The Horse: Diseases and Clinical Management,* eds. CN Kobluk, TR Ames and RJ Geor. WB Saunders, Philadelphia, pp. 1128–1130.
20. Dubielzig RR (2002) Tumors of the Alimentary Tract. In: *Tumors in Domestic Animals* (4th edn), ed. DJ Meuten. Iowa State Press, Ames, Iowa, pp. 402–410.
21. Wahl P (1936) Adamantinoma polycysticum ossificans am Unterkiefer eines Pferdes. *Deutsche Tierartztliche Wochenschrift,* 46, 113–117.
22. Peter CP, Myers VS and Ramsey FK (1968) Ameloblastomic odontoma in a pony. *American Journal of Veterinary Research,* 29, 1495.
23. Vaughan JT and Bartels JE (1968) Equine mandibular adamantinoma. *Journal of the American Veterinary Medical Association,* 153, 454–457.

24. Hanselka DW, Roberts RE and Thompson RB (1974) Adamantinoma of the equine mandible. *Veterinary Medicine for the Small Animal Clinician,* 69, 157–160.
25. Jones SL and Brumbaugh GW (1991) Ameloblastoma in mandible of horse. What is your diagnosis? *Journal of the American Veterinary Medical Association,* 199, 630–631.
26. Summers PM, Wells KE and Adkins KF (1979) Ossifying ameloblastoma in a horse. *Australian Veterinary Journal,* 55, 498–500.
27. Lingard DR and Crawford TB (1970) Congenital ameloblastoma in a foal. *American Journal of Veterinary Research,* 31, 801.
28. Roberts MC, Groenendyk S and Kelly WR (1978) Ameloblastomic odontoma in a foal. *Equine Veterinary Journal,* 10, 91–93.
29. French DA, Fretz PB and Davis GD (1984) Mandibular adamantinoma in a horse; radical surgical treatment. *Veterinary Surgery,* 13, 165–171.
30. Dillehay DL and Schoeb TR (1986) Complex odontoma in a horse. *Veterinary Pathology,* 23, 341–342.
31. Dubielzig RR, Beck KA, Levine S and Wilson JW (1986) Complex odontoma in a stallion. *Veterinary Pathology,* 23, 633–635.
32. Mason BJE (1974) Temporal teratomata in the horse. *Veterinary Record,* 95, 226–228.
33. Fessler JF (1988) Heterotopic polyodontia in horses: nine cases 1969–1986. *Journal of the American Veterinary Medical Association,* 192, 535–538.
34. Lindshaw WA, and Bech KA (1986) Temporal teratoma in a horse. *Compendium of Continuing Education for Practicing Veterinarians*, 8, 168–171.
35. Kramer IRH, Pindborg JJ and Shear M (1993) *Histologic typing of odontogenic tumors* (2nd edn). Springer-Verlag, Berlin, pp. 151–156.
36. Pindborg JJ, Kramer IRH and Torlini H (1971) *Histologic typing of odontogenic tumors, jaw cysts and allied lesions.* World Health Organisation, Geneva, Switzerland. pp. 145–154.
37. Livesey MA and Wilkie IW (1986) Focal and multifocal osteosarcoma in two foals. *Equine Veterinary Journal,* 18, 407–410.
38. Slayter MV, Boosinger TR, Pool RR, Dammrich K, Misdorp W and Larsen S (1994) *Histological Classification of Bone and Joint Tumours of Domestic Animals,* 2nd series, Volume 1. Armed Forces Institute of Pathology, Washington DC, pp. 9–45.
39. Thorp F and Graham R (1934) A large osteosarcoma of the mandible. *Journal of the American Veterinary Medical Association,* 84, 118–119.
40. Jacobson SA (1969) Parosteal osteoma (juxtacortical osteogenic sarcoma) in animals. *American Journal of Pathology,* 58, 85a.
41. Livesey MA and Wilkie IW (1986) Focal and multifocal osteosarcoma in two foals. *Equine Veterinary Journal,* 18, 410–412.
42. Gorlin RJ, Meskin LH and Brodey RS (1963). Odontogenic tumors in man and animals; Pathological classification and clinical behavior – a review. *Annals of the New York Academy of Science,* 108, 722–771.
43. Strafuss AC (1976) Squamous cell carcinoma in horses. *Journal of the American Veterinary Medical Association,* 168, 61–62.
44. Howie F, Munroe G, Thompson H and Murphy D (1992) Palatine squamous cell carcinoma involving the maxillary sinus in two horses. *Equine Veterinary Education,* 4, 3–7.
45. Barker IK, van Dreumel AA and Palmer N (1993) In: *Pathology of Domestic Animals* (4th edn), Volume 2, eds. KVF Jubb, PC Kennedy and N Palmer. Academic Press, New York, p. 27.
46. Knottenbelt DC and Pascoe RR (1994) *Diseases and Disorders of the Horse*. Wolfe London, p. 303.
47. Henson WR (1936) Carcinoma of the tongue in a horse. *Journal of the American Veterinary Medical Association,* 94, 124.
48. Leyland A and Baker JR (1975) Lesions of the nasal cavity and paranasal sinuses of the horse causing dyspnoea. *British Veterinary Journal,* 131, 339–346.
49. Orsini JA, Nunamaker DM, Jones CJ and Acland HM (1991) Excision of oral squamous cell carcinoma in a horse. *Veterinary Surgery,* 20, 264–266.
50. Theon AP (1997) Cisplatin treatment for cutaneous tumors. In: *Current Therapy in Equine Medicine* (4th edn), ed. NE Robinson. WB Saunders, Philadelphia, pp. 372–377.
51. Paterson S (1997) Treatment of superficial ulcerative squamous cell carcinoma in three horses with topical 5-fluorouracil. *Veterinary Record,* 141, 626–628.
52. Pulley LT and Stannard AA (1990) Tumors of the skin and soft tissues. In: *Tumors in Domestic Animals* (3rd edn), ed. JE Moulton. University of California Press, Berkeley, pp. 23–87.
53. Knottenbelt DC, Edwards SER and Daniel EA (1995) The diagnosis and treatment of the equine sarcoid. *In Practice,* 17, 123–129.
54. Pascoe RR and Knottenbelt DC (1999) Neoplastic conditions. In: *Manual of Equine Dermatology.* WB Saunders, Philadelphia, pp. 244–252.
55. Brostrom H (1995) Equine sarcoids: a clinical, epidemiological and immunological study. PhD Thesis, University of Uppsala, Sweden, pp. 9–70.
56. Theon AP (1997) Cisplatin Treatment for Cutaneous tumors. In: *Current Therapy in Equine Medicine* (4th edn), ed. NE Robinson. WB Saunders, Philadelphia, pp. 372–377.
57. Knottenbelt DC and Kelly DF (2000) The diagnosis and treatment of periorbital sarcoid in the horse: 445 cases from 1974 to 1999. *Veterinary Ophthalmology*, 3(2), 169–191.
58. Kelly DF (2003) Diagnostic problems in nasopharyngeal amelanotic melanomas. *Equine Veterinary Education,* 15, 25–26.
59. Goetz TE, Ogilvie GK, Keegan KG and Johnson PJ (1990) Cimetidine for the treatment of melanoma in 3 horses. *Journal of the American Veterinary Medical Association,* 196, 449–452.
60. Goetz TE and Long MT (1993) Treatment of melanoma in horses. *Compendium of Continuing Education for the Practicing Veterinarian*, 15, 608–610.
61. Morse CC, Saik JE and Richardson DW (1988) Equine juvenile mandibular ossifying fibroma. *Veterinary Pathology,* 25, 415–421.
62. Robbins SC, Arighi M and Ottewell G (1996) The use of megavoltage radiation to treat juvenile mandibular ossifying fibroma in a horse. *Canadian Veterinary Journal,* 37, 683–684.
63. Kawcak CE, Stashak TS and Norrdin RW (1996) Treatment of ossifying fibroma in a horse by hemimaxillectomy. *Equine Practice,* 18, 22–25.
64. Fry FL, Knight HD and Brown S (1983) Hemangiosarcoma in a horse. *Journal of the American Veterinary Medical Association,* 182, 287–289.
65. Sweigard KD and Hattell AL (1993) Oral hemangiosarcoma in a horse. *Equine Practice,* 15, 10–13.
66. Stackhouse LL, Moore JJ and Hylton WE (1978) Salivary gland adenocarcinoma in a mare. *Journal of the American Veterinary Medical Association,* 172, 271–273.
67. Head KW (1995) Salivary gland disease in domestic animals. In: *Salivary Glands. Color Atlas and Text,* eds. P de Burgh, JE Norman and M McGurk. Mosby-Wolfe, Philadelphia, pp. 367–389.
68. Biesecker JL, Marcove RC, Huvos AG and Mike V (1970) Aneurysmal bone cysts; a clinicopathological study of 66 cases. *Cancer,* 26, 616–625.
69. Dabezies EJ, D'Ambrosia RD, Chuinard RG and Ferguson AB (1982) Aneurysmal bone cyst after fracture. *Journal of Bone and Joint Surgery,* 64, 617–621.
70. Jacobson SA (1971) *The Comparative Pathology of the Tumors of Bone*. Charles C Thomas, Springfield, Illinois.
71. Williams MA, Dowling PM, Angarano DW *et al.* (1995) Paraneoplastic bullous stomatitis in a horse. *Journal of the American Veterinary Medical Association,* 207, 331–334.

13 Dental and Oral Examination

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Introduction

Oral and dental diseases are common occurrences in horses as evidenced by the results of incidence studies of dental disease carried out on abattoir specimens.[1–3] Signs of dental disease are often not apparent to the owner until the disease is well advanced.[4] Casual oral or dental examination as part of a complete physical examination is not sufficient to detect most oral or dental problems. This has been demonstrated by the reported high incidence and the comparatively low clinical diagnosis of dental disease.[5,6] Clinical signs of dental disease are often not specific and may be reflected in other body systems.[7] An example of this is horses showing signs of lamenesses that are alleviated when dental problems are corrected.[8]

Most equine practitioners consider oral examination as simply parting the lips, casually looking at the incisors and placing a finger in the cheek to feel for points on the first few upper cheek teeth. Only a small percentage of equine dental disease will be detected through this type of examination. A complete oral examination includes observing and feeling both hard and soft tissues for pathology. The hard tissues consist of teeth and osseous structures. The soft tissues consist of the lips, cheeks, tongue, palate, gingiva, oral mucosa and salivary glands.

The basis of modern clinical therapy is diagnosis. This presupposes that disease can be accurately identified and then effectively eliminated by the application of appropriate therapy. It would seem, therefore, that information obtained in making a diagnosis is the foundation for successful treatment. Discipline must be exercised in collecting material for a meaningful diagnosis.

Although a comprehensive history and physical examination of every patient seen while performing routine dental work would be a valuable service to clients, this is not practical in most cases. However, one must establish the presence or history of medical problems that may have an impact on safe delivery of dental care. The minimal dental examination process must be thorough enough to detect abnormalities in their early stages of development. Treatment can therefore be initiated before any pathological process causes deterioration beyond correction. The extent of the examination should evolve and progress depending upon the information obtained in the history and the findings from the minimal examination. The examination process must be performed in a routine fashion to ensure efficiency and quality of the examination. Variations and/or abnormalities detected at the time of the initial examination must be documented. If no notation is made in the record it can be assumed that there was no abnormality at the time of examination. When routine becomes habit, the results are thorough and the time to complete the examination is reduced. A standard dental record form can be an invaluable aid in helping develop good examination habits (Fig. 13.1A,B). Computerized dental records have made information more available for retrieval and case follow-up.[9]

A typical examination starts with a brief history while screening the animal from a distance. The screening process should give the veterinarian a general idea of the type, thriftiness, general use and overall physical condition of the horse. The animal's feed and water sources should be observed, noting the amount and type of feed being consumed. Attention should be directed to the horse's manure to gain an idea of how well feed is being processed and digested. The head is surveyed for shape, symmetry and obvious abnormalities. The head should be palpated for irregularities or tender areas, especially along the upper dental arcades. The lips are parted, incisors inspected and the age is estimated. The oral mucous membranes, interdental space and tongue are evaluated. The range of motion of the jaw is viewed and its grinding sound and vibration are evaluated. The oral cavity is then inspected visually and palpated from the labial edges of the incisor teeth, caudal to the buccal recesses distal to the last molar. Subtle details of the examination process are extremely important. This chapter will attempt to detail the complete dental examination. Special examination considerations and procedures will be outlined for horses of various ages and occupations.

Equipment required for a thorough oral examination

Equipment utilized to examine properly the equine masticatory system is minimal, but certain items are necessary to perform a complete oral examination. The technique for restraint and size of equipment will vary for different ages and sizes of equine patients. Very large (1000 kg or more) draft breeds need restraint with more heavily constructed

EQUINE DENTAL EVALUATION AND MAINTENANCE FORM

(SPACE ABOVE LEFT BLANK FOR ADDRESS OF DVM)
MEMBER AMERICAN ASSOCIATION OF EQUINE PRACTITIONERS

DATE _____ STABLE _____
OWNER _____ TRAINER _____
ADDRESS _____
_____
PHONE _____ PHONE _____

HORSE _____ BREED _____ COLOR _____ AGE _____ SEX _____
PROBLEMS _____

SOFT TISSUE
NORMAL
LIPS
TONGUE
PALATE
GUMS
BARS
CHEEKS

WOLF TEETH
PRESENT
ABSENT
UNERUPTED
REMOVE
ROOT FRAGMENT

INCISORS
NORMAL
TILTED
SMILE
FROWN
STEP
CAPS
ALIGN
TOO LONG U L
SHORTEN
FLOAT
DREMEL
OVERBITE
UNDERBITE
BROKEN TEETH

CANINES
NORMAL
UNERUPTED U L
CUT
BUFF
REMOVE TARTAR
EXTRACT

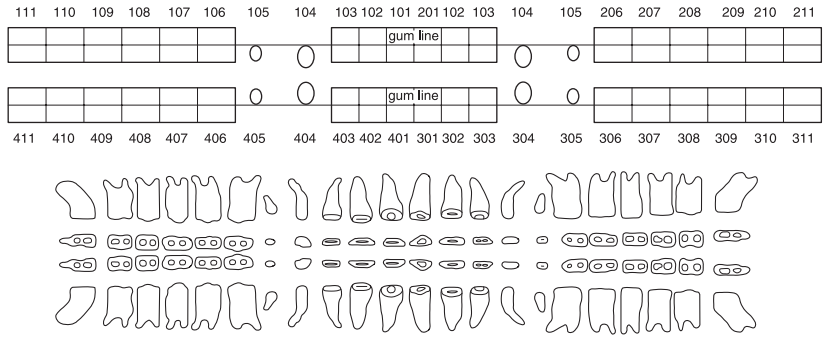
MOLARS
NORMAL
HOOKS F R
HIGH TEETH
WAVE
STEPPED
SHEAR
RAMP
RIMS
SEPARATION
CUPPED OUT
CAPS REMOVED
TABLE ANGLES L____R____
FLOAT
CUT-FLOAT
CUT-FLOAT
CUT-LEVEL
CUT-LEVEL
CUT-LEVEL
CUT-LEVEL
FLOAT
BROKEN TEETH

FEE FEE FEE FEE

SEDATION: DETOMIDINE _____ BUTORPHANOL _____ XYLAZINE _____ ACEPROMAZINE _____

FEE FEE FEE FEE

OCCLUSION RIGHT _____
LEFT _____



TRIP FEE _____ COMMENTS _____

Figure 13.1. (A) and (B) Equine dental evaluation and maintenance form.

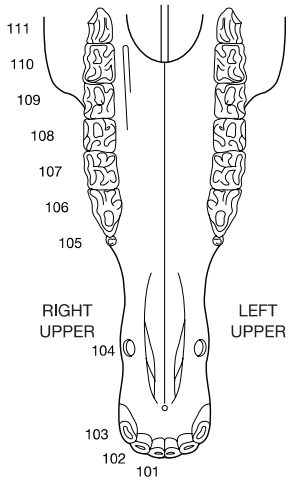
equipment than the typical (500 kg) riding horse. On the other extreme are the small (100 kg) pony and miniature breeds. These horses require downsized equipment (Fig. 13.2). Oral examination and dentistry on small horses may also be aided by walking the horse up on an elevated platform to have the oral cavity at a more comfortable height for the operator's visualization and work plane. Equipment needs include, but are not limited to, a halter and/or lead, a metal-framed dental halter or head stand and a mouth speculum.

An adjustable halter with a nose band large enough to get the horse's mouth opened wide and not place pressure on the cheeks and muzzle is suggested. A short lead shank (1–2 m) with a loop in the end allows the horse to be controlled while the veterinarian works with both hands.[10]

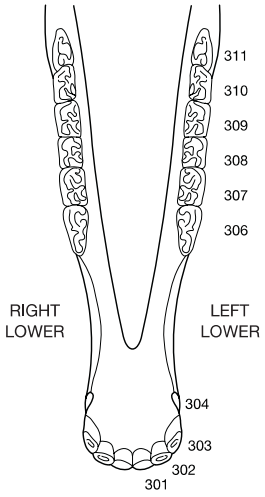
DATE	OWNER	FARM NAME/PHONE		WORK PHONE	
ADDRESS	CITY	STATE	ZIP CODE	HOME PHONE	
HORSE REG. NAME	STABLE NAME	COLOR	BREED	SEX	YR. FOALED

Hair Coat: EX VG G P
Condition Score: _____
Lateral Jaw Excursion: N AB
History: _____

D. Age _____ Reg. Age _____
Feces: Fine Med Coarse
Palpation Response: + –
Soft Tissue _____

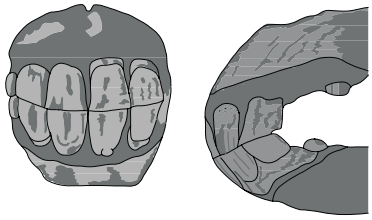


COMMENTS



CANINES

REDUCTION NEEDED? YES NO



INCISOR ALIGNMENT

NORMAL ABNORMAL

INCISOR CORRECTION/RE-ALIGNMENT NEEDED? YES NO

MOLAR TABLES

RIGHT SIDE	LEFT SIDE
Deciduous Teeth (Caps) ____	Deciduous Teeth (Caps) ____
Rostral Hook (PM2) Y N	Rostral Hook (PM2) Y N
Caudal Hook (M3) Y N	Caudal Hook (M3) Y N
Abnormal Occlusion Y N Step/Wave Mouth	Abnormal Occlusion Y N Step/Wave Mouth
Occlusion Correction Needed Y N	Occlusion Correction Needed Y N

CHARGES	Initial Est	Actual Amt Chrg
Farm Call		
Professional Exam		
Sedation/Tranquilization		
Anaesthesia		
Radiography		
Anti-inflammatory		
Antibiotics		
Medication/Treatment Other		
Problem		

RE-EXAM NEEDED
+++++++
DATE

TOTAL AMOUNT DUE
+++++++

All services are strictly cash and must be paid at the time of service.

Figure 13.1, cont'd, (B).



Figure 13.2. Horses come in a variety of sizes. Equipment may need to change scale with the animal.

A metal-framed halter or head stand can be used to restrain the head of a horse in a comfortable position to work in the mouth (Fig. 13.3). A metal-framed halter can withstand pressure placed on the lead rope without the nose band collapsing around the cheeks. The lead can be pulled down or secured like a martingale to hold the head down. The cheek ring can be placed on the top of the halter and a rope suspended from the ceiling to elevate the head of a sedated horse. A cotton rope (1.38 cm diameter, 5 m long) with a quick-release snap works well for either application.



Figure 13.3. A sedated horse being examined with its head suspended in a metal-framed dental halter. A full mouth speculum is in place. A head light is used to give good illumination to the oral cavity.

A mouth speculum is essential for complete evaluation of the equine oral cavity. The two general categories of specula are the gag or wedge type and the full mouth speculum.

There are a number of designs of gag specula. The basic principle is to place a wedge in the mouth between the upper and lower molar arcades to block the mouth open. There are advantages and disadvantages to this type of restraint. The greatest advantages are that the spool or wedge is small and inside the mouth and the device is both lightweight and user safe. It also does not interfere with the incisor teeth, which can be worked on with the speculum in place. Disadvantages of the gag specula include not allowing for good observation or adequate room for digital examination of the oral cavity. Another disadvantage is that a horse chewing on the spool can damage its molar teeth (Fig. 13.4). This usually occurs in the young horse with erupting permanent teeth or in the old horse with short, fragile reserve crowns. These devices can also slip off the molar arcades into the palate and cause damage to the gingiva. Palate lacerations and injury to the palatine artery are also possible. The types of mouth gags available today vary in size, shape and method of retention. Several of the more common ones are: Schoupe mouth gag, Jeffery gag, Landmesser wedge, Bayer mouth wedge, Jupiter spool, and Meiers dental wedge (Fig. 13.5).

The full mouth speculum secures the mouth in the open position by applying pressure on the incisor arcades. These types of specula are, as a rule, larger, heavier and more cumbersome than gags. When using a full mouth speculum, chemical restraint is suggested. With the ability to adjust the amount of opening of the speculum, care must be exercised when using these devices to assure that the horse's mouth is not forced beyond a comfortable limit. The speculum should not be left in the open position for an extended period of time (not more than 15 minutes) without allowing the horse to relax. The advantages of the full mouth speculum are the increased ability to perform visual and digital inspection of the deep recesses of the



Figure 13.4. Radiograph of a fractured upper premolar 3 on a 3-year-old colt. Three weeks prior to presentation, a round spool type mouth gag had been used to hold the mouth open during a dental procedure. It is speculated that the horse fractured this newly erupted tooth while chewing on the gag.



Figure 13.5. Wedge type mouth gags are less apt to damage teeth because the masticatory forces are distributed over several tooth crowns.



Figure 13.7. A stainless steel Haussmann type full mouth speculum with large and small incisor plates and a gum plate. The straight gum plate rests on the palate and is handy for examining horses with severe incisor overjet, or cattle that lack upper incisors.

oral cavity and greater access to perform dental procedures with good access to the inner oral cavity.

The types of full mouth specula available vary in size and shape. The author prefers the McPherson or Haussmann type full mouth speculum with adjustable leather straps (Fig. 13.6). Other full mouth specula are: Arnold's mouth gag, Butler's mouth gag, Guenther mouth speculum, Vernell's mouth gag, McAllen mouth speculum, McClelen mouth speculum, Stubbs screw speculum and Meister speculum. Flat gum plates are available with the Haussmann type speculum that can be used on cattle (Fig. 13.7). This type of gum plate can be placed palatally to the upper incisor table to allow oral examination of a horse with an incisor malocclusion.

A good light source is necessary for proper oral examination. A hand-held light or a head lamp is essential for adequate visualization. A flexible cable fiberoptic light can be used to illuminate the deep recesses of the oral cavity and buccal spaces. Various types of cheek retractors allow better visualization in the buccal spaces. A long-handled mechanic's mirror or fiberoptic scope is useful in visualizing the interproximal spaces. A rigid-handled mirror manufactured by Stubbs is ideal for visualizing the buccal recesses, interproximal spaces and occlusal surfaces of the cheek teeth. The strong rigid handle with mounted light can be used to retract the soft tissues and illuminate hard-to-reach spaces in the oral cavity. Anti-fog treatment on the mirror allows a more consistent and unobstructed view. A laparoscope with a 45°–75°-angled lens can be an invaluable aid in examining between the caudal cheek teeth. A video or still digital camera can be attached to the scope to allow for more relaxed viewing. This process is also a good client education tool (Fig. 13.8).

Dental picks and probes are available in various lengths and blade shapes. The complete oral examination can be greatly aided by the use of long dental picks. These tools are especially useful in the examination of older horses, providing immense help in evaluating pockets between and around teeth that cannot be reached with a digit (Fig. 13.9).



Figure 13.6. A McPherson type full mouth speculum being placed on a horse.

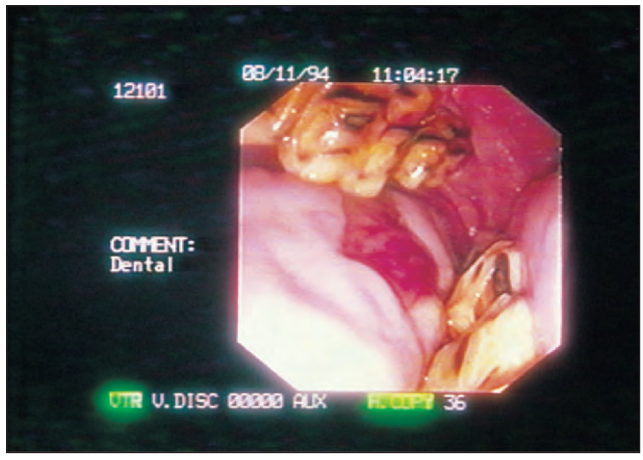


Figure 13.8. Oral (buccal) ulceration resulting from overgrown enamel edges on the caudal cheek teeth. The horse ate slowly and saliva tested positive for blood using a urine dipstick. The lesions were not very obvious when viewed in the conscious horse but endoscopy was usefully employed to establish the nature of the problem.

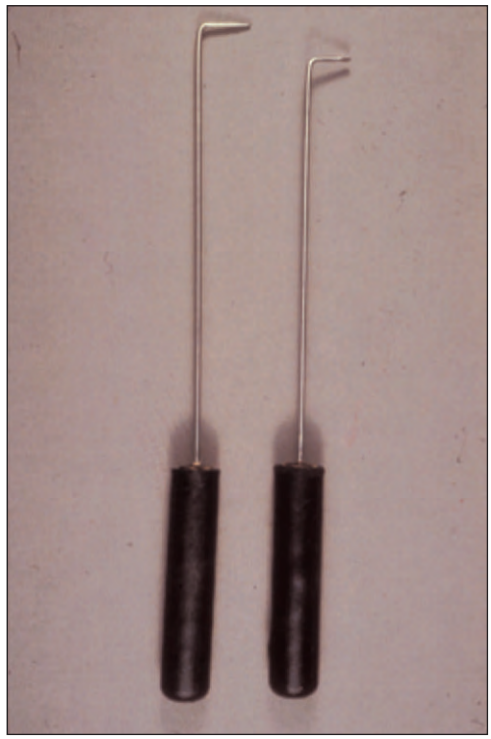


Figure 13.9. A set of thin dental picks used to probe and clean out periodontal pockets between cheek teeth.

A bucket containing diluted disinfectant (chlorhexidine) solution to rinse the horse's mouth, clean hands and clean equipment are essential when evaluating the oral cavity. A 400 ml[3] dose syringe with a blunt tip is useful to rinse the mouth before oral examination and during dental procedures. A high-pressure water irrigation unit can be used to remove debris from deep periodontal pockets. Irrigation will greatly aid in proper evaluation of pocket depth and tooth stability.

Hand care and protection are important factors when working in the mouth. Sharp enamel edges encountered on the teeth and abrasive instruments can cause injuries. Moisture and the bacteriological nature of partially chewed food always present in the horse's oral cavity add to the already less than desirable working conditions present. Moisturizing hand lotion will help lubricate and seal the hands from moisture and dirt. Gloves can be used to protect the hands from abrasions and debris. Soap and a nail brush are necessary for clean up properly after performing dentistry (see Chapter 15).

Dental signalment

Data on the horse's owner and trainer/manager/agent/ groom should include their names, addresses and means of contact. This is especially important for the person granting permission to work on the horse and the person responsible for payment for services rendered. The horse's insurance status and type of policy (mortality, loss of use, major medical and surgical) should be recorded. The stable name and address and the horse's location on the premises (barn number, paddock, stall number, etc.) can be helpful for rechecks and follow-up. The horse should be identified on the record by name and described by breed, color, sex, age, type of work and any special identifying markings, scars, brands or tattoos.

Dental history

The dental history should focus on oral-, dental- and gastrointestinal-related areas. Special consideration must be given to other body systems related to masticatory function or the safety of the animal or veterinarian. A history of cardiac abnormalities, respiratory disease, renal problems, hepatic disease or neurological symptoms could affect the way the animal is approached and restrained for examination and therapy. The animal's breeding history and pregnancy status could have an effect on dental care scheduling. Additionally, the horse's show or race schedule may have an impact on when work is performed and whether drugs used to sedate or treat the horse would be considered prohibited substances. The owner should be questioned about the horse's condition and type of exercise, temperament, stable vices, eating and drinking habits, fecal consistency and physical abnormalities. Specific questions asked could begin with these examples: Has the horse gained or lost weight over the past year? Have the horse's temperament or stable habits changed? Does the horse train well and what type of bridle and bit does he wear? Have any changes been noticed in the horse's head carriage or demeanor when bitted? Does the horse make any noises or wear a tongue tie when exercised? (See Chapter 2.)

Additionally, history notes regarding the horse's eating habits and vices should be taken and changes in eating or drinking patterns described. Animals that fail to chew effectively (physical malfunction, pain or neuromuscular abnormality) exhibit a variety of clinical signs ranging from weight loss and changes in fecal consistency to choke, colic and/or other gastrointestinal problems. Clinical detection of dental disease may at times be difficult because of the subtlety of signs. These may include reluctance to start eating, slow or intermittent eating, dribbling of food from the mouth (quidding), and head shaking or head tilting when eating. Sometimes these signs are only detectable by careful direct observation of the mastication process involving several different foods. This can be time consuming but it is unwise to accept the owner's report of 'normal eating.'[7] Horses with sharp enamel points may pack hay in the buccal space, pushing the cheeks away from the upper teeth before eating grain. Information about water sources and drinking habits should be ascertained and one should question if excessive salivation, oral odor, or nasal or lacrimal discharge has been noticed.

The diagnosis of dental-related head shaking or bit resentment may be relatively easy in cases with obvious dental disease but is often very difficult where there is no overt

evidence. Head shaking is often attributed to the presence of wolf teeth and to their position and size. Likewise, facial pain around the mental or infraorbital nerves may present symptoms of head shaking[7] (Fig. 13.10).

The horse's vaccination and deworming status should be determined. This is a good time to discuss these important preventative health topics. Tetanus toxoid may need to be given if corrective dental procedures such as wolf tooth extraction or abrasion from a sharp float rasp break the oral mucosal barriers. The owner should be questioned about the animal's history of infectious disease as well as the presence of infectious or contagious disease on the farm. This information may affect the degree of sanitation used between patients on the premises and the degree of disinfection or sterilization of equipment and personal items between farms.

If the equine patient is being seen for a particular dental complaint, a complete history of the problem should be ascertained and documented. A complete history is important as it has been statistically proven that horses presented with a dental complaint are 5.8 times as likely to have one or more selected dental abnormalities.[5] However, do not let a complaint of a dental problem distract or deter from taking a complete dental history and performing a thorough dental and physical examination. A systematic approach to history taking may quickly identify a primary problem that could be related to the animal's age, use, previous accidents, illnesses and/or behavioral problems.

Patient observation

Observation of the animal in its normal surroundings can provide information about stable management, eating habits and vices. The area where the dental examination is performed must allow for safe restraint. The area should be free of obstacles that could injure the horse, an attendant or the veterinarian. An area with a high ceiling shaded from bright sunlight with solid walls and a soft non-slip floor is ideal. Access to warm water and electricity may be a consideration.



Figure 13.10. This horse developed a severe headshaking behavior. Hyperesthesia and persistent rubbing of the right nostril and face caused chronic dermatitis and hyperkeratosis. Facial pain was ascribed to secondary sinus involvement from extensive periodontal disease. The caudal three maxillary teeth had been lost and much of the maxillary and lacrimal bones had been destroyed. The infraorbital canal was also virtually destroyed and the exposed infraorbital nerve ran through the inflamed and infected tissues.

Table 13.1. Description of the Condition Score System

Score	Description
1	*Poor:* Emaciated. Prominent spinous processes, ribs, tailhead and hooks and pins. Noticeable bone structure on withers, shoulders and neck. No fatty tissues can be palpated.
2	*Very Thin:* Emaciated. Slight fat covering over base of spinous processes. Transverse processes of lumbar vertebrae feel rounded. Prominent spinous processes, ribs, tailhead and hooks and pins. Withers, shoulders and neck structures faintly discernible.
3	*Thin:* Fat built up about halfway on spinous processes, transverse processes cannot be felt. Slight fat cover over ribs. Spinous processes and ribs easily discernible. Tailhead prominent, but individual vertebrae cannot be visually identified. Hook bones appear rounded, but easily discernible. Pin bones not distinguishable. Withers, shoulders and neck accentuated.
4	*Moderately Thin:* Negative crease along back. Faint outline of ribs discernible. Tailhead prominence depends on conformation, fat can be felt around it. Hook bones not discernible. Withers, shoulders and neck not obviously thin.
5	*Moderate:* Back is level. Ribs cannot be visually distinguished but can be easily felt. Fat around tailhead beginning to feel spongy. Withers appear rounded over spinous processes. Shoulders and neck blend smoothly into body.
6	*Moderate to Fleshy:* May have slight crease down back. Fat over ribs feels spongy. Fat around tailhead feels soft. Fat beginning to be deposited along the sides of the withers, behind the shoulders and along the sides of the neck.
7	*Fleshy:* May have crease down back. Individual ribs can be felt, but noticeable filling between ribs with fat. Fat around tailhead is soft. Fat deposits along withers, behind shoulders and along the neck.
8	*Fat:* Crease down back. Difficult to palpate ribs. Fat around tailhead very soft. Area along withers filled with fat. Area behind shoulder filled in flush. Noticeable thickening of neck. Fat deposited along inner buttocks.
9	*Extremely Fat:* Obvious crease down back. Patchy fat appearing over ribs. Bulging fat around tailhead, along withers, behind shoulders and along neck. Fat along inner buttocks, may rub together. Flank filled in flush.



Figure 13.11. (A) A pregnant saddlebred mare in good flesh with a nice fall hair coat and a body condition score of 6. (B) A thin saddlebred mare with a rough hair coat and a body condition score of 3.

The horse should be observed and his temperament assessed. Hair coat and body condition should be evaluated by observation and palpation and recorded.[11] Condition scores range from 1–9, with 1 describing an extremely emaciated animal and 9 describing an obese one (Table 13.1). The optimal condition score is between 5 and 6 on the scale (Fig. 13.11). Objective data such as photographs and weight measured with a scale or tape can be recorded. This data can be a valuable tool in management of dental health and patient nutrition. The animal's posture and stance should be observed and abnormalities such as swellings, injuries and hoof problems should be brought to the attention of the groom and noted in the record.

The stable floor should be surveyed for grain dropped from the horse's mouth or partially chewed boluses of hay that appear much like a cow's cud. This would indicate quidding (Fig. 13.12). Feces should be examined for volume and consistency as this can reflect how well the horse is masticating its feed (Fig. 13.13). Normal manure should be semimoist and fecal balls should be formed. Feces with long forage stems or whole grain indicate poor mastication. Long stems in poorly masticated feed can predispose the horse to esophageal choke, intestinal impaction colic or diarrhea (Fig. 13.14).

The horse's body and head type should be assessed and recorded. Head conformation can be reflected in the conformation of the dental arcades. Horses with short dished faces typical of the Arabian breed may have a more curved arcade with the last two lower molars ramped up in the curve of the mandible (curvature of Spee).



Figure 13.12. Severe quidding in a 6-year-old thoroughbred gelding as a result of chronic dysmastication caused by multiple dental abnormalities (including gross dyscongruity of the occlusal surfaces (shear mouth) and temporomandibular degenerative joint disease – see Fig. 13.16). This pile of semichewed food was accumulated overnight. The horse had a ravenous appetite but was quite unable to chew any fibrous food. Severe weight loss was reported.

Breeds that typically have long straight heads (Thoroughbred and some Warmblood breeds) are predisposed to malocclusion of the molar arcades, leading to rostral and caudal hook formation. Miniature horses and ponies are more prone to dental crowding and misplaced or malerupted dentition.



Figure 13.13. Abnormal equine manure. Notice the whole grain and large forage stems, signs of poor masticatory function. The coin is for size reference (2.5 cm diameter).

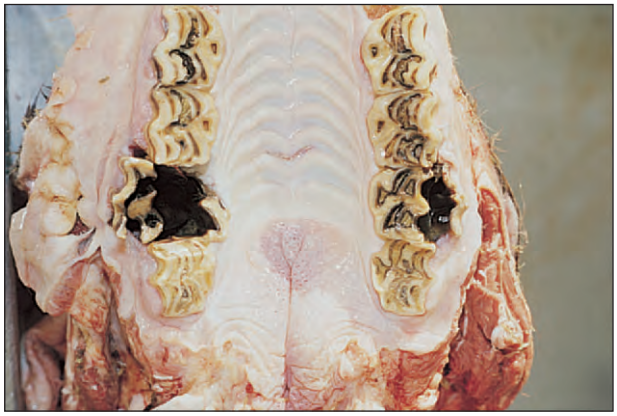


Figure 13.14. Diarrhea and severe weight loss were attributed to failure of masticatory efficiency in a 2-year-old pony. The feces contained significant amounts of unchewed long-fiber food. The pony had considerable difficulty chewing but managed to swallow reasonable amounts of hay and concentrate food and was diagnosed with severe decay of both fourth cheek teeth.

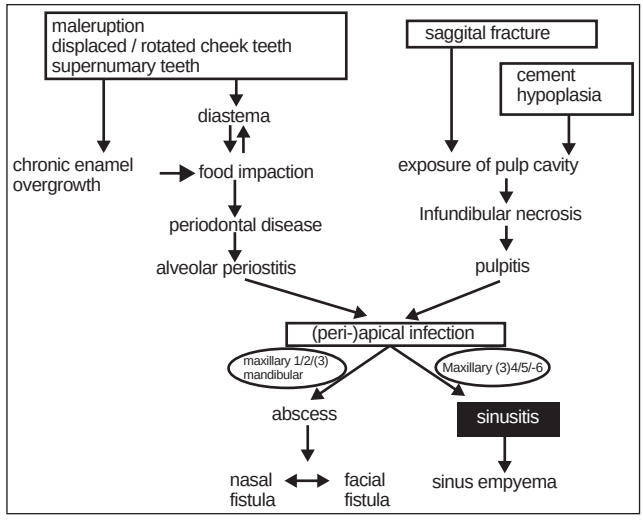


Figure 13.15. Algorithm showing the clinical consequences of dental disease on the surrounding anatomical structures.

Extra oral physical examination

During the basic physical examination (temperature, pulse, respiratory rate, auscultation of heart, lungs and abdomen) the clinician can assess the horse's temperament. The examination should be performed using techniques of good horsemanship that will gain the confidence of the patient (horse) and owner.

The head should be evaluated for symmetry, balance and gross abnormalities which may give clues to dental problems. Standing at the horse's side, head shape and conformation should be assessed and bumps or protuberances noted. Young horses between the ages of 2.5 and 4 years of age will have symmetrical, non-painful bony enlargements on the mandible and/or over the maxillary region. These enlargements are the result of normal tooth crown and root development of erupting permanent teeth and the shedding of deciduous caps. If these enlargements are hot, swollen, asymmetrical or associated with a draining tract, tooth pathology should be suspected (Fig. 13.15).

The eyes should be clear and free from lacrimal discharge. Standing directly in front of the horse, evaluate the head for symmetry. The ears, eyes, facial crests and nasal bones should be the same on both sides of the head. Observe and palpate the temporalis and masseter muscles and temporomandibular joints (Fig. 13.16).

Open the mouth slightly and percuss the frontal and maxillary sinuses. Palpate the parotid salivary glands and intermandibular lymph nodes. The ventral aspect of both sides of the mandible should be manually examined for enlargements; the vessels and parotid salivary duct at the rostral edge of the masseter muscle should be observed. Place open hands under the nose band of a loose halter and exert pressure on the cheeks at the level of the upper dental arcade (Fig. 13.17). Palpation from the level of the medial canthus of the eye, progressing rostrally over the masseter muscle to the level of the nasal notch allows detection of abnormal wear patterns on the upper dental arcades. If the horse resists this maneuver by tossing its head, it is most



Figure 13.16. Degenerative joint disease of the temporomandibular joint, which caused severe progressive pain with dysprehension and dysmastication, and progressive overgrowth of the cheek teeth resulting in a severe shear mouth. No lateral movement of the mandible was possible due to a 2 cm overgrowth of the buccal aspect of the maxillary cheek teeth. Severe, chronic weight loss was the reported primary sign.



Figure 13.17. The palm of the hand is placed under the loose nose band of the halter and pressure placed against the cheeks. Sharp enamel points as well as uneven upper dental arcades can be detected.

likely the result of pain from sharp enamel points pressing against the buccal mucosa. If sharp points are present, they should be floated prior to employment of a full mouth speculum into the oral cavity. Otherwise, as the mouth is opened, the cheeks are pushed tightly against the sharp enamel points and the horse will object to opening its mouth. Thus, a normally painless examination procedure will cause excruciating pain for the patient and subsequently the examiner.

Observe the nasal passages and palpate the false nostrils. Asymmetry of air flow, odor or discharge from the nostrils should be noted. Some of the most important secondary effects of dental disease relate to the close relationship between the cheek teeth and the nasal cavity and paranasal sinuses. Sepsis of the cheek teeth commonly results in either nasal or paranasal sinus sepsis or respiratory obstruction.

Observe and palpate the lips for bit injuries, noticing especially any scars or ulcers in the commissures. The lips of gray horses are a common area to find melanomas. The upper lip should be rolled up and the underside examined for a tattoo (Table 13.2). The labial mucosa should be salmon-pink in color and glisten with saliva. Ulcers or erosions should be documented and their cause determined. Keep in mind the possibility of vesicular stomatitis which is a reportable zoonotic disease. If any abnormalities are detected from the history or examination, consider observing the horse eating. This is best done before the mouth is washed for the oral examination and before sedation is administered.

When evaluating the horse's eating patterns, make a distinction between the horse having trouble with prehension and mastication and the horse that is unable to swallow (Fig. 13.18). Prehension requires neuromuscular coordination and an intact jaw and incisor arcade. Mastication is usually altered by tooth problems or abnormalities in the bones, muscles or temporomandibular joints. Tongue lesions or basal ganglion problems can adversely affect prehension and mastication. Swallowing is a more complex process and neurological, muscular or mechanical abnormalities in the pharynx or esophagus should be considered over a dental problem. Rabies is a fatal zoonotic disease that, in its early stages in the horse, mimics other types of eating abnormalities. Equine practitioners and any assistants working in horses' mouths should be vaccinated for rabies and have an antibody titer check periodically in those parts of the world where rabies is endemic.[12]

While standing in front of the horse, part the lips and examine the incisor arcade. Evaluate the incisor teeth for number, shape and symmetry. When viewed from the front, the occlusal line of the upper and lower incisors should be horizontal. When viewed from the side, the incisor table angle should be parallel to the angle of the facial crest which is usually about 10°–15° relative to the lower molar table surface. The incisors should be checked for anatomical characteristics used in assessing the horse's dental age. Compare the estimated age with the horse's real age as a discrepancy between these two could indicate abnormal incisor development or wear.[13] Important to keep in mind is the variation between horses of the same and different breeds in their dental characteristics and real age[14–17] (see Chapter 5). Observe the incisors while the jaw is moved through a full range of motion. Rostral–caudal movement of the mandible can be evaluated by observing the relationship between the upper and lower incisor arcades when the chin is raised

Table 13.2. A Note on Lip Tattoos
Most horses that race in the USA are permanently identified with a freeze brand on the neck or a tattoo on the upper lip. Each breed registry has a different alphanumeric system for identifying horses by their upper lip tattoo.
The Jockey Club of North America uses an alphanumeric system that consists of a letter of the alphabet followed by numbers. The letter corresponds to the year the horse was foaled, with 1997 starting a new 26-letter series. Therefore, 1996 would be Z and 1998 would be B. Horses imported into the USA are identified with an asterisk (*) at the beginning instead of a letter.
The American Quarter Horse Association uses a more random alphanumeric system of five numbers in older horses and more recently, four numbers followed by a letter.
The American Paint Horse Association uses a numbering system that consists of five digits. The first digit corresponds to the last digit of the horse's year of birth. These first digits would be repeated every 10 years.
Since 1982 the United States Trotting Horse Association has been using a system starting with A followed by three or four numbers. This would make foals born in 1998 have an upper lip tattoo that would start with the letter T followed by three or four digits. Horses born prior to 1982 were tattooed with three digits followed by a letter.
Arabian and Appaloosa horses that race in the United States require lip tattoos for identification. Their six-digit registration number is tattooed on their upper lip.



Figure 13.18. Dysmastication and dropping of saliva-soaked wads of hay (quidding) as a result of chronic dental infection in a 3-year-old Arabian colt which presented for weight loss over the previous 6 months. A very limited amount of hay was actually swallowed. Soft food and grass were eaten almost normally.

and lowered. The normal foal will have 3–4 mm (adult horse 6–8 mm) of rostral–caudal jaw excursion when the head is raised and extended as much as possible and the head flexed back into the vertical position.[18] Horses with severe wear abnormalities such as hooks or a step mouth may have limited rostral–caudal range of motion.

Lateral jaw excursion is best evaluated by standing to one side of the horse and holding the head stationary with a hand on the bridge of the horse's nose. The other hand is used to grasp the mandible and while pressing the mouth shut, move the jaw from side to side. During this maneuver, the jaw moves laterally from pivot points at the temporomandibular joints. As the jaw is moved from one side to the other, observe the range of lateral motion before tooth contact is made with a sloped portion of the molar table. The more rostral portion of the arcade will contact first. This will work more caudal as the jaw moves farther to each side. Horses that have had the table surface from the rostral cheek teeth reduced in height will have to move the lower jaw further to one side before the sloped upper and lower molar tables grind over one another and the incisors gradually separate.

Two distances are noted during this maneuver. The first is the lateral distance noted before the sloped molar tables make the upper and lower incisor arcades begin to separate. The second distance noted is the total lateral distance the mandible travels. By observing the incisors and listening to and feeling the molar arcades grind on one another, one can gain information about the slope of the molar tables and symmetry of the occlusal contact of the upper and lower arcades.[19,20] Normal lateral excursion will possess a relatively even, subtle to moderate vibration and sound. Deviations from this can be an indication of abnormal dental contact due to long or tall teeth, hooks, etc. (see Chapter 16). It must be kept in mind that this maneuver does not mimic the chewing motion of the horse as outlined in Chapter 4. Remember, if the horse resists this part of the dental examination, sedation may be indicated to help the horse relax and allow a more thorough physical examination. Detomidine (0.01–0.04 mg/kg, i.v.), xylazine (0.05–1 mg/kg, i.v.) or romifidine (0.04–0.1 mg/kg, i.v.) alone or in combination with butorphanol (0.025–0.1 mg/kg, i.v.) will give satisfactory sedation even in particularly fractious horses. With sedation, a complete dental examination can be carried out safely and thoroughly.[21]

Oral examination

The mouth is the window into the body. The oral mucous membrane is a thin sheet of tissue that permits the veterinarian to view changes in vessels and connective tissue beneath the oral mucosa. There are relatively few sensory nerve endings in the gingiva, which makes it a safe area to depress for observing vascularity and capillary refill time (CRT).

The oral examination begins with the interdental space and adjacent structures. This area often reveals the performance horse's bitting history. The lip commissures, bars of the lower jaw, tongue and palate are evaluated. In male horses over 4 years of age canine teeth can be observed. The lower canines lie rostral to the upper canines making for a longer lower diastema. The upper canines erupt in the suture between the incisive and maxillary bones and usually break through the mucosa 2–8 months after the lowers. Young adult stallions and geldings between the ages of 4 and 6 years of age may have canine teeth in various stages of eruption. Eruption cysts or tenting of the mucosa with ulceration over these teeth can cause oral pain and bitting problems. It bears repeating that long sharp canine teeth can be a danger to the examiner and care should be exercised to avoid injury when manually examining the mouth. These teeth can be shortened and blunted to aid in examination. About 25 per cent of all mares have one to four rudimentary canine teeth.[22] Dental plaque or tartar accumulation around the canines leading to gingivitis will often be seen in older horses.

The upper and lower interdental spaces should be observed and palpated. Firmly run a thumb over the mucosa, feeling for protuberances above or below the gum line and observe the horse's response to pressure. The lower bars should be checked for sharpness, bony irregularities, mucosal ulcers or thickenings.[23] The presence of lower first premolars can be

detected in horses and should be palpated for, just rostral to the lower first cheek teeth. The upper edge of the diastema is palpated for bony abnormalities and upper first premolars. These caniniform teeth are referred to as 'wolf teeth' and erupt between 6 and 18 months of age. If present, they can be found along the edge of the maxillary and palatine bones from the palatal side of each upper PM 2 to 2–3 cm rostral to this location. These teeth usually erupt through the oral mucosa but can migrate under the mucosa and remain there as bumps. The unerupted wolf teeth, referred to as 'blind wolf teeth,' can cause oral discomfort and training problems in bitted horses. Wolf teeth come in a vast array of shapes and sizes with the visible crown shape having no relation to the size or shape of the root. The distance from the commissures of the lips to the rostral edge of the first cheek teeth should be noted as this varies among horses. This distance will affect the ease with which one works on the rostral teeth and may affect the comfortable position of the bit in a working horse.

The tongue should be checked for function and any anatomical abnormalities noted. Tongues are frequently injured from harsh bits or neglected tongue ties. Calluses or ulcerations are the result of chronic trauma from sharp teeth. Observe and palpate the hard palate. Lampas, or thickening of the palatal mucosa just behind the upper incisors, is common in young horses that are erupting permanent dentition (Fig. 13.19). The hand can be introduced into the interdental space and a thumb pressed on the hard palate to make the horse open its mouth.

The non-speculum intraoral inspection and palpation is carried out next. Each clinician should develop his/her own technique for oral palpation and inspection as a prelude to rinsing the mouth and insertion of the full mouth speculum. The horse should be encouraged to open its mouth either by pushing a finger from the outside of the cheeks between the dental arcades or by hooking a thumb into the commissures of the lips. Great care should be exercised whenever a finger is placed in the mouth to avoid serious injury.



Figure 13.19. Edema of the hard palate (lampas) due to the physiological effects of incisor eruption. Note the level of the hard palate is below the occlusal margin of the upper incisor teeth.

In the two-handed method of oral palpation, the right side of the dental arcade is palpated by approaching the horse from the left side. The left labial commissure is parted with the right hand and the tongue is grasped and pulled through the left interdental space. Care should be taken so that the tongue is not traumatized by canine teeth. The cheek teeth of the right maxilla can then be examined by manipulating the right side of the lips with the left hand and by inserting the left hand into the horse's mouth. This process is then reversed to permit a similar inspection of the left arcades. A flashlight or head lamp greatly facilitates this examination. The examination must not be conducted in a leisurely fashion as it may annoy the horse to have its tongue grasped for long periods.

For experienced operators, the one-handed of oral palpation, technique is recommended. The horse is approached from the front and the right side and the mouth is palpated by inserting the right hand into the right interdental space with the palm facing laterally. The hand should be slightly cupped and the back of the hand used to force the tongue between the left cheek teeth arcades. This prevents the horse from completely closing the mouth and enables the examiner's fingers to run quickly along the arcades palpating both the upper and lower arcades for hooks, spaces, overgrowths and irregularities. The procedure is then repeated on the other side of the mouth using the left hand to examine the left dental arcades. As previously stated, when using the two-handed technique it is best to carry out this procedure as quickly as possible. The hand is examined when removed from the mouth, taking note of unchewed feed materials and/or odor. This method is certainly not suitable for attempting to demonstrate specific lesions to owners. At this point the mouth should be rinsed, taking notice of the volume, consistency and smell of material flushed from the mouth. Neither of the above techniques is adequate to evaluate the buccal aspects of the upper cheek teeth and the caudal recesses of the mouth.

The easiest and safest way to thoroughly evaluate the oral cavity is by using a full mouth speculum. To place the McPherson type speculum in the mouth, the examiner stands to the left side of the horse. With the left hand holding the mouthpiece and the right hand holding the poll strap, the mouthpiece is introduced between the incisors in the same manner as a bit. The left thumb and forefinger are used to open the mouth and guide the mouthpiece into place between the incisors while the right hand applies steady tension to the halter strap from behind the horse's poll. When the speculum is properly positioned, the left hand is maneuvered to the halter's buckle to adjust the strap length until the speculum strap is snug. The mouthpiece is adjusted from the front to square it with the incisors. A final check is made to ensure that the teeth and incisor plates are free of tongue, lips and examiner's digits so they are not pinched when opening the speculum. It is important to adjust the nose band and chin strap to allow a stable yet comfortable fit on the horse. The jaws of the speculum are opened one notch at a time alternating each side until the jaws are opened two to

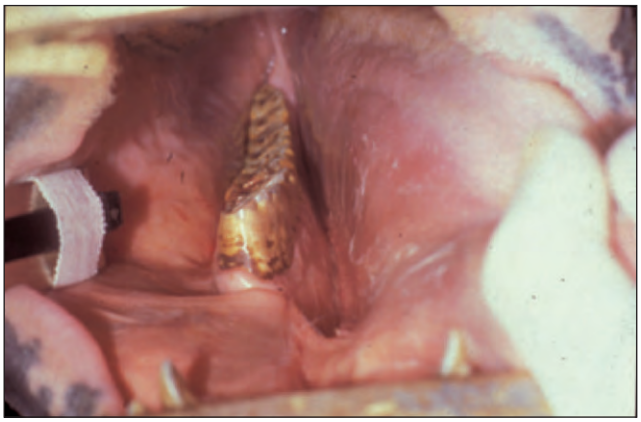


Figure 13.20. The oral cavity of a restrained horse can be visualized with a full mouth speculum, cheek retractor and a good light source.

three notches. If the mouth resists being opened with the speculum in place, the temporomandibular joints and bony structures of the jaw should be carefully evaluated before excessive force is placed on the jaw. At this point the oral cavity is ready for visualization and palpation (Fig. 13.20). Use of a head support stand or metal frame dental halter is recommended to elevate the head of a sedated horse to a comfortable height for good visualization and palpation.

To examine the oral cavity, good illumination is critical. A battery-operated halogen light or transilluminator with a 7–15 cm extension will work. A head light provides good illumination while allowing both hands to be free for instrumentation inside the mouth. A blade retractor fitted with an illuminator will aid in the evaluation of the buccal recesses. A basket retractor designed by Stubbs will keep the tongue and buccal mucosa pulled away from the teeth for good visual access to the last few cheek teeth (Fig. 13.21) (RC Stubbs, personal communication, 1997). A flexible fiberoptiscope, rigid laparoscope or intra-oral camera prove useful in obtaining a close-up and detailed view of the caudal recesses of the mouth. The oral soft tissues should be observed with special attention paid to the palate, tongue and buccal mucosa (Fig. 13.22). The teeth should be evaluated for conformation, position and number. Common premolar findings include hooks, ramps, erupting teeth or loose caps or cap slivers. In the center of the molar table, one may observe long teeth, wave mouth, cupped-out or decayed infundibula, missing teeth or split or misplaced crowns. The caudal dental arcades should be inspected and buccal ulcers, sharp enamel points or hooks, supernumerary or missing teeth or ramped dental arcades should be noted. Enamel points that normally form on the buccal and lingual enamel folds or cingula usually do not protrude beyond the level of occlusal surface of the cheek teeth. The acute angle between the vertical edge of the tooth and the occlusal surface can cause sharp enamel points to look and feel quite prominent. Inspect the mesial and distal dental margins for abnormal tooth contact or feed packed into gingival pockets. A 3.5 mm diameter dental pick with a shaft 31cm long can be used to probe the four corners of the cheek teeth to detect and clean out periodontal pockets (Fig. 13.23). A calibrated pick can be used to measure gingival pocket depth, which will range from 0.5–12 mm for normal teeth. It has been shown that gingival pocket depth measurements at the corner of the teeth significantly increased with periodontal disease.[24,25] Defects have been found on the occlusal surfaces of a large number of periapically infected teeth.[3] These defects can be detected by carefully probing the occlusal surfaces of suspect teeth.

Figure 13.21. A stainless steel basket retractor designed by Stubbs will keep the cheek and tongue out of the field of vision while performing an oral examination.

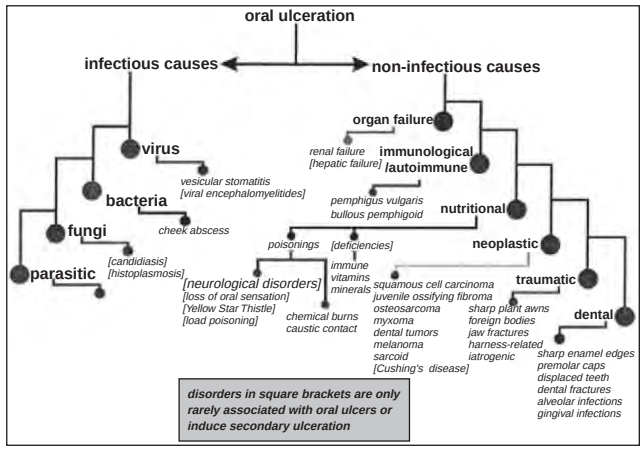


Figure 13.22. Oral ulceration: a flow chart to facilitate the investigation and diagnosis of oral ulcers.



Figure 13.23. A dental pick being used to remove feed from a gingival pocket adjacent to fractured 407.

The oral cavity should be palpated, feeling the buccal, occlusal and lingual surfaces of all four arcades. The gingival margins of the cheek teeth should be uniform with no feed packed between them. The crown height should be the same on the mesial and distal aspect of each tooth. The crown height should be longer on the buccal aspect of the uppers and the lingual aspect of the lowers. This reflects the normal 10°–15° slope of the molar arcade. Any deviation or asymmetry in molar table height or angle should be noted. Each exposed tooth crown should be grasped between the thumb and forefinger and checked for stability, noting any movement. The occlusal surfaces of the arcades should be palpated, noting any defects or asymmetry in the occlusal crown surface. Keep in mind that any defect in one arcade will usually be reflected in a wear abnormality or defect in the opposite occlusal arcade.

Ancillary diagnostic tests

If the initial dental examination findings reveal signs of dental disease, other diagnostic techniques can be employed to make a more definitive diagnosis. A more thorough oral examination can be carried out on a sedated and restrained or anesthetized horse. Endoscopic examination of the nasal passages, larynx and oral cavity is often indicated. Skull radiographs, both plain and contrast film studies, give added information about dental, osseous and sinus structures. Details of skull radiology are outlined in Radiology. Standing skull films with the mouth open can give the veterinarian a more complete assessment of the occlusal pattern of the dental arcade.[26,27] Other imaging modalities such as ultrasonography, computerized tomography, nuclear scintigraphy or fluoroscopy may reveal a more accurate picture of some dental pathologies. Radiographic examination of the equine skull can add valuable information to physical examination, oral examination and endoscopic findings. Most equine practitioners have access to portable radiographic equipment, making this a routine part of the complete work-up of any case with suspected dental pathology. Often, radiology is indicated before major dental corrections can be safely undertaken. Other imaging modalities such as ultrasonography, digital radiology, computerized tomography and gamma scintigraphy are also available to many practitioners. These diagnostic tools can aid in making an accurate diagnosis.

Cases of dental disease that involve the upper last four cheek teeth may be associated with sinus disease. The most common presenting sign for sinus disorders is a unilateral nasal discharge. Percussion of the maxillary and frontal sinuses with the mouth open may help in the detection of masses or fluid that fill the sinuses. Sinus centesis or sinuscopy singularly or combined with culture, cytology and/or histopathology can help differentiate primary sinus conditions from sinusitis secondary to dental disease or oral contamination. Endoscopy of the nasal passages can confirm whether or not the discharge is coming from the nasomaxillary opening. The nasomaxillary area cannot be directly observed with endoscopy, but drainage limited to the caudal aspect of the middle nasal meatus suggests sinus drainage. Malerupted teeth have been seen to narrow the nasal passages, which can make passing the scope difficult if not impossible on the affected side.

Sinuscopy has been valuable in diagnosing and treating some sinus disorders without the need for exploratory flap sinusotomy.[28] Lavage can be accomplished through the arthroscope cannula and suction can be used to remove ancillary debris and fluid. Endoscopic examination has proven useful in the diagnosis of sinusitis, neoplasia, cyst formation, tooth root abscesses and primary hemorrhage.

Standing radiography of the head is a practical, routine procedure to aid in the diagnosis of sinus involvement or disease. Fluid accumulation in the sinuses or dense masses confirms the presence of sinus disease. Sinus centesis or trephination and biopsy can be carried out in the standing horse under local anesthesia. Cytologic evaluation of fluid collected from the sinus can help in the differential diagnosis of sinus infection, tumor or cyst. Bacteriologic culture of the aspirate can be helpful in differentiating primary sinus disease from a pure culture of one bacterial species as opposed to a mixed growth of bacteria from diseased tooth roots. Bacterial sensitivity testing can indicate the correct antimicrobial agent to use for therapy. Care must be taken in evaluating microbiological results because of the presence of secondary organisms superimposed over primary infections or neoplastic conditions.

A description of examination of the paranasal sinuses using an arthroscope or endoscope has been detailed in the

literature.[29,30] Endoscopic examination of the sinuses can be performed utilizing local anesthesia in the standing sedated horse or under general anesthesia. The references describe portals for examining the frontal, caudal, and rostral maxillary sinuses. Examination of the dorsal conchal and sphenopalatine sinuses is performed using the frontal sinus and caudal maxillary sinus portals.

Dental records and treatment planning

The horse's signalment, use and management should be recorded. Pertinent history should be noted with special emphasis on digestive system or performance problems. The horse's general body condition should be recorded and a numbered body score assigned. The results of the masticatory system examination should be recorded and problems listed in order of significance. A plan for treatment of each problem should be outlined based upon the results of history, clinical findings and oral examination before proceeding with any dental work. This problem orientated approach is important because the owner and/or trainer should be informed of any abnormalities, given a plan for treatment and an estimate of the cost before any corrective procedure is carried out. An owner consent statement is often included in record forms and can minimize problems should a legal claim be filed against the veterinarian or a bill come in dispute for collection.

Oral and dental charting

Charting is the process of recording the state of health or disease of the teeth and the oral cavity.[31] To properly chart the mouth, the dental formula and anatomical locations in the mouth must be standardized to make documentation consistent. Use of standard abbreviations for dental terms to describe anatomical boundaries, pathology, diagnostics and therapeutic procedures have made communication possible between colleagues in both the veterinary and human dental professions.[12,32]

The American Veterinary Dental College Nomenclature and Classification Committee has endorsed the use of the Triadan tooth numbering system. Numbering is based on a fully phenotypic dentition made up of 44 teeth. This three-digit system uses the first digit to designate the quadrant and arch location and whether the dentition is deciduous (primary) or permanent (adult). The quadrant implies the right or left side of the individual. The arch denotes maxillary or mandibular. The numbering sequence is upper right, upper left, lower left and lower right. The permanent (adult) dentition utilizes numbers 1–4, and the deciduous (primary) dentition uses numbers 5–8. In each quadrant, the first or central incisor is always 01, with incisors numbered 01–03, and the canines, whether present or not, take up the 04 position in the formula. The premolars are numbered 05–08 and the molars are numbered 09–11.[33,34]

To fully understand equine tooth development and anatomy and to properly document abnormalities for dental record keeping, certain oral topographical terms have been defined. For a unique identification of each surface of a tooth, the following descriptions are used:

apical – toward the apex or root
buccal – toward the cheeks
coronal – toward the tooth crown
distal – posterior or caudal (interproximal surface farthest from mandibular symphysis)
labial – toward the lips
lingual – toward the tongue in the lower arcade
marginal – border or edge near the gingival margin
mesial – anterior or rostral (interproximal surface nearest to mandibular symphysis)
occlusal – masticating surface
palatal – toward the palate in the upper arcade
proximal or interproximal – between the teeth.

Computerized dental charting and record keeping is used in human and veterinary dentistry. Standard abbreviations and record forms are essential to make this transition into equine practice. Some common dental abbreviations are listed below.

SP – sharp enamel points
BI (L, A or U) – buccal injury (laceration, abrasion, ulcer)
LI (L, A or U) – lingual injury (laceration, abrasion, ulcer)
CH – crown hook
BK – beak
RP – ramp
Sp – step
Wa – wave
ETR – excessive transverse ridges
Cup – cup in central portion of crown
TC – tall crown
PD – periodontal pocket
Fx – fracture
CAL – calculus
DT/R – deciduous root sliver or fragment
CA – cavity
SN – supernumerary
O – missing tooth
WC – worn crown
ROT – rotated
X – extraction.

Other shorthand systems have been used to grade dental lesions.

Grades of periodontal disease[3]

+	Local gingivitis with hyperemia and edema
++	Erosion of the gingival margin

+++ Periodontitis with gum retraction
++++ Gross periodontal pocketing and destruction of alveolar bone.

Grading the severity of dental caries[3]

Grade 1 Caries of the infundibular cementum
Grade 2 Caries of infundibular cementum and surrounding enamel
Grade 3 Caries of infundibular cementum, enamel and dentin
Grade 4 Splitting of the tooth as a result of caries
Grade 5 Loss of tooth due to caries.

This system is utilized on the sample dental charts provided. A dental chart can be used to record the examination, assessment and pathology. A second diagram can be used to denote the specific treatment and post-treatment result or a single diagram can be used as a combined report form (Fig. 13.1A,B).

Prophylactic oral examination by age

Weaning dental examination (birth to 18 months)

Foals should be examined soon after birth and again at weaning for any congenital defects in head symmetry or masticatory function. Foals should be evaluated for proper eruption sequence and alignment of the incisors. If facial asymmetry or severe malocclusion are detected at an early age, a genetic consultation and possible surgical or orthodontic correction can be discussed with the owner. The premolars should be examined every 6 months for sharp enamel points, abnormal wear and improper number and position.

Young performance horse examination (18–52 months)

Young athletic horses are asked to respond to bitting when the mouth is most active. During this 3-year period, all 24 of the deciduous teeth are shed and 36–44 permanent teeth erupt. Eruption cysts in the gums over erupting permanent teeth, gingivitis, periodontal disease and loose caps or cap slivers can cause oral pain and associated eating and training problems. Asymmetrical shedding of deciduous teeth can unbalance the occlusal table, leading to abnormal mastication and tooth wear. Cheek or tongue pain and ulcerations from sharp enamel points on the premolars, molars and wolf teeth can cause trouble with mastication and severe difficulty for the trainer and/or rider trying to cue the horse from the bit. Unequal eruption of permanent incisors leads to abnormal wear and a smiling or frowning incisor table. This may prohibit normal lateral jaw excursion. Retained or displaced incisor caps or supernumerary incisors can also lead to malocclusion or oral pain.

Wolf teeth can cause bitting problems or interfere with rounding the rostral edges of the premolars for the bit. Horses that have not had previous dental work performed may have wolf teeth present. Blind, unerupted, displaced or lower wolf teeth should be identified.

The canine teeth should erupt in most male horses between 4.5 and 5 years of age. Some canines may not penetrate the loose oral mucosa that tents up over the crown. This often leads to painful eruption cysts.

All young athletic horses should have a biannual oral examination. Horses that are shedding caps may need attention more often. Keep in mind that when PM 2 caps are shed, the permanent teeth will be in wear and sharp within 3–6 months.

Adult performance horse examination (4–10 years)

The adult horse should have a full set of permanent dentition by 5 years of age. Most of these horses have already been exposed to training and bitting. The long-crowned hypsodont teeth continue to erupt into the oral cavity. Discrepancy in occlusal contact will begin to show in the young adult and become progressively worse as the horse ages. In the male horse, canine teeth are fully erupted by middle age and the sharp crowns can be formidable weapons.

It is important to critically evaluate the incisor and molar tables for occlusal contact and balance. The caudal cheek teeth must be thoroughly evaluated by palpation to detect hooks or ramps, sharp buccal or lingual enamel points and arcade balance. Regular tall transverse ridges are present at this age (Fig. 13.24). Transverse ridges are most prominent in the 3–5-year-old and should become less pronounced as the horse approaches 10 years of age.[35] The mandible should be moved from side to side with the mouth closed to evaluate lateral jaw excursion and balance. During this part of the examination the incisors should be evaluated for symmetry, balance, length and contact. Adult performance horses should be examined every 6–12 months and corrective procedures performed as needed.

Mature horse examination (10–18 years)

Abnormalities of wear are a significant problem in mature horses. Slight malocclusions and uneven wear patterns place abnormal stresses on teeth in the dental arcades. This predisposes the dental table to abnormal crown wear, crown fracture and periodontal disease. Annual dental examinations are the rule, but some horses with dental and facial abnormalities may benefit from more frequent attention. At this age, the reserve crowns begin to show excessive wear and the older upper molars (109 to 209) may begin to suffer from infundibular decay or attrition that can lead to a 'wave' mouth forming on the central portion of the molar arcade. Ramps, hooks and beaks can be quite prominent and may require extensive correction to return the horse to its normal dental form.



Figure 13.24. An upper molar arcade from a 7-year-old horse. Notice the regular exaggerated transverse ridges. These ridges are a normal feature in most 3- to 10-year-old horses.

Terms that may be encountered in describing abnormalities of wear can cause confusion. Some common terms are ramp, hook, beak and curved arcade. A ramp is an area where the dental arcade gently slopes and the exposed crown is taller at one end of the tooth than the other end. A hook is an abrupt elevation at the rostral or caudal edge of the molar arcade that involves the occlusal surface of the tooth. Quite often a hook will appear as a ramp from the buccal aspect, but a cupped-out, steep elongation seen on the palatial side of the tooth identifies the formation of a hook as opposed to a ramp. A beak is an enamel point that forms on the rostral or caudal edge of the arcade. Curved arcade describes the occlusal surface where it gently slopes to follow the curve of the jaw and the exposed crown length is the same in the front and the rear of the tooth.

Incisor abnormalities are more prominent in the middle-aged horse than in younger horses. Small discrepancies in occlusion or tooth alignment begin to manifest themselves as abnormalities of wear or as severe tooth angulation deformities. The incisor arcades may take on a grinning, stepped, irregular, tilted or frowning conformation. Horses that have had abnormal masticatory patterns may not wear the incisors evenly on both sides of the arcade, leading to a tilted or diagonal bite. Hook formation on the rostral upper and caudal lower edges of the molar arcades causes the arcade to shift caudally and can bring the incisors out of full occlusion. When this occurs the central upper incisors no longer contact the central lower incisors and they become elongated. From the standpoint of correcting oral balance, this is important. When the molar hooks are removed, the lower jaw can shift forward into a more normal position and the elongated incisors will meet in full occlusion. This can bring the molar tables out of occlusal contact and lead to problems with mastication. Correction would entail shortening and balancing the elongated incisor table.

By middle age, canine teeth become formidable weapons in stallions and geldings. Some mares may have small or rudimentary canines. Large, sharp canines can injure the horse's tongue or a groom's hands. Prominent canines can also reduce the space for the tongue and some horses suffer from excess tongue pressure when bitted. Mature horses benefit from annual oral examinations and corrective procedures performed as needed.

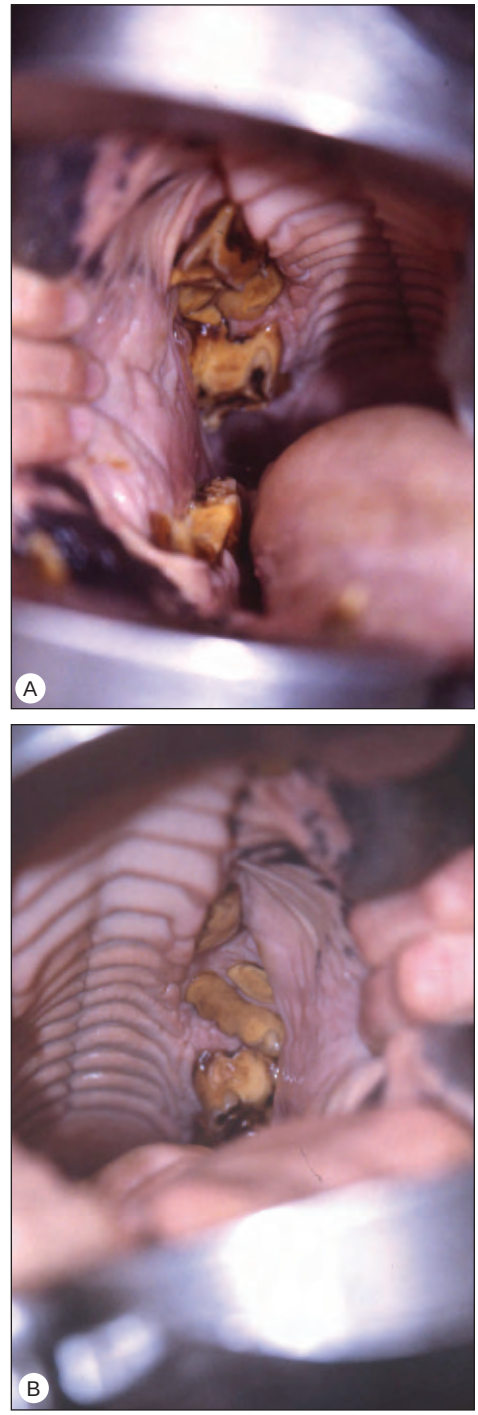


Figure 13.25. (A) and (B) The left and right upper molar arcades of a 24-year-old horse. The upper molars have worn to the bottom of the crown and have become smooth.

Geriatric horse examination (18 years and older)

Most old horses suffer from some form of dental disease. There is an accumulating effect throughout life of occlusal or wear abnormalities. The abnormal wear patterns previously discussed often become more pronounced. Abnormal stresses on teeth with less reserve crown cause teeth to shift from their normal occlusal position (Fig. 13.25A,B). Tartar accumulation around lower canines is quite common and leads to gingivitis and periodontal disease. Aged horses suffer from a high incidence of periodontal disease.[7]

The importance of a general physical examination should be emphasized before dental procedures are considered. Even if severe dental disease is present, it may not be the main contributing factor to weight loss or poor condition. Many older horses suffer from kidney or liver disease, anemia and low-grade sepsis. Neoplasia is also more commonly encountered in the older horse. Musculoskeletal problems, respiratory disease, laminitis and pituitary dysfunction are common in older horses. Body systems should be carefully evaluated before an aged horse is sedated or extensive dental procedures, such as extractions, are performed.[36,37]

Care should be taken when working on an old horse's dentition not to damage, further loosen or accidentally extract teeth. However, if an old horse's teeth can be digitally extracted with minimal force, they should be removed. Owners should be educated about normal dental attrition and recognize the importance of oral health in keeping the horse productive into old age. All horses wear their teeth to the root if they live long enough. The teeth will become smooth on the occlusal surface because the crown contains enamel and the root does not. These smooth-mouthed horses will develop severe abnormalities of wear in a relatively short time, requiring frequent oral examinations and dental maintenance in order to keep the mouth healthy. Since the teeth have lost their sharp enamel edges, mastication and digestion of forage becomes more difficult and less efficient. Therefore, most old horses need to have their diet adjusted by reducing the amount of rough forage that can predispose them to esophageal choke or colic from intestinal impaction. The nutritional needs of older horses must be met with the use of processed geriatric diets. Geriatric horses may need evaluation whenever problems arise with eating or change in disposition, but at least once a year.

Summary

The basis for a complete equine dental examination is the development of a routine treatment plan that is used on each patient. By utilizing proper restraint techniques and equipment, a thorough examination can be performed with minimal stress to the horse and risk of injury to the veterinarian. Finally, a complete written record of the dental examination, findings, treatment plan and follow-up recommendations is essential for the long-term management of equine oral health.

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REFERENCES

1. Kirkland K (1994) A survey of equine dental disease and associated oral pathology. Abstract, *Proceedings of the 3rd World Veterinary Dental Congress,* Philadelphia, p. 103.
2. Baker GJ (1997) Some aspects of equine dental disease. *Equine Veterinary Journal,* 2, 105.
3. Baker GJ (1979) A study of equine dental disease. PhD Thesis, University of Glasgow, pp. 78–82.
4. Greene S and Basile T (2002) Recognition and treatment of equine periodontal disease. *Proceedings of the 48th Annual Convention of the American Association of Equine Practitioners,* pp. 463–466.
5. Uhlinger C (1987) Survey of selected dental abnormalities in 233 horses. *Proceedings of the 33rd Annual Convention of the American Association of Equine Practitioners,* pp. 577–583.
6. Orsini P (1989) Handout notes from Orsini's presentation at the Eastern States Veterinary Conference, Orlando, FL.
7. Knottenbelt DC (1999) The systemic effects of dental disease. In: *Equine Dentistry* (1st edn), eds. GJ Baker and J Easley, WB Saunders, London, pp. 127–138.
8. Baker GJ (1998) Dental physical examination. *Veterinary Clinics of North America, Equine Practice,* August, 1998, 247–257.
9. Earley ET (2002) Creating a dental form for interactive use in the field. *Proceedings of the 48th Annual Convention of the American Association of Equine Practitioners,* pp. 453–457.
10. Fischer DJ and Easley J (1993) Equine dentistry: proper restraint. *Large Animal Veterinarian,* 48, 14–22.
11. Henneke DR (1985) A condition score system for horses. *Equine Practice,* 7, 13–15.
12. Centers for Disease Control and Prevention: Humane Rabies Prevention–United States (1999) Recommendations of the Advisory Committee on Immunization Practices (ACIP). *Morbidity Mortality Weekly,* 1999, 48 (No. RR-1).
13. Galvayne S (1886) *Horse Dentition: Showing How to Tell Exactly the Age of a Horse up to Thirty Years.* Thomas Murray and Son, Glasgow, p. 16.
14. Walmsley JP (1993) Dental aging in horses between five and seven years of age. *Equine Veterinary Education,* 5, 295.
15. Richardson JD, Lane JG and Waldron KR (1994) Is dentition an accurate indication of age in the horse? *Veterinary Record,* 135(2), 31–34.
16. Richardson JD, Cripps PJ and Lane JG (1995) An evaluation of the accuracy of aging horses by their dentition. *Veterinary Record,* 137(5), 117–121.
17. Muylle S, Simoens P and Lauwers H (1996) Aging horses by an examination of their incisor teeth: an (im)possible task. *Veterinary Record,* 138(13), 295.
18. Allen T (2003) Examination. In: *Manual of Equine Dentistry,* ed. T Allen, Mosby, St Louis, pp. 49–70.
19. Rucker BA (1996) Incisor procedures for field use. *Proceedings of the 41st Annual Convention of the American Association of Equine Practitioners,* p. 22.
20. Rucker BA (2002) Utilizing cheek teeth angle of occlusion to determine length of incisor shortening. *Proceedings of the 48th Annual Convention of the American Association of Equine Practitioners,* pp. 448–452.

21. Baker GJ and Kirkland KD (1995) Sedation for dental prophylaxis in the horse: a comparison between detomadine and xylazine. *Proceedings of the 42nd Annual Convention of the American Association of Equine Practitioners,* p. 40.
22. Miles AEW and Grigeon C (1990) *Colyer's Variations and Disease of the Teeth of Animals* (rev. edn). Cambridge University Press, Cambridge, p. 121.
23. Johnson TJ (2002) Surgical removal of mandibular periostitis (bone spurs) caused by bit damage. *Proceedings of the 48th Annual Convention of the American Association of Equine Practitioners,* pp. 458–462.
24. Stock S (1997) Periodontal parameters in the normal and pathological equine tooth. *Proceedings of the World Equine Dental Congress,* Birmingham, April 2, 1997, pp. 92–95.
25. Dixon PM, Tremaine WH, Pickles K *et al.* (1999) Equine dental disease part 2: a long-term study of 400 cases: disorders of development and eruption and variations in position of cheek teeth. *Equine Veterinary Journal,* 31, 519–528.
26. Easley J (2002) A new look dental radiology. *Proceedings of the 48nd Annual Convention of the American Association of Equine Practitioners,* pp. 412–420.
27. Barakzai SZ and Dixon PM (2003) A study of open-mouthed oblique radiographic projections for evaluating lesions of the erupted (clinical) crown. *Equine Veterinary Education,* 5(3), 183–188.
28. Freeman DE (2003) Sinus disease. *Veterinary Clinics of North America, Equine Practice,* 19, 209–243.
29. Ruggles AJ, Ross MW and Freeman DE (1991) Endoscopic examination of normal paranasal sinuses in horses. *Veterinary Surgery,* 20, 418–423.
30. Ruggles AJ, Ross MW and Freeman DE (1993) Endoscopic examination and treatment of paranasal sinuses in 16 horses. *Veterinary Surgery,* 22, 508–514.
31. Wiggs RB and Lobprise HB (1997) *Veterinary Dentistry Principles and Practice,* Lippincott-Raven Publishers, Philadelphia, New York, p. 96.
32. Pence P (2002) The dental examination. In: *Equine Dentistry: A Practical Guide,* ed. P Pence, Lippincott, Williams and Wilkins, Philadelphia, pp. 53–78.
33. Floyd MR (1991) The modified Triadan system: nomenclature for veterinary dentistry. *Journal of Veterinary Dentistry,* 4, 18.
34. Foster DL (1993) Nomenclature for equine dental anatomy based on the modified Triadan system. *Proceedings from the Annual Meeting of the International Association of Equine Dental Technicians,* Detroit, p. 35.
35. Michael J (2001) ATR therory and analysis. Thesis, Academy of Equine Dentistry, Idaho, pp. 7–8.
36. Brosnahan MM and Paradis MR (2003) Demographic and clinical characteristics of geriatric horses: 467 cases (1989–1999). *Journal of the American Veterinary Medical Association,* 223(1), 93–98.
37. Brosnahan MM and Paradis MR (2003) Assessment of clinical characteristics, management practices and activities of geriatric horses. *Journal of the American Veterinary Medical Association,* 223(1), 99–103.

14 Dental Imaging

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Introduction

Imaging is a widely used and constantly evolving diagnostic aid in many spheres of equine clinical practice and is particularly indicated for the investigation of disorders of the head, because some of its components, including the teeth, are relatively inaccessible to direct visual or manual evaluation. It has been recognized for many years that conventional radiography can make an important contribution to the demonstration of certain dental conditions, particularly those affecting the mandibular and maxillary cheek teeth.[1–16] Furthermore, developments which have led to increased efficacy and reliability of sedative drugs now permit the application of a wider range of radiographic procedures in standing animals and have led to significant improvement in our diagnostic capability.[13–16]

Although basic X-ray units of the type available in most general practices are capable of producing images of acceptable quality, practitioners are often deterred from using radiography routinely in equine dentistry because of a perception that procedures are technically demanding and the anatomical complexity of the area presents a challenge to radiological interpretation. However, the combination of air spaces and mineralized tissue in the equine head produces excellent radiographic contrast, which allows demonstration of relatively minor gross pathological changes, provided that suitable projections are selected and accurately executed. Alterations in the number, size, shape and configuration of the teeth can be identified, together with disruption of skeletal components and abnormal proliferation or destruction of bone. Accumulation of fluid or soft-tissue material in the paranasal sinuses and nasal airways may also be detected.

The perceived limitations of conventional radiography have prompted consideration of alternative imaging techniques. Scintigraphic investigation is now well established as a complementary technique to radiography for the localization of subtle osseous or vascular changes associated with dental disease[11,17–19] and has been shown in one study to be more sensitive than radiography, although less specific, for the diagnosis of disorders of the cheek teeth.[19] Facilities for such investigations are now widely available to equine practitioners. Computed tomography (CT) generates high-quality images in multiple planes, but depends on the availability of expensive and sophisticated equipment and has the clinical disadvantage of requiring prolonged general anesthesia. This imaging modality undoubtedly provides excellent diagnostic information and is now routinely used in certain specialist imaging centers.[20–25] Practical application of the technique and image interpretation require specific training, which normally falls within the remit of specialist diagnostic imagers and is currently beyond the scope of the average equine practice. High-resolution ultrasound equipment, however, is now available in most equine practices, and examinations can be performed with minimal interference to the patient at a low cost. Although this procedure has limitations in anatomical regions in which gas and bone predominate, it may have a place in the evaluation of dental disorders associated with soft-tissue swelling or major disruption of alveolar bone.[11] Magnetic resonance imaging (MRI)[25] is a powerful diagnostic tool for investigation of soft tissues, particularly those of the nervous and locomotory systems, but limitations in its applications for structures containing mineralized material and gas mean that the technique does not lend itself immediately to dental imaging.

Despite the increasing popularity of alternative modalities, radiography remains the imaging procedure most commonly used for the investigation of dental disease in horses. With knowledge, care and experience, valuable clinical information can be obtained from radiographs in a wide range of disorders. The main objective of this chapter is to assist the reader to acquire the skills to obtain it.

Radiographic techniques

Equipment

X-ray machines

Any type of X-ray unit can be used for equine dental radiography. Generally speaking, exposure requirements are not high, so even low-output portable machines are suitable. Of greater importance is the system used for mounting or suspension of the X-ray tube, because it is usually necessary to carry out dental radiography with the horse standing. A facility must therefore be provided for directing the horizontal X-ray beam at high level. It is also helpful if the tube head can be moved through a range of angles in order to permit a

variety of oblique projections. The most effective arrangement is a telescopic ceiling mount with a range of vertical travel between 75 and 200 cm and a tube connection which allows both vertical and horizontal rotation through 180°. A horizontal travel facility in two directions is desirable but not essential. Column-mounted X-ray tubes, either fixed or mobile, tend to be less convenient to manipulate and often have limited facilities for tube rotation and angulation. In this case, positioning of the patient becomes more critical, and sufficient space must be available around the machine to allow the horse to stand at a variety of angles to the X-ray beam. It is a great advantage if the light-beam diaphragm unit is swivel mounted, so that it can be adjusted to provide accurate primary beam collimation in any plane. Whenever possible, equine dental radiography should be carried out in conditions of low environmental light intensity. This maximizes visibility of the margins of the light beam, making accurate positioning and collimation easier.

Imaging systems

It is important to use film/intensifying screen combinations appropriate for the radiographic examination being carried out. Rare-earth intensifying screens are now in general use in veterinary practice and are available in a wide range of speeds and detail. It is important to choose film which is compatible with the type and speed of the screens being used. For most equine dental examinations involving the cheek teeth, medium-speed and medium-detail screens give acceptable results (e.g. Lanex Regular (Kodak Ltd) or FG8 (Fuji Ltd). If a high-output generator (>300 mA) is available, slower speed, finer detail imaging systems can be considered. Very fast screens should be avoided, not only because detail and contrast are sacrificed, but also because exposure factors are more critical and may be difficult to select reliably. For the incisors and canines, intra-oral examinations of the cheek teeth and the full dentition of young foals, a fine-detail system is desirable, for example, MinR (Kodak Ltd), which uses a single screen and film emulsion, or FG3 (Fuji Ltd), which is a high-detail conventional system.

In human medicine, computed radiography is rapidly superseding conventional imaging systems and processing. Such facilities are also now becoming available in veterinary clinical institutions and even larger equine practices.[26] Automated exposure selection, the facility to manipulate digitized images post processing and ease of storage and transmission have clear advantages, but the higher demands for accurate projection selection, positioning, centering and collimation will represent a significant challenge to the equine dental radiographer. In addition, flexible imaging plates suitable for intra-oral work are not yet available.

Accessory equipment

Grids. It is rarely necessary to use a grid for equine dental radiography. This is because there is little bulky soft tissue in the regions under investigation, so that the amount of scattered radiation produced is small. The use of grids in standing animals should be positively discouraged, because of the increased exposure requirements and difficulties associated with alignment for oblique projections. The only examination in which sufficient scatter is produced to possibly justify the use of a grid is the ventrodorsal/dorsoventral view of the caudal arcades, where the great thickness of the mandibles must be penetrated in addition to the partially superimposed cheek teeth.

Cassettes. For external placement and intra-oral positioning for the incisor and canine teeth, any type of rigid film cassette can be used, but lightweight plastic construction is preferred for ease of handling. If the entire arcades of cheek teeth and adjacent facial structures are to be included on a single film, a large cassette (35 × 43 cm) is needed. To obtain intra-oral radiographs of individual cheek teeth, it is necessary to place the film far back in the oral cavity, and the only feasible way of doing this is to use a flexible film package or cassette. The use of human dental film packs has been described in detail,[9] but they have the disadvantage of being too small to demonstrate more than one or two teeth on each radiograph. Plastic envelope cassettes containing either one or two intensifying screens mounted on firm but slightly flexible card are available commercially in a variety of sizes, ranging from 13 × 18 cm upwards (e.g. CEA detail; CEA Sweden). They are suitable for equine intra-oral use, but there is a risk that radiographic sharpness may be marred by less than perfect film/screen contact. Their standard rectangular shape and relatively thick seams present some limitations to the anatomical areas which can be included on the radiograph. An alternative is to improvise cassettes of a more suitable shape for the equine oral cavity (i.e. 10 × 25 cm approx.) by double wrapping the film and card-mounted intensifying screen(s), cut down to size, in closely fitting light-proof bags. The most suitable material is heavy-duty black polythene, the edges of which are closed with light-proof adhesive tape. The inner bag, which remains open at one end, fits the screen(s) and film tightly. The outer bag is very slightly larger and is slipped over the inner bag from the open end. The outer bag is then temporarily sealed with tape which can easily be opened to remove the film for processing. Again, the weakness of this system is poor film/screen contact (see Fig. 14.24B). Films in flexible cassettes are susceptible to pressure damage, so care must be taken when removing them from between tightly clenched teeth.

Cassette holders. For dental radiography of standing horses, it is essential to use some kind of cassette holder whenever possible. The potential need for a variety of oblique projections means that a flexible and readily adjustable system is required. In this respect, the standard vertical suspension linked to the X-ray tube in many fixed units may not be entirely suitable, particularly if the cassette holder is not capable of a full range of independent movement. A simple method of suspending the cassette in a vertical position

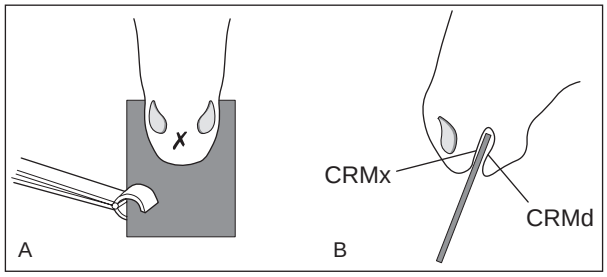


Figure 14.1. Diagrams showing relative positions of cassette and X-ray beam for (A) dorsoventral (DV) and (B) ventrodorsal (VD) intra-oral radiographs of the incisors and canines. X = centering point; CRMx = central ray for DV projection; CRMd = central ray for VD projection.

while allowing free movement is to place it in a fabric bag hanging from a mobile drip stand. Adjustable ties at each side allow varying heights and angles to be used (Fig. 14.2). A standard long-handled holder large enough to accommodate a 35 × 43 cm cassette should be available if ventrodorsal or dorsoventral radiographs of standing animals are to be attempted (Fig. 14.5). Alternatively, if dental examinations are performed frequently, it is worth considering construction of a specially designed head-stand, which can be used to support the cassette for dorsoventral views.[15] The small rigid cassettes used for intra-oral radiography of the incisor dental arcades can conveniently be held at a distance by grasping them with long-handled hoof trimmers or farriers' pincers (Fig. 14.1).

Positioning aids. Some form of support is useful for the drooping heads of heavily sedated horses. Swaying movement is reduced and the head remains at a constant level. A comfortable cushion is simply provided by placing a foam pad on a stool or table, or a custom-made head-stand can be used.[15] For open-mouth projections, a suitable gag or a series of 10 cm lengths of PVC drainpipe (7.5, 9 and 11.5 cm diameter) should be available as well as cotton or nylon ropes for use as snares to steady the horse's head. Positioning aids, such as sandbags and radiolucent foam pads, will be needed for radiography of recumbent patients.

Labelling. All films should be labeled permanently with the identification of the patient and date of radiography. Markers suitable for indicating sides, projections and anatomical landmarks should be available.

Radiation protection

Use of a horizontal or near-horizontal X-ray beam at high level increases the risk of radiation exposure to the upper bodies, heads and necks of human attendants. Obviously, generally accepted protection practice must be observed,[27,28] but special precautions may also need to be taken. Conscious animals should be restrained by one person only, from a position which allows them to be at least 1 m and preferably 2 m from the nearest margin of the primary beam. All persons present during radiography should wear a body dosemeter under the protective lead gown[27] and those whose hands are at risk of being closer than 2 m from the source of X-rays must be provided with protective gloves and sleeves.

Cassettes and holders must be handled by members of staff trained in the techniques being used. For those required to undertake such procedures regularly, consideration should be given to providing extremity dosemeters and thyroid protection. Should an occasion arise when the need to hold a cassette is unavoidable, the largest size available (35 × 43 cm) should be used and held at arm's length by the corners of the short side. The X-ray beam must be closely collimated and centered as far as possible from the hands.

Restraint and setting up

It may be possible to radiograph quiet, sensible horses in the standing position without the use of pharmacological restraint, but it is essential that they are co-operative enough to keep still for several minutes in a predetermined position while equipment is moved around them. They must also stand voluntarily with the head low enough to accommodate the range of travel of the X-ray tube. It is not acceptable to use close manual restraint to retain the head in the required position. It has been suggested that blindfolding may aid restraint.[15]

Sedative drugs are now used routinely for restraint of horses for clinical examinations, including radiography. Care should be taken to avoid overdosing, when there is a tendency for the head to be held too low and postural instability may predispose to sudden movement. Radiography is often carried out in conjunction with other clinical interventions requiring sedation. For obvious reasons, it is best if the examination is done before the animal is disturbed by any invasive procedure such as rasping or direct sinus endoscopy.

Manual restraint should be confined to control the head and arranged so that a safe distance can be maintained between the closest part of the holder's body and the nearest margin of the primary beam. A simple rope or webbing halter without metal components should be used. In some circumstances it may be necessary to raise and extend the head manually by placing a snare around the muzzle, which must be long enough to allow manipulation from a distance of at least 1 m.

To obtain radiographs of the exposed crowns of the cheek teeth, it is necessary to obtain oblique views with the mouth open. This can be done by using a Butler's gag or by placing PVC pipe between the incisor teeth.[15,16]

To set up the tube and film cassette, two people are needed. The radiographer must be clearly aware of the exact projections and centering points required and position the X-ray tube accordingly. The introduction of digital imaging systems will demand a particularly high level of skill in this respect. An assistant, under the guidance of the radiographer, should then position the cassette. At this stage, it is often helpful if the person restraining the horse checks for alignment in the third dimension from a position at right

angles to the primary beam. Immediately before exposure, all participants must retreat as far as possible from the path of the X-rays.

The increasing tendency to perform dental procedures in the standing horse has reduced the demand for radiography under general anesthesia. While this trend has led to innovations in the application of standard techniques in the standing animal, the need to radiograph anesthetized horses has not been entirely eliminated, particularly when intra-operative or immediate postoperative examinations are indicated. Preoperative radiography under general anesthesia should be encouraged as an alternative to standing examinations whenever possible, because there is no doubt that radiographic accuracy is improved and radiation exposure to personnel minimized. For laterally orientated projections, image quality is optimal if the patient lies with the affected side on the cassette, although this entails the inconvenience of turning the patient before further intervention can take place. As it is not usually feasible to re-turn the animal during or immediately after surgery, intra- and postoperative radiographs are usually taken in less than ideal circumstances, with the affected side uppermost. When ventrodorsal views are required, it may be necessary to withdraw the endotracheal tube. The design of some operating tables makes it difficult to obtain a full range of movement of the X-ray tube around the patient's head, thus restricting projection options. In such cases, it may be necessary to devise some form of extension platform onto which the head can be drawn to give surrounding unobstructed space.

Projections

Introduction

In equine dental radiography, selection of the correct projection is critical. While the incisor arcades and canines can be demonstrated with ease, the anatomical conformation and location of the cheek teeth present a major radiographic challenge. Various projections and techniques have been reported in the literature.[2,6,9,15,16] The standardized projections described below are in regular use. However, it is sometimes necessary to resort to tube and film positions contrived specifically to show a particular lesion. Such projections are referred to as 'lesion-orientated obliques.'

Incisors and canines

Standardized projections (Fig. 14.1). The film cassette is positioned intra-orally, as far caudally into the mouth as possible. For the upper arcade, the primary beam is directed from the dorsal aspect and centered on the midline of the muzzle between the nostrils at an angle approximately 20° rostral to the perpendicular plane. This angle matches approximately the lie of the incisors in the premaxilla and therefore helps to compensate for image distortion caused by their curved conformation. For the lower arcade, the X-ray beam is directed ventrally and centered on the chin, using a similar rostral beam angle.

Other projections. Lateral radiographs tend to be relatively unhelpful because of superimposition of the right and left sides, but may be indicated in cases where there is gross distortion of the incisor arcades (e.g. brachygnathia)[29] or to check fracture displacement and alignment after repair.[30] Lesion-orientated oblique projections may occasionally be indicated for demonstration of individual incisors and for the canines.

Cheek teeth

Lateral projection (Fig. 14.2). Although superimposition of the right and left arcades prevents identification and evaluation of individual teeth, true lateral radiographs are of value for demonstrating associated disease processes in the maxillary bones and paranasal sinuses. This projection is easily obtained in the standing horse. The cassette is positioned vertically on the clinically affected side, parallel with the outer contour of the face and as close as possible to it. The whole facial area should be included, from just above the eye caudally to the mid-diastema rostrally and the X-ray beam centered at the level of the facial crest, on its rostral extremity. Slight adjustments may be required to allow for conformational variations and aging changes. To avoid unnecessary distortion, it is essential that the primary beam is exactly perpendicular to the centering point in both rostrocaudal and ventrodorsal planes. This means that individual adjustments

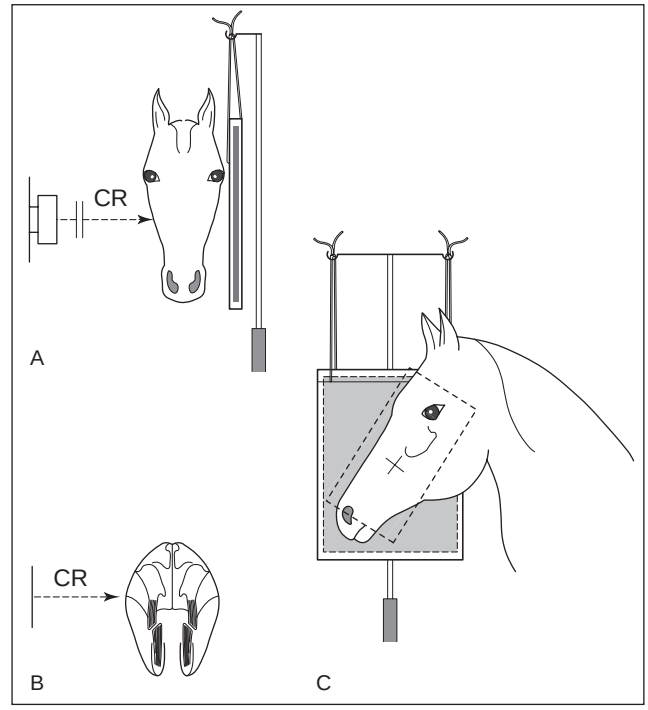


Figure 14.2. Diagrams showing a system for cassette suspension (see text) and X-ray beam/film orientation for lateral radiography of the cheek teeth in standing horses. (A) Frontal view, (B) lateral view and (C) rostrocaudal view represented by a section through the head at the level of PM4 (1–4 08s). X = centering point (1–2 cm rostral to facial crest at the level of PM4 (1–4 08s).

need to be made to accommodate the angle of the head. To achieve accurate collimation, the light-beam diaphragm unit should be rotated so that alignment of the X-ray beam conforms to the angle of the head (Fig. 14.2). If selected sections of the dental arcades are required, collimation and centering may be modified accordingly. To obtain a lateral projection in a recumbent horse under general anesthesia, the muzzle should be raised on a pad or sandbag, so that the sagittal plane is parallel to the film.

Indications for lateral projections of the mandibular arcades occur infrequently. When required, the X-ray beam should be centered dorsoventrally at the level of the commissure of the lips on a rostrocaudal point over the region of interest. Separate exposures are needed for the rostral and caudal teeth, because of the difference in overall tissue thickness.

Standard laterodorsal–lateral and lateroventral–lateral oblique projections (Fig. 14.3). Oblique projections provide the most convenient method for producing radiographs of the right and left arcades of the cheek teeth separately. For the maxillary arcades, in order to minimize distortion of the dental images, whenever possible, the laterodorsal–lateral oblique should be used, with the affected side next to the film. For the anesthesised horse, the cassette is positioned as for the lateral projection, but it will need to be offset ventrally, so that it is aligned with the emergent beam (Fig. 14.3A). The X-ray beam is angled at approximately 30° dorsal to lateral. Narrower heads will require slightly larger angles (up to 40°). As for the lateral projection, if the whole arcade is to be included, the centering point is the distal extremity of the facial crest.

For the mandibular arcades, the preferred projection is the lateroventral–lateral oblique (Fig. 14.3D). The centering point, exposure factors and primary beam margins are selected according to the tooth or teeth under investigation. As the mandibular arcades are closer together than their maxillary counterparts, the angle of the beam to the vertical needs to be increased. For heads of average conformation, and the central portion of the arcade, an angle of 45° is suitable. A slightly increased angle of about 50° may be needed for narrower heads and the more rostral teeth [PMs

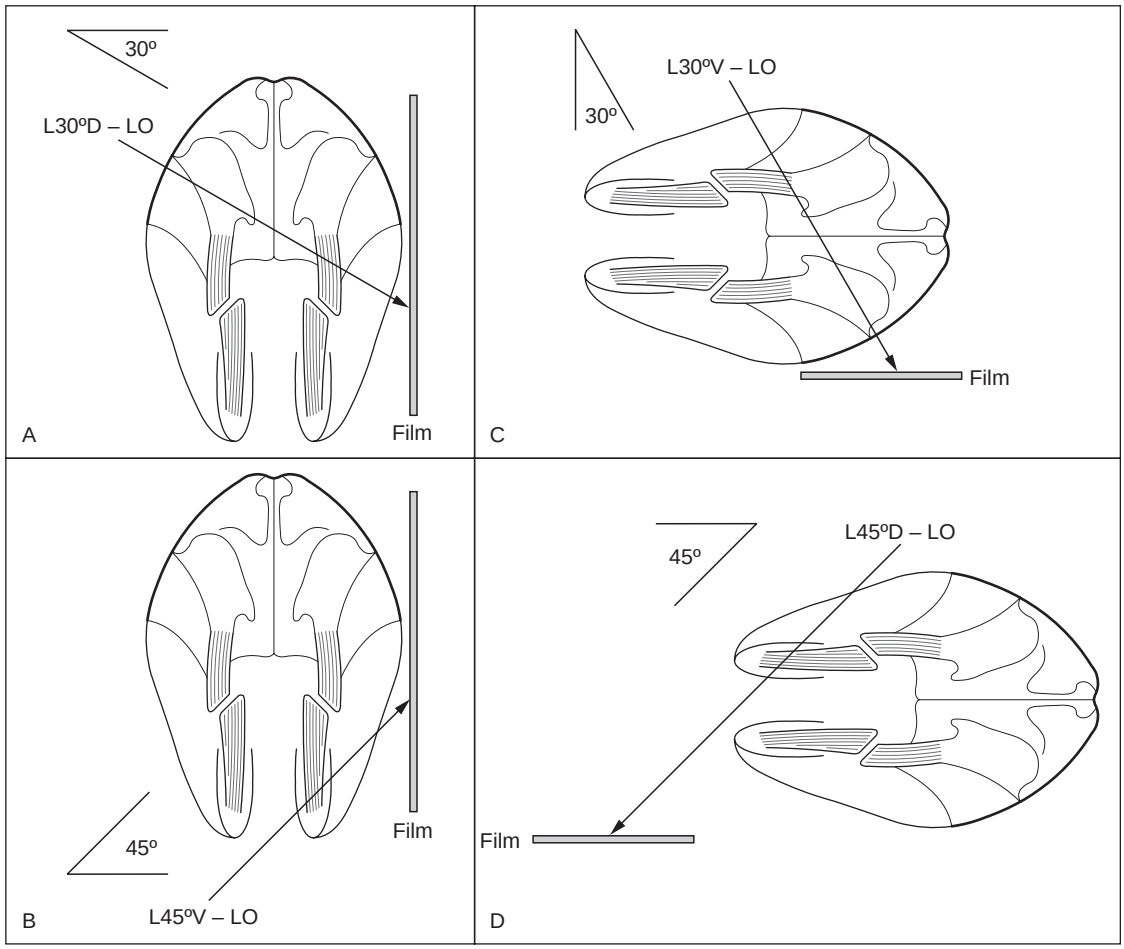


Figure 14.3. Diagrams of a section of the head through PMs4 (1–4 08s) showing position of head, cassette and X-ray beam for standard oblique projections to show the roots and reserve crowns. D = dorsal; L = left; V = ventral. (A) and (B) show horse standing and maxillary (A) and mandibular (B) teeth closer to film (preferred positioning). (C) and (D) show horse recumbent and maxillary (C) and mandibular (D) teeth farther from film.

2, 3 and 4 (3s and 4s 06s–08s)], while for the more caudal teeth [Ms 2 and 3 (3s and 4s 10s and 11s)] a smaller angle of about 35°–40° can be used. The increased beam angles mean that the distance at which the cassette must be offset is also greater (Fig. 14.3D).

These projections can be difficult to obtain accurately in the standing animal, because without the reference point of a table-top, the vertical and horizontal angles of the central ray must be assessed simultaneously by eye. Minor deviations from the required angles will produce marked image distortion with superimposition of adjacent teeth, which obscures the root and alveolar structures and may obliterate diastemata.

If it is not possible to arrange for the side to be examined to be next to the film, for example, limitations in maneuverability of equipment or intra- and postoperative examinations under general anesthesia, acceptable, but incomparable images of the arcade closer to the X-ray tube can be obtained by using the same beam angles in the opposite direction; i.e. dorsolaterally from a lateroventral direction for the maxilla and ventrolaterally from a laterodorsal direction for the mandible (Fig. 14.3C,D).

Laterodorsal–lateral and lateroventral–lateral projections with open mouth (Fig. 14.4). These projections have been developed to give improved information about the reserve and exposed crowns of the cheek teeth by displacing the images of the teeth in the opposite ipsilateral arcades.[15,16] A perfectly relaxed patient is required, so that a mouth gag will be accepted without resentment. Either a Butler's gag or a 10 cm length of PVC piping of appropriate diameter can be used to hold the mouth open to its maximum extent.

For a general view of the crowns and roots, the standard oblique angles are used (see above and Fig. 14.4A). However, to penetrate the dense dental tissue adequately, these projections require increased radiographic exposure, so detail of the surrounding maxillary and mandibular bone and alveoli is lost.

For the exposed crowns and occlusal surfaces, a different approach is used, with the X-ray beam angled in the opposite direction (Fig. 14.4B). Optimal angles for these projections

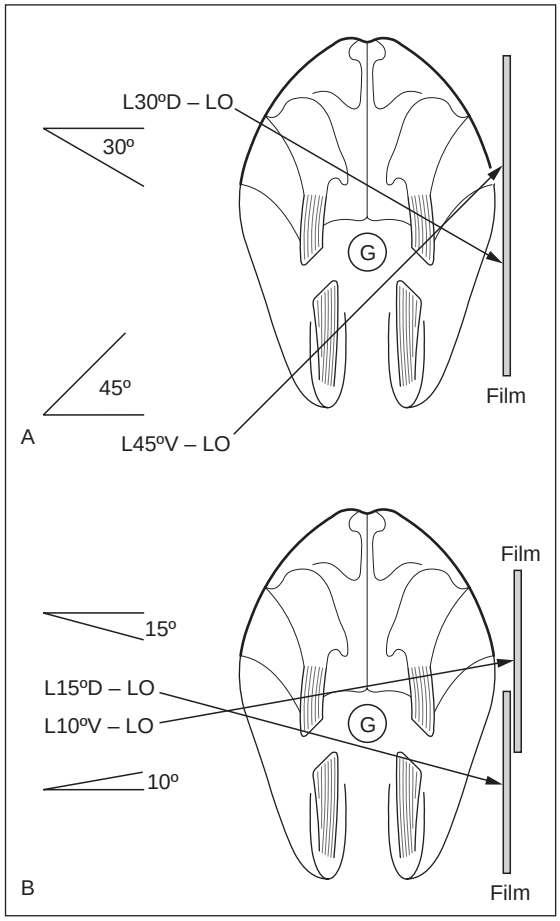


Figure 14.4. Diagrams of a section of the head through PMs4 (1–4 08s) showing position of head, cassette and X-ray beam for open-mouth oblique projections for reserve crowns and roots; horse standing. D = dorsal; L = left; V = ventral. (A) Standard 30° and 45° oblique projections for maxillary and mandibular reserve and exposed crowns; (B) latero-15°-ventral–lateral oblique for maxillary exposed crowns and latero-10°-dorso–lateral oblique for mandibular exposed crowns.

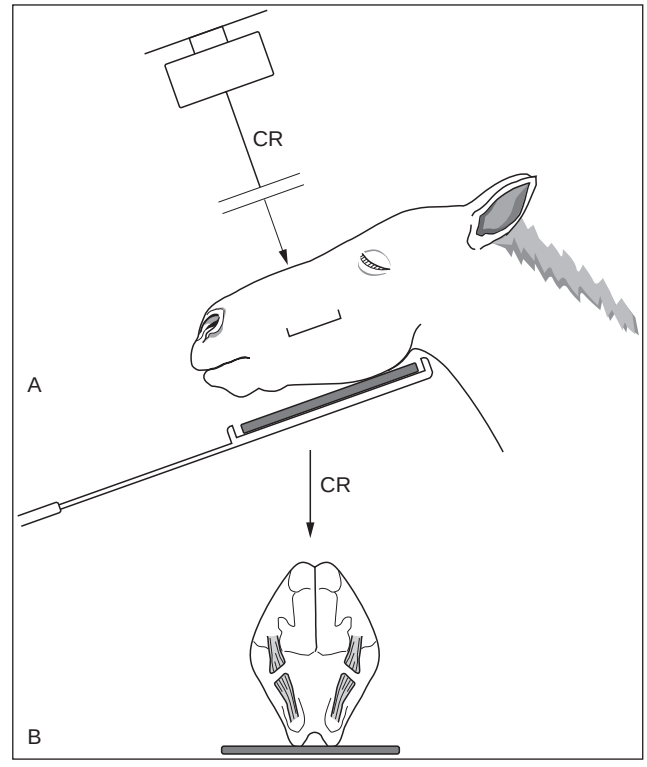


Figure 14.5. Diagram showing position of standing horse, cassette and X-ray beam for a dorsoventral projection. Ideally, the long handled cassette holder should be replaced by a custom-made head-stand. CR = central ray. (A) Lateral view; (B) rostrocaudal view represented by a section of head at the level of PMs4 (1-4 08s).

have recently been established.[16] For the maxillary teeth, with the affected side next to the film, the X-ray beam is angled approximately 15° ventrally (latero-15°-ventral–lateral oblique). For the mandibular teeth the angle is 10° dorsal (latero-10°-dorsal–lateral oblique) (Fig. 14.4B). Exposures do not need to be as high for this region.

Ventrodorsal and dorsoventral projections. These projections are of little value for demonstration of the teeth, because the upper and lower arcades are partially superimposed and the dense bone of the mandible is virtually impenetrable. However, important information can be obtained about peripheral structures such as the nasal chambers, paranasal sinuses and surrounding alveolar bone. For standing horses, the dorsoventral projection is usually considered more convenient (Fig. 14.5), although it may be difficult to obtain sufficient extension to avoid slight obliquity on the resulting image. Accommodation can be made for this restriction by angling the X-ray beam so that it is perpendicular to the cassette. There is also a limit to the caudal extent to which the cassette can be placed. Ventrodorsal views are possible, provided that suitable cassette holding facilities are available and are to be preferred if clinical interest is centered on the more caudal teeth. When setting up for this projection, meticulous care must be taken to ensure that the head is absolutely straight. Even slight tilting of head, X-ray tube or cassette produces sufficient asymmetry to preclude a meaningful comparison of right and left sides. Ideally, ventrodorsal radiographs should be obtained under general anesthesia with the horse in dorsal recumbency, when the neck can be fully extended and positioning more efficiently controlled. For evaluation of midline structures, the endotracheal tube must be removed prior to exposure. The centering points for dorsoventral and ventrodorsal radiographs are on the midline at a rostrocaudal level determined by the region of interest.

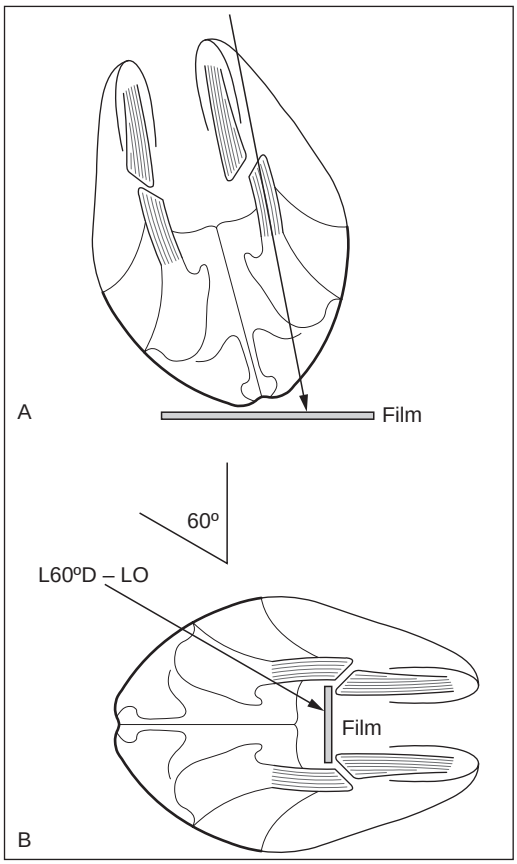


Figure 14.6. Diagrams of a section of the head through PMs4 (1–4 08s) showing positions of anesthetized horse, film/cassette and X-ray beam for (B) intra-oral view of individual maxillary cheek teeth and (A) ventrodorsal view with offset mandible and slight rotation of the head to show the abaxial margins of the maxillary cheek teeth (D = dorsal; L = left; V = ventral).

Ventrodorsal and dorsoventral projections with offset mandible (Fig. 14.6A). Using this projection, it is possible to demonstrate the buccal surfaces of the cheek teeth, together with their abaxial alveoli and the adjacent maxillary bone. It can be performed on either sedated or anesthetized horses. The mandible, which is slightly mobile, is displaced to the contralateral side and its position maintained by applying tension to a snare placed around the diastema. The X-ray beam is tightly collimated to minimize scattered radiation. For the rostral premolars, it may be necessary to rotate the head slightly away from the affected side. This projection is particularly useful for demonstrating subtle alveolar disease and maxillary osteitis.

Intra-oral oblique projection for maxillary teeth (standardized) (Fig. 14.6B). A system for obtaining intra-oral projections of the apices and reserve crowns has been described,[9] but the indications for its use will have declined since the introduction of the standardized open-mouth oblique views described above. Envelope-wrapped human dental film 5 × 7.5 cm is recommended. The anesthetized patient is positioned in lateral recumbency with the side to be examined uppermost. The film is placed in the oral cavity parallel to the hard palate at the level of the tooth of interest. The location is determined by reference to a lateral radiograph on which the distance from the rostral aspect of the first incisor to the desired centering point is measured. The film is introduced taped to a perspex ruler, so that the required distance can be confirmed. For most teeth, the X-ray beam can be directed at an angle of 60° to the horizontal, but for the longer reserve crowns in 2-year-old horses increased incident angles of 70°–80° were required. The centering point is at or up to 6 cm dorsal to the facial crest, depending on the length of the tooth being radiographed. The disadvantage of this technique is that if the affected tooth is not identified beforehand, several exposures may be required.

Lesion-orientated projections. Projections contrived to demonstrate specific lesions must be set up with care and if an oblique X-ray beam is needed, the required angle will be judged visually or by palpation. They may be performed externally, or intra-orally, using custom-made cassettes.

The indication for these projections is usually a clinically identifiable abnormality, such as swelling or obvious disruption of teeth or bone. It is important to arrange the X-ray beam and film so that the area to be examined is shown to best effect, which is usually obtained by placing the lesion on the 'skyline.'

Exposure selection

The choice of exposure factors will depend on the output of the X-ray machine and speed of imaging system in use. To maximize radiographic contrast between mineralized tissue and adjacent air spaces, a relatively low tube voltage of the order of 55–60 kV is desirable, but with low-output machines, the consequent need to increase exposure time may be unacceptable, because of the risk of movement blur. Compromise will therefore be necessary. When choosing exposure factors, consideration must be given to the relative radiographic density of the structures under investigation, particularly the cheek teeth. Lower exposures are needed to show the root apices and contents of the paranasal sinuses and nasal chambers than for demonstration of the crowns. Therefore, for a full examination it may be necessary to take two radiographs of the same area using different exposure factors.

Tube–film distance is the factor which requires special consideration in equine dental radiography, particularly when setting up for oblique views, when it may not be easy to obtain reliable measurements. It is important that distance is consistent within a few centimeters, because with high-speed imaging systems, small variations produce a noticeable effect on exposure. To limit the effects of magnification, a tube–film distance of at least 1 m is desirable, but to overcome positioning constraints, it may sometimes be expedient to compromise by using shorter distances, in which case the tube output factors will need appropriate adjustment.

Contrast studies

The use of positive contrast to determine the source of draining tracts in the equine head has been illustrated[29] and described in some detail.[11] As such lesions often originate from infected teeth, particularly in the mandible, there may occasionally be an indication for performing these examinations if the tract cannot be related specifically to an abnormal tooth on plain radiographs. The simplest form of contrast study is to place a radiopaque probe as deeply as possible into the tract and obtain radiographs in orthogonal planes, which must be selected carefully in relation to the location of the tract. The alternative is to perform fistulography. Any type of water-soluble iodinated compound may be used. To avoid leakage, injection should be made through a self-retaining catheter with an inflatable bulb (e.g. Foley) and discontinued immediately resistance is felt. Again, two orthogonal projections will probably be required and incremental doses with sequential exposures may be needed.

Quality evaluation

Before attempting to identify pathological changes, it is important that each radiograph is inspected carefully, the quality of the image assessed and any shortcomings which might interfere with interpretation noted. A decision must then be made as to whether the defects present are acceptable in the particular circumstances of the case under investigation, or whether it is necessary to obtain repeat of additional radiographs. Some factors to be considered are listed below.

Exposure and development

In the maxillofacial region, correct exposure is critical for assessing the relationship between mineralized structures and adjacent air spaces. It is important that the contents of the paranasal sinuses and nasal chambers are not overexposed (too dark), when subtle changes in their contents will be obscured. For all projections, overexposure at the periphery of the image is an inherent problem, which is exacerbated along the ventral border of the mandible in oblique views. It may therefore be necessary to use bright-light transillumination to examine these dark areas. Low-intensity environmental light is also helpful. Deliberate overexposure is used for examination of the dental crowns specifically. In these cases, there must be sufficient penetration of dental tissue to show the details of crown structure on the image. Underdevelopment, which is a common problem, even using automatic processing, gives rise to poor radiographic contrast and can be recognized by paleness or uneven blackening of the background of the film.

Centering and field size

A check should be made to ensure that the X-ray beam is centered so that the area of interest is represented on the radiograph without unacceptable distortion and that any relevant adjacent structures are included. The primary beam should be collimated so that all four borders appear on the radiograph.

Positioning

Inaccurate positioning is by far the commonest fault in equine dental radiography, because the procedures are difficult and require skill, experience and patience. Each radiograph should be evaluated critically for positioning faults, and radiographers must always be prepared to repeat exposures.

Artifacts

A search should be made for artifacts which might confuse interpretation. Extraneous marks may be caused by traces of mud or contamination by lotions, ointments or dressings. Images of rope or webbing restraining devices may be visible. Pressure marks on films from flexible intra-oral cassettes appear as light, unexposed linear streaks (see Fig. 14.7).



Figure 14.7. Dorsoventral intra-oral radiograph of the mandibular incisor and canine teeth of a 5-year-old thoroughbred gelding showing fully erupted Is1 and Is2 (3 and 4 01s and 02s) and partially erupted Is3 (3 and 4 03s). Parts of the periodontal membranes can be seen as narrow radiolucent bands adjacent to the roots of Is1 and Is2 (3 and 4 01s and 02s). Lamina dura is partly visible along the axial rostral alveoli of Is1 and Is2 (3 and 4 01s and 02s). The pulp canals are mostly visible and the outlines of the infundibular spaces can just be traced on the occlusal surfaces, together with thin bands of radiolucent cement. The canines (3 and 4 04s) are well developed and partially erupted; their pulp canals are wide. DIs3 (7 and 8 03s) are retained and represented by small, rootless masses of dental tissue between the crowns of Is 2 and Is3 (3 and 4 02s and 03s). On clinical examination, it was not possible to distinguish with certainty between the partially erupted Is3 (3 and 4 03s) and the retained DIs3 (7 and 8 03s). The irregular white streaks superimposed on the right canine were caused by pressure damage to the envelope wrapped film.

Radiographic anatomy

Deciduous teeth

Incisors [Is 1–3 (5–8 1s–3s)]

Temporary incisors are shell-like in appearance and can be distinguished from their counterparts by the absence of elongated roots (Fig. 14.7).

Premolars [PMs 2–4 (5–8 4s–6s)]

There is no temporary PM1 (wolf tooth). Temporary premolars have short, spicular roots. Figure 14.8 shows that they can be distinguished from developing permanent molars by their greater mineral content, relative lack of internal structure and small or almost radiographically absent alveoli (dental sacs).

Permanent teeth

Incisors and canines [Is 1–3, C (1–4 01s–04s)]

On standardized DV and VD intra-oral radiographs, as shown in Fig. 14.7, the incisor roots and crowns are closely packed. Curvature of the arcade leads to partial superimposition of the roots of Is 2 and 3 (1–4 02s and 03s), but interdental spaces should be visible between right and left Is1 (1–4 01s) and Is 1 and 2 (1–4 02s). Normally, only parts of the periodontal membrane and lamina dura can be seen. On correctly exposed radiographs, the pulp cavities should be clearly defined. In recently erupted teeth, the infundibular spaces can be recognized on the obliquely projected occlusal surfaces and in those which have been longer in wear, traces of cement may be visible as thin, elliptical conical radiodense



Figure 14.8. Lateral radiograph of the facial and mandibular areas of a yearling pony under general anesthesia showing the right and left arcades of cheek teeth partially superimposed. DPMs 2–4 (5–8 03s and 06s) are erupted and in wear; they are short and dense and their spicular root conformation can be seen well in the mandible, despite superimposition of right and left arcades. Ms1 (09s) are well developed, erupted in the maxilla and partially erupted in the mandible. Ms2 (10s) are in a fairly advanced stage of eruption and contrast with the deciduous premolars by their lesser radiopacity, striated pattern and radiolucent dental sacs delineated by lamina dura.

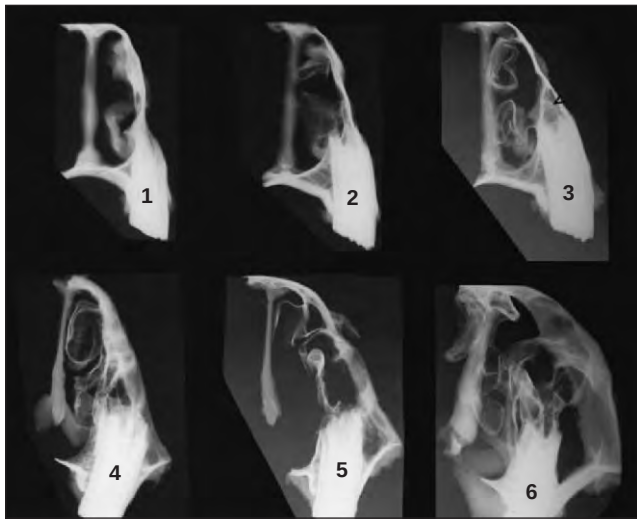


Figure 14.9. Radiographs of slices through the interdental spaces of a specimen of the left facial area and nasal chamber of a 7-year-old horse showing the relationship between PMs 2–4 (2 06s, 07s and 08s) (1–3) and Ms 1–3 (2 09s, 10s and 11s) (4–6) and adjacent structures. The axial radicles of the premolars (06s, 07s and 08s) are in contact with the lateral wall of the nasal chamber and the abaxial radicles are close to the external wall of the maxilla. Parts of the apices of PM4 (208) lie within the rostral compartment of the maxillary sinus (arrow) and the molar roots are closely associated with the sinus cavities.

shadows (see Fig. 14.23). In this projection, the images of the canine teeth are superimposed on the roots of Is3 (1–4 03s) so if specific information is needed about either structure, customized oblique projections should be taken. Although unerupted, canines are usually present and quite well developed in mares.

Maxillary cheek teeth [PMs 1–4, Ms 1–3 (1–4s 5s–8s and 9s–11s)]

General considerations. As disorders of the roots and reserve crowns of the maxillary cheek teeth may be associated with pathological changes in adjacent structures, familiarity with the radiographic anatomy of the whole facial area is essential if subtle radiographic abnormalities are not to be overlooked. Figure 14.9 demonstrates the anatomical relationships of the grinders [PMs 2–4 (206–208) and Ms 1–3 (209–211)] with the supporting maxillary bone and sinus within which they are embedded.

Lateral radiographs. This projection is probably the most useful for overall assessment of the relationships between the cheek teeth and other facial structures, which are illustrated diagrammatically in Fig. 14.10A. With the exception of the ethmoturbinate, all landmarks are paired, but on radiographs, image distortion as a result of beam divergence prevents them from being accurately superimposed. There are also anatomical differences in configuration of the maxillary sinuses between right and left sides. Accuracy of positioning can be assessed by checking the premaxillary borders of the nasomaxillary notch, which should be close together and parallel to each other.

Figure 14.10B–E shows lateral radiographs of horses at various ages. The reserve crowns of the grinders become progressively shorter with advancing age and the shape of the root apices changes from rounded to pointed and eventually spicular. The roots of Ms1 (109 and 209) consistently lie most ventrally, reflecting their early age of eruption. PMs2 (106 and 206) are usually shorter and squarer than the other teeth in the arcade and angled caudally. Ms3 (111 and 211) also tend to be set at an angle to the hard palate, pointing rostrally. Other individual features of dental anatomy are not clearly shown on this projection.

There are marked variations in the size and shape of the paranasal sinuses among individual horses. 'Dish-faced' Arabians have relatively small airspaces, while those in 'Roman-nosed' heavy horses are more capacious. The course of the infra-orbital canal is also conformation related, so the position of the reserve crowns relative to this structure is not a reliable guide to age or dental wear. The rostral walls of the maxillary sinuses are consistently seen at the level of PMs4 (108 and 208) and are usually caudal to the rostral radicles. There is often an indistinct condensation of bone in this area, giving rise to an increase in radiopacity which obscures the apices of PMs4 (108 and 208) and may be difficult to distinguish from pathological proliferation. The position, course and integrity of the bony septa separating the rostral and caudal compartments of the maxillary sinuses are more variable and sometimes differ between right and left sides. They may not be distinguishable from other shelves of bone within the sinuses, but usually traverse them in a dorsal direction, originating from a point between the roots of Ms1 (109 and 209) and Ms2 (110 and 210). The clarity with which the nasal conchae can be seen is highly variable. The ventral conchae are well shown in Fig. 14.10D.

If present, the wolf teeth (105 and 205) can be seen on lateral radiographs (Figs 14.10C, 14.21A, 14.33, 14.35). They vary in dimensions from a few millimeters to about 2 cm and are triangular or trapezoid in shape. In a survey of radiographs of 134 horses, the frequency of occurrence of wolf teeth was 30 per cent, but this may not be a true reflection of their incidence because of the common practice of 'therapeutic' removal.[7]

Standardized oblique radiographs (mouth closed). Figure 14.11A shows the relationship between individual cheek teeth of the right maxillary arcade on a left dorso-30°-lateral–right lateral oblique radiograph of an 8-year-old horse with normal dental roots and reserve crowns. Relative underpenetration of M3 (111) and dense blackening of the maxillary sinus area illustrate the problem of obtaining correct exposure of all teeth in the arcade and the normal sinus airspaces. (A normal M3 (111) is shown more clearly in Fig. 14.27.) The size distribution of the interdental spaces between the reserve crowns is fairly consistent between

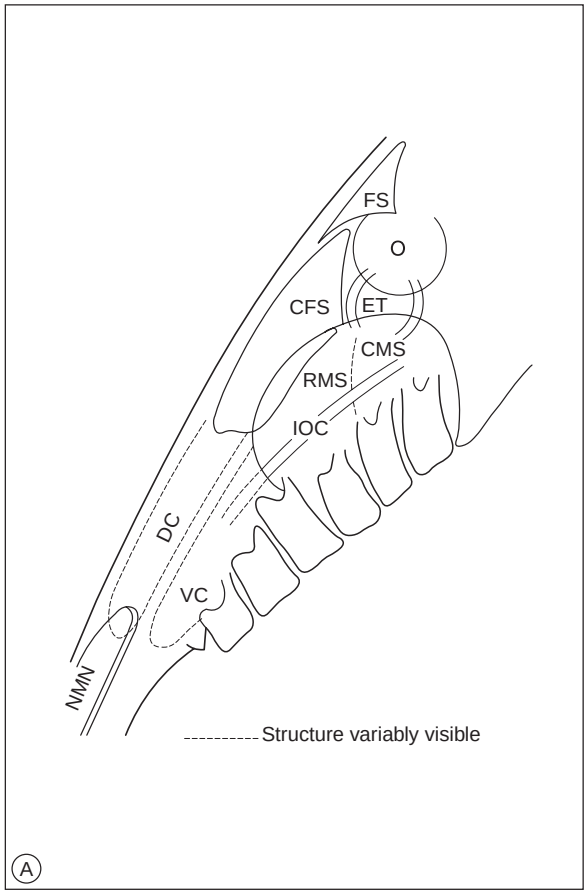






Figure 14.10. (A) Diagrammatic representation of the facial area of an adult horse, indicating the anatomical structures related to the maxillary cheek teeth which may be recognized on lateral radiographs. FS = frontal sinus; CFS = conchofrontal sinus; RMS = rostral compartment of maxillary sinus; DC = dorsal concha; VC = ventral concha; O = orbit; ET = ethmoturbinate; IOC = infra-orbital canal; NMN = nasomaxillary notch. (B)–(E) Lateral radiographs of the facial areas of thoroughbred or part thoroughbred horses at the ages of (B) 3 years.

Continued

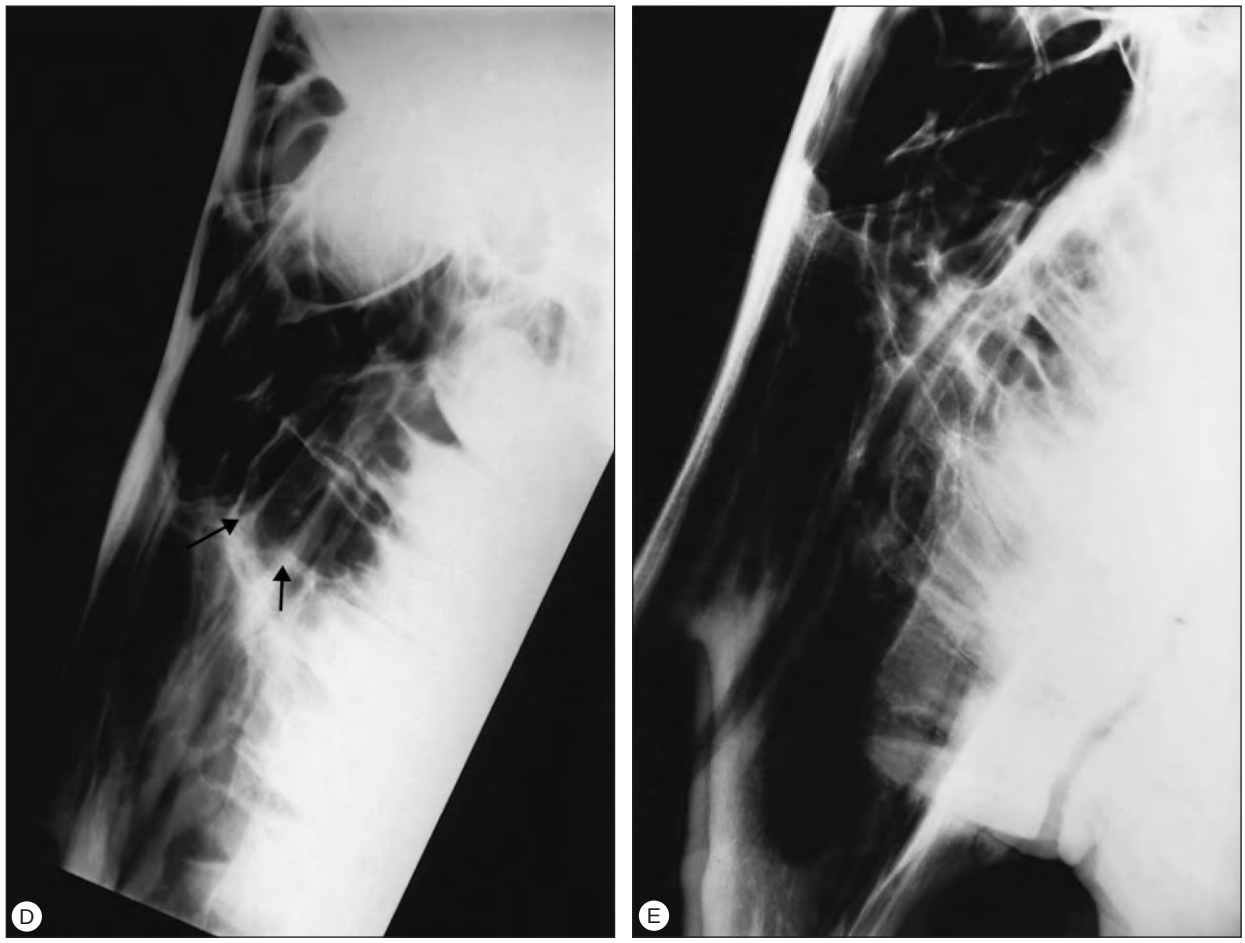


Figure 14.10, cont'd, Lateral radiographs of the facial areas of thoroughbred or part thoroughbred horses at the ages of (C) 6 years and (D 9 years (ventral conchae arrowed). Lateral radiographs of the facial areas of thoroughbred or part thoroughbred horses at the ages of (E) 13 years. Small wolf teeth [PMs1 (1 or 2 05s)] can be seen rostral to PMs2 (1 and 2 06s) in (C) and (E).

horses, but the width of the spaces is variable (see Figs 14.11 and 14.29B). Inconsistent demonstration of the lamina dura and periodontal membranes is a normal feature of this projection. These structures are visible only where the X-ray beam passes directly through them. Thus, their absence or partial absence is not necessarily an indication of alveolar disease. The line of the occlusal surface of the arcade is better demonstrated on open-mouth projections (see below). The rostral wall of the maxillary sinus should be clearly delineated and its site of origin in relation to the radicles of PM4 can normally be identified. The intercompartmental septum is less well defined but may be partly represented by a highly radiodense linear shadow extending dorsally from a point adjacent to the root of M2. The images of the infra-orbital canals are widely separated, but the one on the contralateral side may not be visible because of overexposure.

A left latero- 30° -ventral–right lateral oblique radiograph of the same arcade of the same horse is shown in Fig. 14.11B, which illustrates the characteristic elongation of the dental images which is a feature of this projection. The image is slightly magnified, but sharpness usually remains fairly good, although details of root and alveolar anatomy tend to be less clear. Despite its limitations, this projection is adequate and convenient for post-extraction evaluation in anesthetized horses (see Fig. 14.27).

Mandibular cheek teeth (Fig. 14.12) have several characteristics which distinguish them from their maxillary counterparts. Young roots are tubular in appearance, with open apices. They are surrounded by large, rounded radiolucent areas which represent the soft-tissue content of the dental sacs. These 'eruption cysts' are most prominent around PMs 2–4 (307 and 308) between the ages of 2 and 4 years. The mandibular cortex below them tends to become thin and bulges ventrally as shown on the latero-45°-ventral–lateral oblique radiograph in Fig. 14.12A. As the teeth mature and migrate orally, the root apices become more pointed, but the pulp canals remain open. The periodontal membrane and

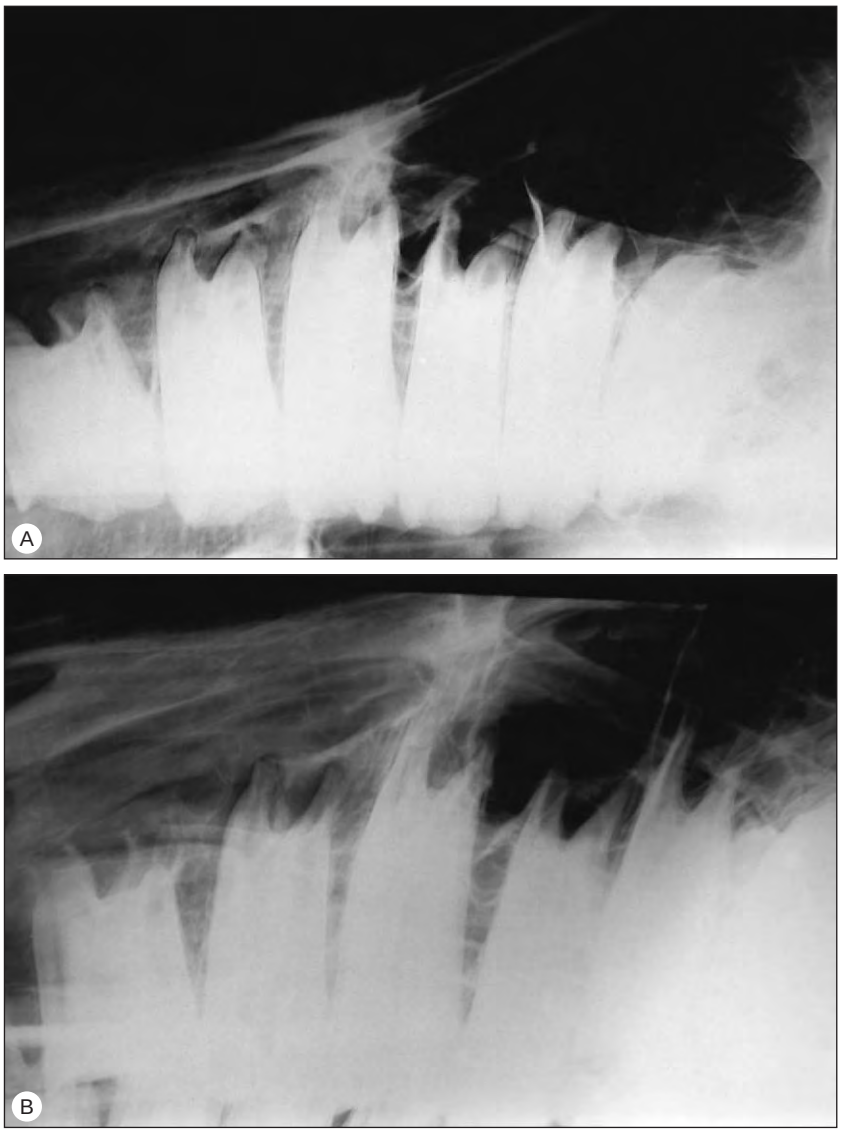


Figure 14.11. Standardized oblique radiographs of the normal right maxillary arcade of an 8-year-old hunter. (A) Left dorso-30°-lateral–right lateral oblique projection (right side closer to film). M3 (111) cannot be evaluated properly because of relative underexposure. The root apices are variable in shape, but mostly pointed, except rostral PM2 (106) and M3 (111). The interdental spaces are relatively large between PMs 2 and 3 (106 and 107), PMs 3 and 4 (107 and 108) and PM4-M1 (108 and 109) and much narrower between Ms 1 and 2 (109 and 110) and Ms 2 and 3 (110 and 111). Parts of the lamina dura can be seen around the root apices and reserve crowns. In no tooth are they complete. The rostral border of the maxillary sinus can be seen through a hazy radiopaque area and arises at the caudal tip of the rostral radicle of PM4 (108). A vertically orientated, sharp linear opacity superimposed on the rostral radicle of M2 (110) and the infra-orbital canal may represent a part of the septum separating the rostral and caudal compartments of the maxillary sinus. (B) Left latero-30°-ventral–right lateral oblique projection (right side farther from the film) showing the effects of magnification and distortion. Compared with (A), the dental images are longer and the roots appear more spicular. The distal reserve crowns are superimposed on their contralateral counterparts. Note that in this projection, the periodontal membranes and lamina dura tend to be less clearly delineated. The linear shadow, which is probably the intercompartmental septum of the maxillary sinus, is more clearly demonstrated on this view.

lamina dura are often indistinct and incomplete. The gross anatomical effects of aging on the mandibular cheek teeth have been illustrated by radiographs of specimen hemimandibles in two detailed studies.[29,30] Figure 14.12B shows the same projection of a middle-aged horse. M3 (411) is not included. More caudal centering, and higher exposure factors would be required to demonstrate it to best effect. The degree of pointing of the roots of PMs 3 and 4 (407 and 408) and Ms 1 and 2 (409 and 410) reflects the relative ages of the teeth. The distinctive, squarish shape of PM2 (406) is a consistent feature.

Standardized oblique radiographs (mouth open). To show the full length of the crowns, oblique projections using approximately the same angles as for the standard closed-mouth views are used, although to clear the contralateral maxillary arcade completely, laterodorsal angles slightly greater than 30° may be needed. Correctly penetrated images (Fig. 14.13A) show detail of the parallel, linear, striated structure of the crowns and the relationships between them. Interdental spaces are normally present between the reserve crowns, but the exposed crowns should be in contact with each other. Although obliquity of the image produces some distortion of the occlusal surfaces, adjacent teeth should be almost level, giving an uninterrupted appearance to the whole arcade. The distinctive shapes of PM2 (106) and M3 (111) are well shown in this view.

The exact contour of the mandibular occlusal surface and an undistorted representation of the exposed crowns is shown in Fig. 14.13B, which is a left latero-10°-ventral–right lateral oblique projection of most of the right arcade. Minor 'stepping' between adjacent teeth is considered to be within normal limits.

Ventrodorsal radiograph (offset mandible). This projection is illustrated in Fig. 14.14 in which the radiographic

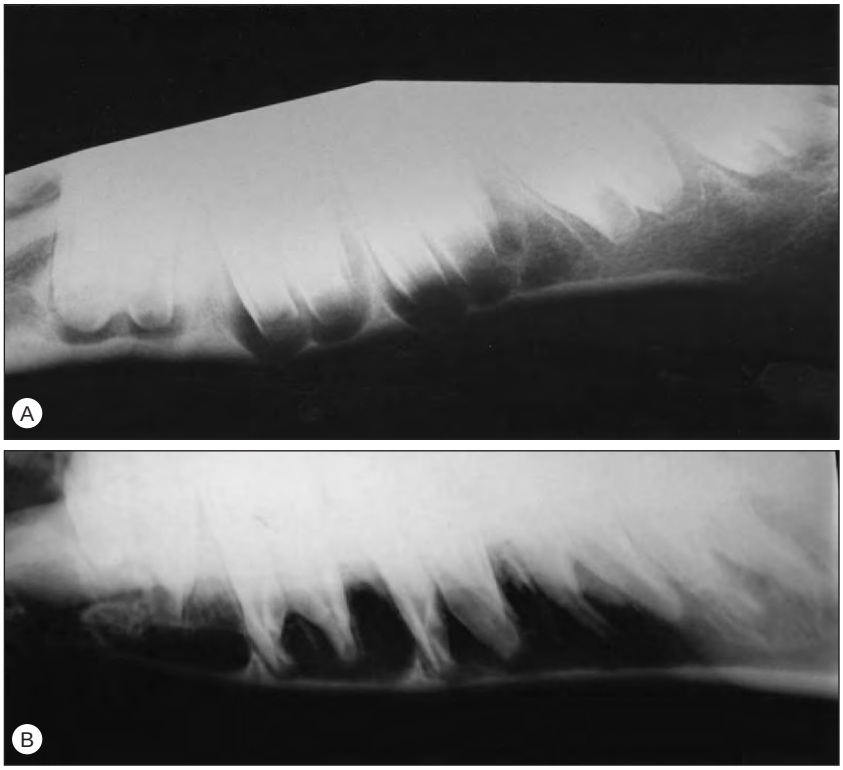


Figure 14.12. (A) Right latero-45°-ventral–left lateral oblique radiograph of the mandible of a 4-year-old Dartmoor pony presented with painless non-discharging swellings on the ventral mandibular border. The roots of the visible teeth [PMs 2–4; Ms 1 and 2 (306–10)] are normal, but the dental sacs of the premolars are large and those of PM 2 and 4 (307 and 308) lie very close to the ventral cortex, which has become thinned to accommodate them. There is no evidence of underlying dental disease. It was concluded that the swellings were due to prominent normal dental sacs. The indistinct lamina dura on the caudal aspects of PM4 and M1 can be explained by the obliquity of the X-ray beam. This image is slightly unsharp as a result of magnification (cassette too far from head). (B) Left latero-45°-ventral–right lateral oblique radiograph of the right mandible of a 9-year-old horse. The cortex is straight, but variable in thickness, and the teeth lie well above its endosteal border. From PM3 (407) back, the roots become more caudally orientated and the slope of the reserve crowns increases. Despite earlier eruption, the apices of PM2 (406) remain rounded. The buttresses of cortical bone rostral to the apices of PMs 3 and 4 (407 and 408) are normal variants.

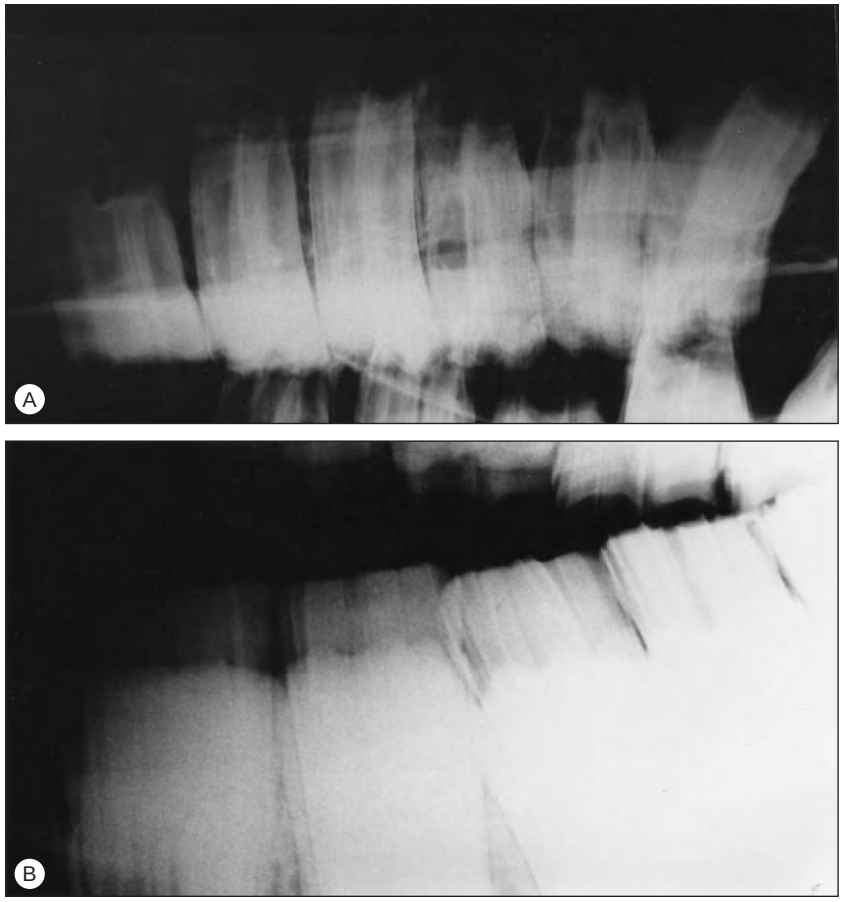


Figure 14.13. (A) Left latero-30°-dorsal–right lateral radiograph of the normal right maxillary arcade of a 10-year-old shire cross taken with the mouth open. A high radiographic exposure has been used to penetrate the reserve and exposed crowns. As a result, detail of the maxillary sinus, root apices and alveolar structures has been 'burnt out.' The crowns show even, linear striation, with no marginal defects and adjacent rostral and caudal borders of the exposed crowns are in contact. The occlusal surface of the arcade is slightly undulant, but full detail is obscured by the root apices of the contralateral maxillary arcade. Note the superimpositions of the images of the left infra-orbital canal and maxillary sinus walls. (B) Left latero-10°-ventral–right lateral oblique radiograph of a 4-year-old Welsh mountain pony taken with the mouth open to show the exposed crowns and occlusal surfaces of the right mandibular arcade. Horizontal alignment is almost perfect and there are no diastemata between the crowns. Although this animal had a mandibular periapical infection, the crowns were considered to be normal.



Figure 14.14. Ventrodorsal radiograph centered 2 cm rostral to the right facial crest showing the maxillary cheek teeth which have been revealed by displacing the mandible toward the left. The abaxial surfaces of caudal PM4 (108) and Ms 1-3 (109–111) are clearly shown in contrast with the air-filled maxillary sinuses surrounding them.



Figure 14.15. Intra-oral oblique radiograph showing the reserve and exposed crowns of left PMs 2–4 (206–208) and Ms 1 and 2 (209 and 210) of a 7-year-old hunter. The slight unsharpness of the images of the more caudal teeth is due to poor screen/film contact. Note the sharply pointed margins of the occlusal surfaces and caudally decreasing interdental spaces. The distinctive shape and caudal angulation of PM2 is clearly demonstrated. Periodontal membranes and lamina dura can be seen in the interdental spaces through which the X-ray beam has passed tangentially.

field is collimated to show the right maxillary cheek teeth. The relationships between the wall of the maxillary sinus and buccal surfaces of the reserve crowns of caudal PM4 (108) and the molars are clearly shown, but details of crown structure cannot be appreciated. PMs 2 and 3 (106 and 107) and rostral PM4 (108) are superimposed on the rostral wall of the maxilla. To reveal the junction between them, slight leftward rotation of the head would be required (see Fig. 14.26).

Intra-oral radiographs. A customized projection using intra-oral film is illustrated in Fig. 14.15. To demonstrate the lower reserve and exposed crowns of the maxillary arcade, the film was wedged at an angle close to the lingual surfaces of the teeth and the X-ray beam directed at about 20° dorsal to lateral.

Clinical indications

Radiography is generally used to confirm or provide additional information about dental problems already suspected or identified by more direct clinical examination.

Cases involving trauma to the facial bones or mandibles severe enough to consider interventional management should be routinely radiographed and checked carefully for dental involvement (see Figs 14.23–14.25).

Gross disorders of the exposed crowns are recognized easily by visual inspection and palpation, but precise details of defects thus discovered are often difficult to ascertain. Radiography may therefore contribute to making a decision about the most suitable management strategy. In contrast, disease of the roots and reserve crowns of the grinding teeth

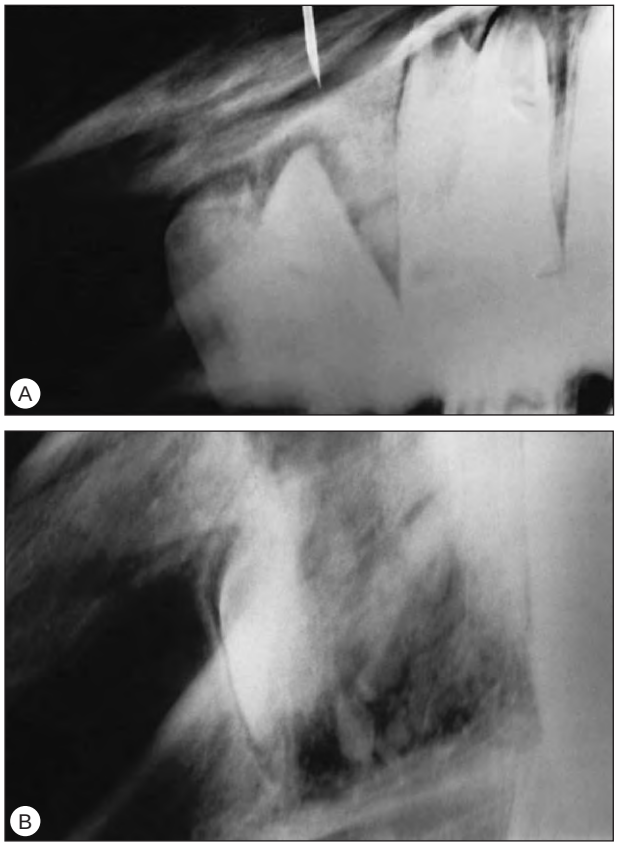


Figure 14.16. Left latero-30°-ventral–right lateral oblique radiographs showing (A) a radiopaque marker (hypodermic needle) used as a guide to the location of a diseased tooth (left PM2; 206) and (B) close-up postoperative study following removal of the tooth showing many retained dental fragments.

may not be associated with visible or palpable dental abnormalities and the presenting signs of nasal discharge, facial swelling, discharging tracts and sometimes pain are nonspecific. In such cases, radiography plays a crucial part in diagnostic confirmation and the identification of the affected tooth or teeth. It should be remembered that although maxillary sinus endoscopy gives direct visual access to the roots of the caudal cheek teeth, chronic inflammatory changes within the cavity may obscure the clarity with which they can be seen.

Radiography makes an important contribution to a number of interventional procedures. Landmark guides can be established by taking radiographs immediately preoperatively with radiopaque markers placed externally to assist in the identification of an individual diseased tooth at surgery (Fig. 14.16A). Intra- or postoperative radiographs are indicated routinely to check for the removal of all dental fragments following repulsion of diseased teeth (Fig. 14.16B) and to confirm correct alignment following fracture reduction or stabilization.

Follow-up radiographs may be indicated to check the progress of healing in trauma cases, or to investigate complications arising from earlier interventions (see Fig. 14.27).

Radiological interpretation

Developmental disorders

Abnormalities may occur in the number, size, shape and position of the teeth, and often several problems occur in combination. Gross abnormalities of the exposed crowns can be evaluated visually, but radiography is useful for showing the full effects of any defects by revealing the structure and location of the roots and reserve crowns. The radiographic appearance of the teeth associated with gross facial abnormalities such as wry nose and brachygnathia have been illustrated in a comprehensive review of congenital dental disorders.[31]

Supernumerary teeth

Figure 14.17 shows an example of the consequences of the development of an extra maxillary cheek tooth.

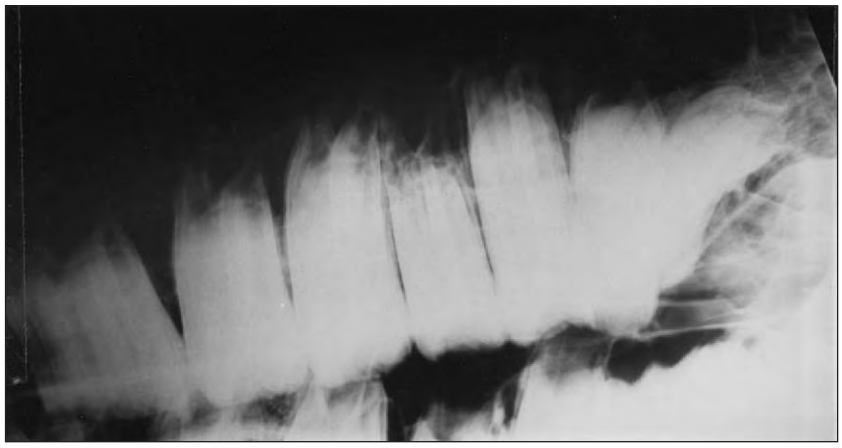


Figure 14.17. Supernumerary molar. Left latero-30°-dorsal–right lateral open-mouth oblique radiograph of the right maxillary arcade of a 6-year-old English warmblood presented with palpable abnormality of the right caudal maxillary cheek teeth and reported to be difficult to ride. There are seven maxillary cheek teeth, the most caudal of which is curved and partially superimposed on the penultimate tooth, suggestive of over-riding and buccal or gingival dislplacement. This tooth does not contribute to the occlusal surface, which appears unremarkable. Exposure is too high for accurate evaluation of the root apices and maxillary structures, but there is no evidence of gross radiographic change around the affected teeth.



Figure 14.18. Malformation. Dorsoventral intra-oral radiograph of the premaxilla of a 3-year-old filly showing a small, distorted left Is1 (201). The root is surrounded by an uneven radiolucent halo but the crown has erupted. Right Is1 (101) is more normal in shape but root and crown are both small and eruption is not complete. Is2 (102 and 201) are normal in size and erupted. DIs 3 (51 and 52) are still present and Is3 (103 and 203) are well formed but not yet erupted. The asymmetry between right and left sides is probably due to slight rotation.

Malformation

Deformity of the permanent maxillary Is1 (201 and 101) is illustrated in Fig. 14.18. It is not possible to determine whether this is a true congenital anomaly or whether the affected teeth were damaged during development.

Maleruption

Figure 14.19A shows grossly abnormal eruption of maxillary Is2 (102 and 202), which is bilaterally symmetrical. The affected and adjacent teeth are relatively normal in size and shape. In Fig. 14.19B, right maxillary PMs 2–4 (106–108) show varying degrees of deformity and maleruption.

Malocclusion

Any abnormality of eruption of permanent teeth which interferes with the integrity of the arcade in which it is situated predisposes to abnormal wear of the opposing arcade and a tendency to malocclusion. In the case illustrated in Fig. 14.20, the absence of an erupted right M1 (109), remnants of which lie in the maxillary sinus, has resulted in rostral migration of Ms 2 and 3 (110 and 111). Although the space is almost obliterated, a diastema in the occlusal surface remains and as a consequence, the opposing mandibular M1 (410) is overgrown. The presenting signs of oral discomfort and general malaise were attributed to this problem. Retention of permanent cheek teeth is an unusual but well-recognized developmental anomaly.[32]

Anadontia

Complete absence of individual teeth can give rise to dramatic effects of malocclusion. The animal illustrated in Fig. 14.21 had severe problems with mastication and was in poor body condition. Absence of temporary and permanent mandibular PM4s (86 and 408) had led to complete lack of wear of the opposing maxillary deciduous tooth and severe malocclusion. The condition was bilateral.

Temporal terratoma (dentigerous cyst)

Development of anomalous dental tissue in the parietotemporal region of the calvarium is a widely recognized cause of discrete, hard swellings and discharging tracts on the heads of young horses. Figure 14.22 shows a well-developed terratoma which was situated just rostral to the base of the right pinna. Radiography contributes to accurate localization and preoperative evaluation of the size and structure of the lesions.

Trauma[33,34]

The use of radiography for evaluation of mandibular fractures is illustrated in Figs 14.23 and 14.24. Figure 14.25 shows multiple fractures of the maxillary and nasal bones and molar teeth. The injury was of 11 days duration and clinical signs of severe sinusitis had already developed. The radiographic findings of gross disruption of permanent teeth contributed to the decision to recommend euthanasia rather than attempting salvage surgery. Fractures of the crowns are best shown on open-mouth oblique projections. Figure 14.26 shows the use of the latero-15°-ventral–lateral oblique projection to demonstrate a fracture of the exposed crown of a maxillary first molar (109). Part of the crown has been lost, leaving a large diastema. A 30° oblique radiograph confirmed that periapical infection was also present. Figure 14.27 is a radiograph taken 3 months after removal of a diseased molar. The iatrogenic fracture of the adjacent tooth probably explained why signs of maxillary sinusitis had persisted.

Infection

Maxillary periapical disease

Infection involving the roots of the caudal cheek teeth (Ms 1–3; 109–111, 209–211) appears high on the list of differential diagnoses of unilateral purulent nasal discharge. Involvement of more rostral teeth (PMs 2 and 3; 106 and 107, 206 and 207) is the commonest cause of lateral facial swelling and discharging tracts in the maxillary region between the facial crest and diastema. Periapical infection of PM4 (108–208) may produce either or both clinical

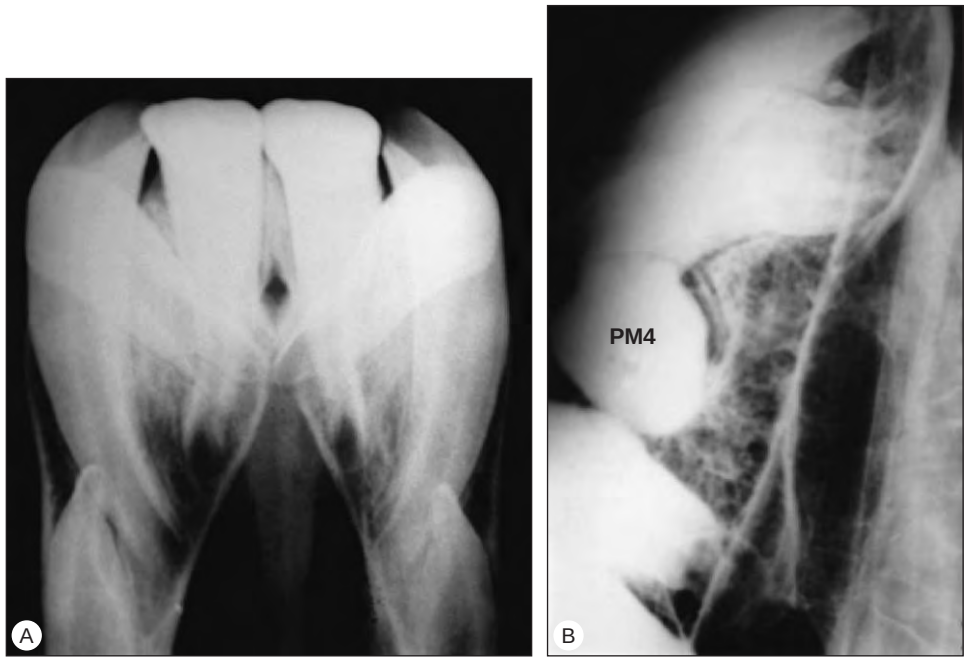


Figure 14.19. Maleruption. (A) Dorsoventral intra-oral radiograph of the premaxilla of a 5-year-old pony gelding showing maleruption of both Is2 (102 and 202). The root apices lie medial and rostral to those of Is1 (101and 201) and the alignment of the teeth is at an angle of about 40° abaxial to their normal location. Is3 (103 and 203) appear to originate from a normal position, but they are elongated and curved. The large infundibular spaces indicate relatively recent eruption. (B) Intra-oral 'lesion-orientated oblique' radiograph of a 5-year-old part thoroughbred showing right PMs 2–4 (106–108) and Ms 1 and 2 (109 and 110). PM2 (106) is directed caudally, PM3 (107) is bent caudally at the center of the reserve crown and PM4 (108) is rotated through 90° from its normal position to be viewed end on. M1 (109) appears to be unaffected.

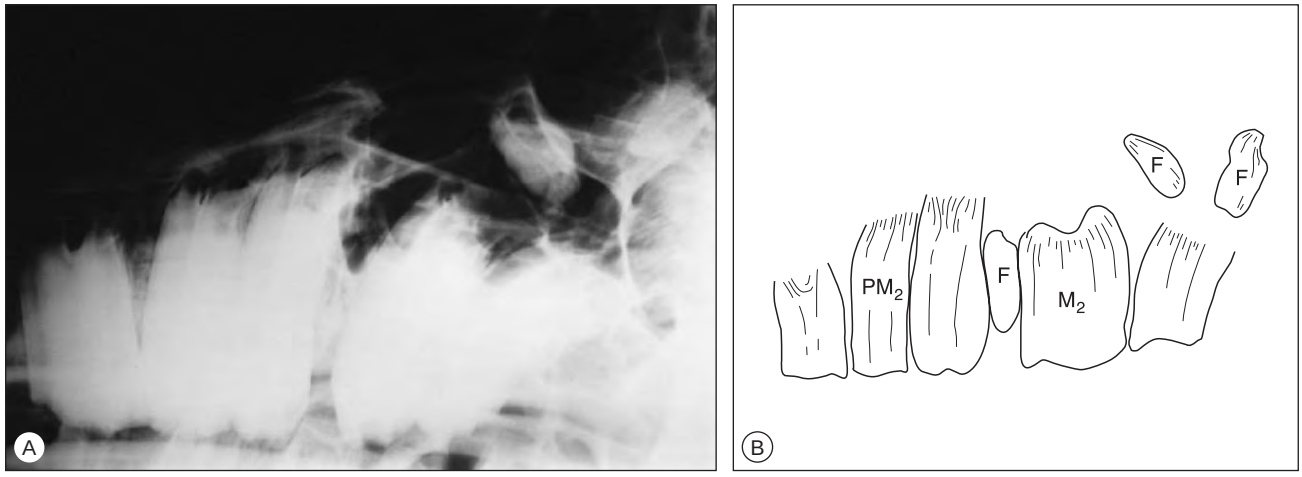


Figure 14.20. Retention and malocclusion. (A) Right latero-30°-dorsal–left lateral oblique projection of the left maxillary cheek teeth of a 6-year-old Arabian mare. A small fusiform mass of mineralized tissue is situated in the position where M1 (209) should be. PMs 2–4 (206–208) appear normal. Ms 2 and 3 (210 and 111) are erupted, but have migrated rostrally so that the space normally occupied by M1 (209) is almost closed. There is a marked defect in the occlusal surface at this site. There are two radiopaque mineralized masses in the caudal compartment of the maxillary sinus. The more rostral is homogenous in texture and probably represents an accumulation of cement. The other, which is partly superimposed on the ethmoturbinate, has a striated pattern suggesting that it is part of a deformed tooth. There is no evidence of any other pathological change in the sinus cavity or maxillary bone. (B) Line drawing; F = fragment.

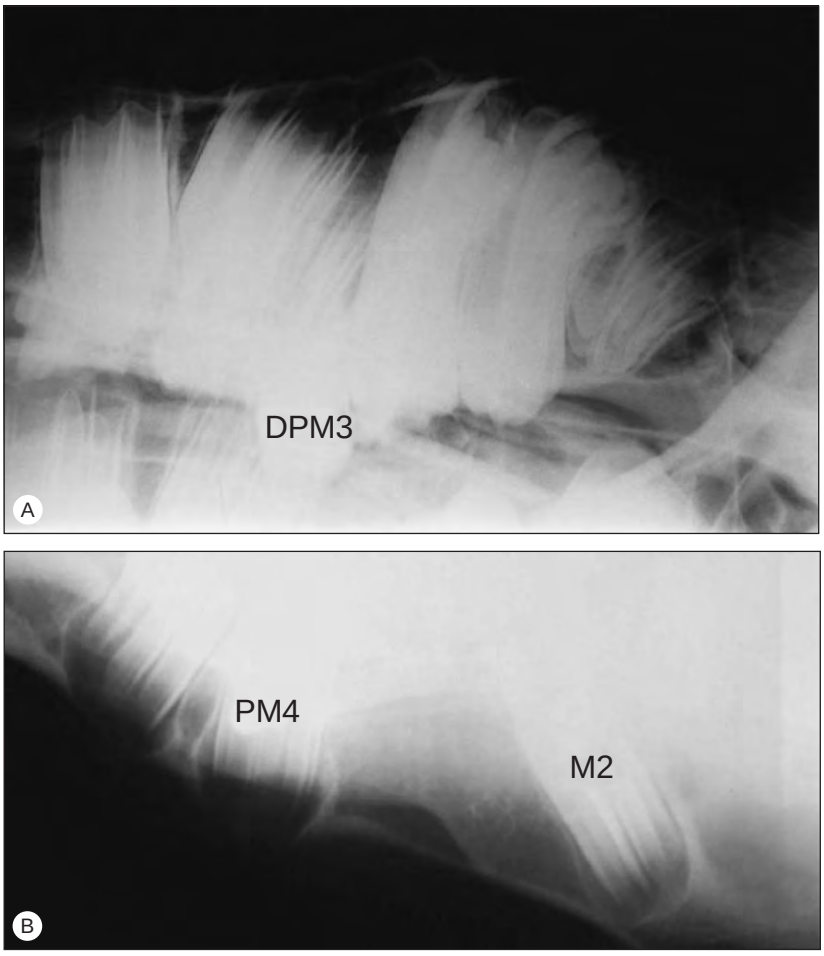


Figure 14.21. Anadontia and malocclusion. (A) Left latero-30°-dorsal–right lateral oblique and (B) left latero-45°-ventral–right lateral oblique radiographs of the right maxilla and mandible of a 2-year-old thoroughbred showing absence of mandibular M1 (409) (B) and abnormal elongation of maxillary DPM3 (56) (A). Maxillary PM3 (107) is angled rostrally and the developing PM4 (108) is very narrow and also rostrally orientated. Maxillary DPMs 1 and 2 (54 and 55) are still present and there is a well-developed wolf tooth (105). The molars appear normal; M3 (111) is in the midstage of development. Mandibular PMs 2 and 3 (407 and 408) appear relatively normal. M2 (409) is fully developed, but its rostral angulation is exaggerated. Unerupted M3 (411) is not included in the radiographic field.

presentations. Standing lateral radiographs may show evidence of osteitis at the level of the affected tooth (Figs 14.28, 14.29) and often indicate the presence of maxillary sinusitis (Figs 14.29, 14.30). Only occasionally can indications of dental involvement be seen on this projection (Fig. 14.30), so oblique views are almost always needed for diagnostic confirmation. It may be necessary to take two radiographs: one with the mouth closed using a relatively low exposure to show the root apices, alveoli and maxillary bone (Figs 14.28–14.31) and one with the mouth open using a higher exposure to demonstrate the reserve and exposed crowns (Fig. 14.26). On closed-mouth films, signs to look for are localized, ill-defined areas of increased radiopacity surrounding affected teeth, with coarsening of the texture of the overlying bone. This appearance is an indication of proliferative change associated with osteitis of the maxilla adjacent to infected tooth roots. In Fig.14.28, the lateral radiograph, (A) shows such changes and the oblique view, (B) confirms that the problem originates in PM3 (207). In horses in which periapical infection has led to maxillary sinusitis, a diffuse increase in radiopacity of the whole sinus cavity will be seen on standing lateral radiographs (Fig. 14.29) and fluid levels may also be present (Fig. 14.30). However, these findings are non-specific and also occur in other types of paranasal sinus disease. The ease with which teeth with periapical infection can be recognized on oblique radiographs is variable. A study carried out in 1987[7] showed that independent observers failed to identify the correct tooth in more than 50 per cent of cases, but since that time, rigorous efforts to improve radiographic technique, increased experience and training have markedly increased accuracy of identification (Gibbs, 1997, unpublished data). Introduction of the open-mouth oblique projection has also increased the reliability of detection of diseased teeth. The most consistent sign of periapical disease is an area of increased lucency around the affected apex or apices, referred to as a 'halo.' There may also be loss of lamina dura, but as this structure is an inconsistent feature on radiographs of normal horses its absence is not a reliable indicator of pathological change. In many cases the roots are damaged. They may be partly destroyed or distorted, increased in density and 'clubbed.' In cases in which infection is a result of a displaced fracture or fragmentation of the crown, the radiographic effects are graphically displayed, particularly on open-mouth views. Deposits of cement, appearing as fragments of unstructured mineralized tissue

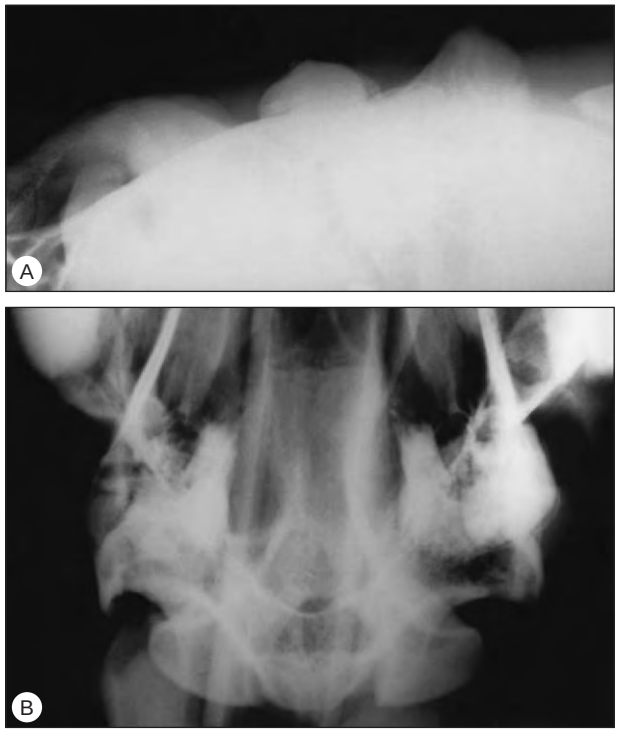


Figure 14.22. Temporal terratoma. (A) Lesion-orientated oblique and (B) ventrodorsal radiographs of the caudal skull of a yearling thoroughbred colt. On (A), an ovoid, laminated structure can be seen partly superimposed on the parietal bone. On (B), the same structure can be seen immediately abaxial to the left petrous temporal bone. A radiolucent margin and thin outer shell are visible caudally. This appearance is typical of a temporal terratoma.



Figure 14.23. Rostral mandibular dental fracture. Ventrodorsal intra-oral radiograph of the rostral mandible of a 7-year-old gelding showing a fracture through the left side. All three incisors (301–303) are displaced rostrally. There is an oblique fracture through the root of left I1 (301) and the abaxial border of I3 (303) is separated from the alveolar bone. Note the clearly defined pulp cavities and infundibular cement layers. A lateral projection would be required to evaluate fully the degree of displacement, but this could probably have been determined clinically.

('cement pearls') are sometimes seen adjacent to infected roots. Maxillary osteitis may obscure radiographic detail of affected teeth, making interpretation more difficult. Examples of maxillary periapical infection showing the above features appear in Fig. 14.31. Use of the ventrodorsal projection with offset mandible to demonstrate low-grade periapical infection, alveolar disease and chronic osteitis is shown in Fig. 14.32. This projection should be considered if the findings on conventional oblique radiographs are inconclusive.

Dental rhinitis

Dental infection which causes erosion of the internal wall of the maxilla can lead to severe rhinitis, which may be associated with dystrophic calcification of intranasal structures. Premolar teeth are most likely to be involved. Such a case is illustrated in Fig. 14.33.[35]

Mandibular periapical disease

The radiographic features of mandibular periapical infection vary according to the age of the tooth or teeth involved and the severity and degree of activity of the septic process. In immature and recently erupted teeth (Fig. 14.34A,B) the outline of the dental sac is usually distorted and irregular with deposition of smooth, well-organized new bone peripherally, sometimes in large amounts. In highly active infections, the proliferative bone may be fluffy and irregular. In well-established cases, the affected roots become distorted and may be partly destroyed (Fig. 14.34). Discharging tracts are often visible as radiolucent defects in the mandibular cortical bone (Fig. 14.34B and D). In cases of infection involving mature teeth, radiographic changes tend to be more chronic, with bone proliferation predominating over alveolar destruction (Fig. 14.34C). Root deformity is the feature by which affected teeth can be identified (Fig. 14.34D).

Periodontal disease

Radiography is not commonly indicated in the investigation of periodontal disease *per se*, but may be useful for demonstrating the effects of dental loss, displacement or malocclusion (Fig. 14.35) and for confirming the presence, location and configuration of diastemata (Fig. 14.36).

Tumors and cysts involving teeth

Primary dental neoplasms are rare, but their clinical and radiological characteristics have been reviewed in detail.[36] Ameloblastomas (adamantinomas), which mainly affect the mandible, tend to be locally destructive, possibly cystic and expansile. Ameloblastic and complex odontomas are expansile and contain mineralized material. Compound odontomas

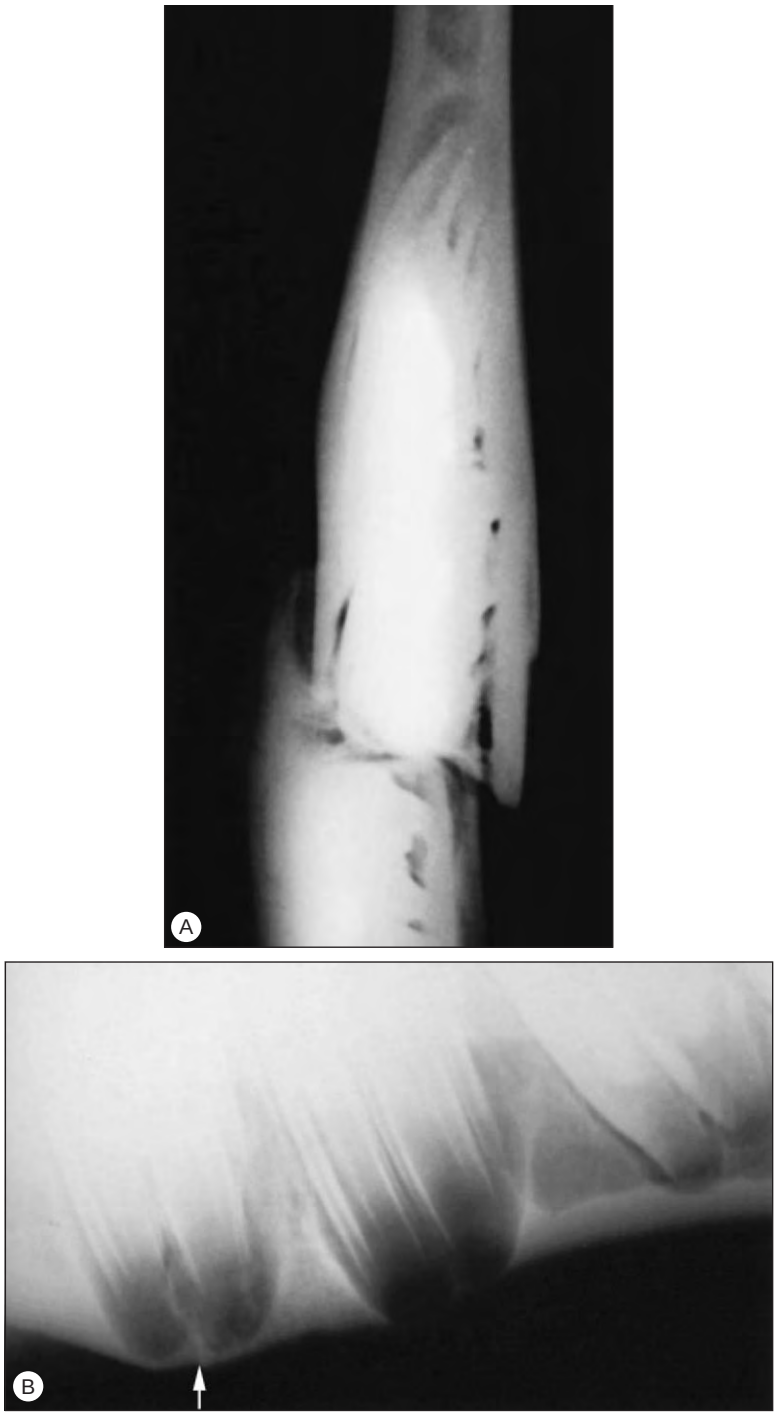


Figure 14.24. Fractured mandibular ramus with dental involvement. (A) Ventrodorsal intra-oral and (B) latero-45°-ventral–right lateral radiographs of the right mandibular ramus of a 5-year-old horse. The ventrodorsal projection shows a slightly oblique fracture just caudal to PM3 (407) with minor axiocaudal displacement. In this view, the tooth appears to have been spared. However, on the oblique projection, the slightly over-ridden fracture line, which is seen from the ventral aspect (arrowed), runs between the radicles of PM3 (407). PM2 (406) is excluded from the radiographic field.

contain an orderly pattern of dental tissue and cementomas are composed of dense, mineralized material and are often associated with root apices.

Cyst-like swellings of non-dental origin in the maxillae and mandibles of juvenile horses are well recognized. Teeth may be displaced and their development impaired. Numerous histological diagnoses have been claimed for such lesions, or they have simply been referred to as 'developmental cysts.' Some involve the maxillary sinuses.[1–3,5,7,12,29] The case shown in Fig. 14.37 was classified as an ossifying fibroma. Caudal cheek teeth may be displaced as result of the expansile effects of maxillary sinus cysts. In a report of 15 cases, radiographic evidence of dental displacement was recorded in four of the 10 adults in the series.[37]

Malignant tumors of osseous, connective and epithelial tissues tend to occur in older horses and are usually

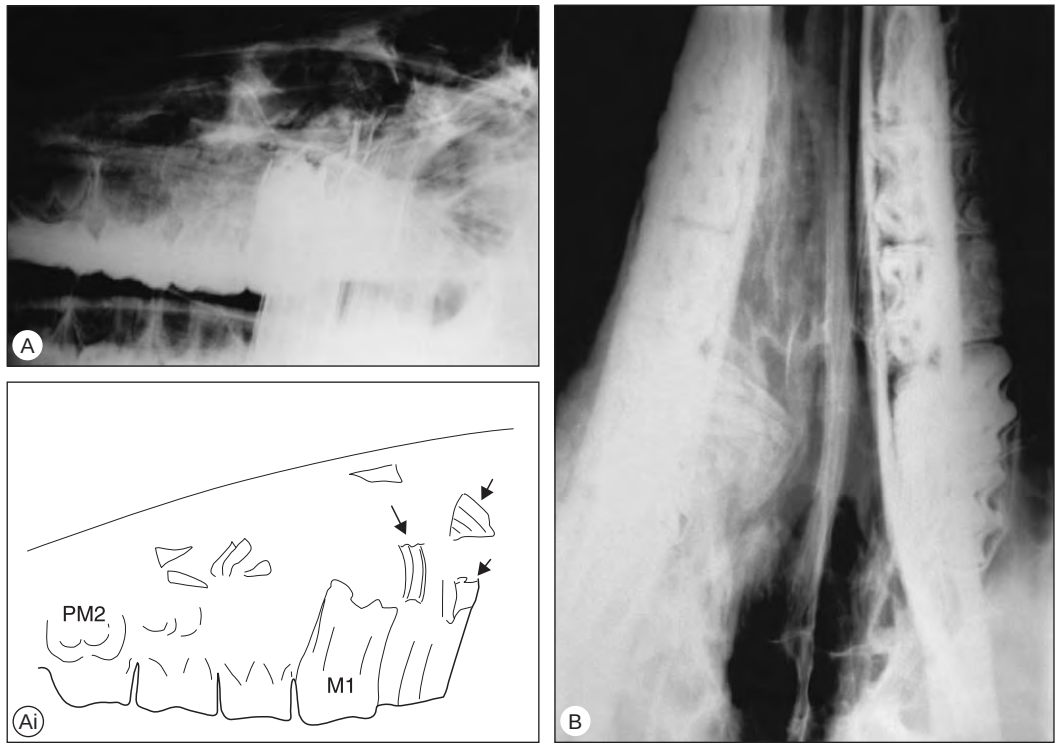


Figure 14.25. Facial and dental fractures. (A) Left latero-30°-dorsal–right lateral oblique and (B) ventrodorsal radiographs of a thoroughbred yearling taken after severe trauma to the right side of the head. In (A), the contents of the maxillary sinus are increased in opacity, giving a cloudy effect. Several free bone fragments can be seen within it. The dorsal portion of M1 (109) is missing, leaving a jagged border; several radiodense dental fragments lie adjacent to it. The rostral radicle of M2 (110) is also disrupted. DPs 2–4 (54–56) are in wear and developing germs of PMs 2 and 3 (106 and 107) are present. (Ai) Line drawing; dental fragments arrowed. (B) shows patchy increase in radiodensity of the right maxillary sinus and nasal chamber, extending rostrally to the level of PM2 (106). The nasal septum is deviated to the left. Bone and dental fragments can be seen in the maxillary sinus, including a large segment of M1 (109).

manifested by aggressive radiographic signs in which destruction predominates. If structures adjacent to teeth are involved, displacement is a likely consequence (Fig. 14.38). It should also be remembered that in the earlier stages, localized swelling caused by malignant tumours may be clinically similar to that associated with dental periapical infection, so that radiographic demonstration of bone destruction in the presence of normal teeth may be an important differentiating feature (Fig. 14.39).



Figure 14.26. Dental crown fracture. Left latero-15°-ventral–right lateral radiograph of the right maxillary exposed crowns of a 7-year-old Dutch warmblood. M1 (109) is fractured, with the rostral part of the crown missing, creating a diastema. There is stepping between the occlusal surfaces of M1 and M2 (109 and 110) and M2 and M3 (110 and 111).

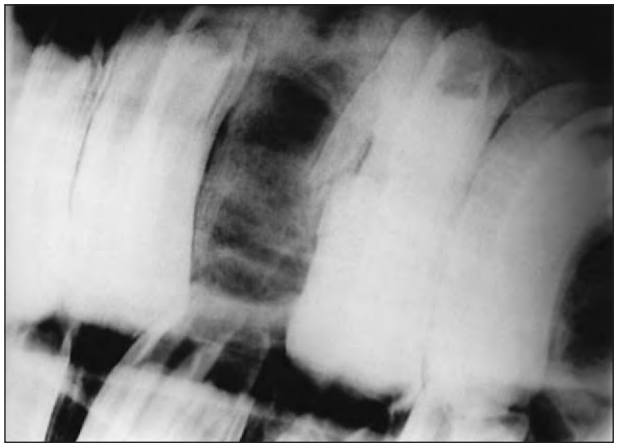


Figure 14.27. Iatrogenic dental fracture. Left latero-30°-ventral–right lateral oblique radiograph of the caudal left maxillary arcade of a 4-year-old thoroughbred taken several months after removal of M1 (109). A large portion of the rostral border of the reserve crown of M2 (110) has separated. The maxillary bone in the space originally occupied by M1 (109) has a dense, woven appearance, indicating that in-filling has taken place. Note the elongation of the dental images as a result of the radiograph being taken with the affected side uppermost.

Alternative imaging

Nuclear scintigraphy

In nuclear scintigraphy, radioactive material is introduced into the body, and its uptake by specific organs or lesions is measured using some form of photon counter, usually a gamma camera. The radionuclide is linked to a carrier substance with an affinity for the tissue under investigation. In the case of bone, the carrier most commonly used is ^{99m}Tc-MDP. This radiopharmaceutical is injected intravenously and after an appropriate time (approx. 3 hours for bone), the area under investigation is scanned, the emitted photons counted, and their distribution and intensity displayed electronically. A number of display modalities are available, including microdot imaging and color mapping. Microdot imaging appears to remain the modality of choice for bone (Fig. 14.40).

The use of nuclear scintigraphy is increasing in popularity for demonstration of areas of high bone turnover in the equine skeleton and is now available in many specialist practices. Its main advantages are that it can be performed with the horse standing and that once the investment in equipment has been made, operational costs are not unreasonably high. The main disadvantage is the potential radiation hazard, which entails strict control of isotope and patient handling.[27] In the United States, radiation sources (including radioisotopes for medical purposes) are governed by the National Council on Radiation Protection and Measurements; however, in each state there is something equivalent to a Radiation Protection Act which is administered by the state. In the case of Illinois, it is through the

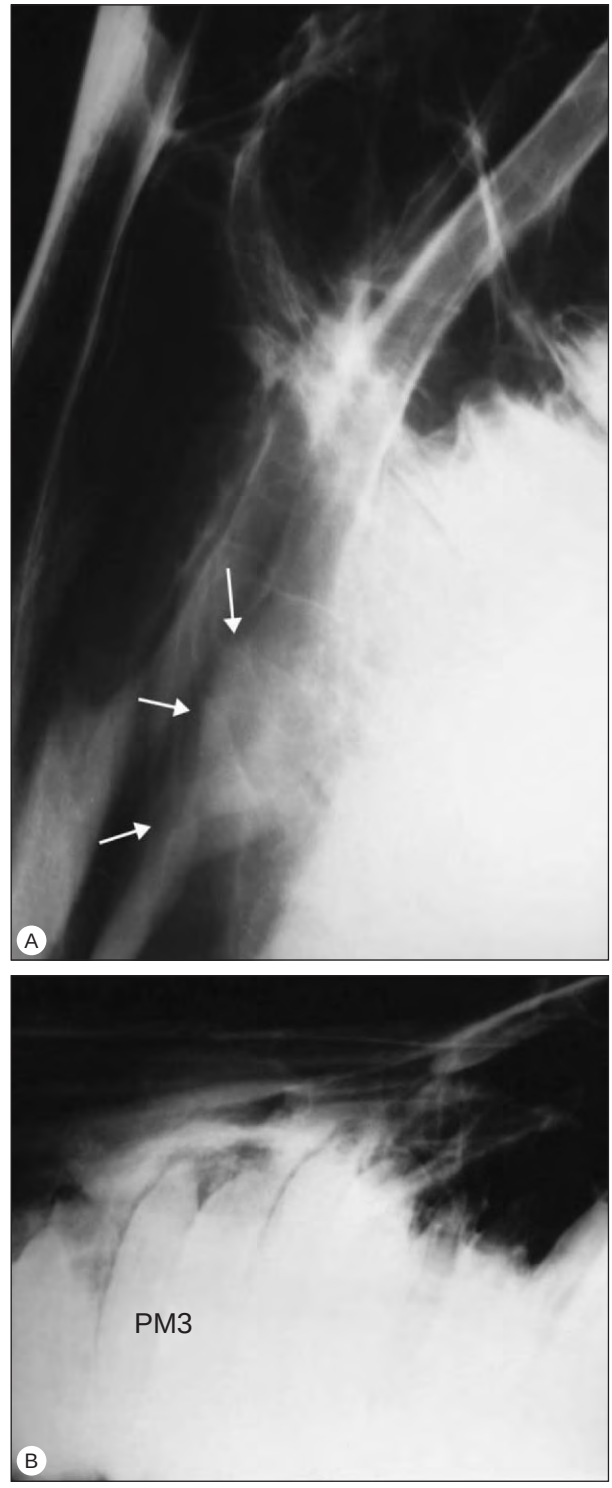


Figure 14.28. Maxillary periapical infection PM3 (107). (A) Standing lateral radiograph of the facial area of a 6-year-old hunter with a right-sided swelling rostral to the facial crest showing a localized, poorly defined area of increased radiopacity (arrowed) immediately dorsal to the roots of PMs 2 and 3 (106, 107) and well rostral to the normal condensation of bone which marks the rostral border of the maxillary sinus. (B) Left latero-30°-dorsal–right lateral oblique radiograph of the same animal showing a poorly marginated radiolucent halo around the root of PM3 (107), the caudal radicle of which is blunted. The increase in radiopacity of the surrounding maxillary bone is a result of proliferation due to secondary osteitis.

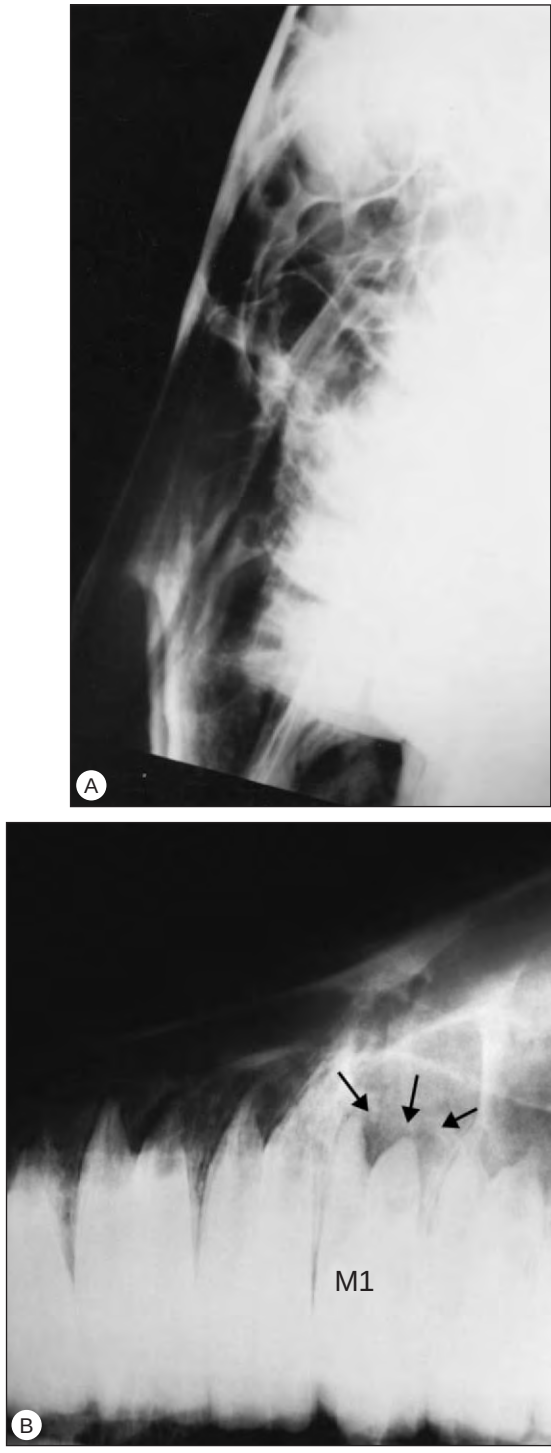


Figure 14.29. Maxillary periapical infection M1 (109) with sinusitis. (A) Standing lateral radiograph of a 6-year-old part thoroughbred with chronic, right-sided malodorous nasal discharge and no visible defects in the exposed crowns of the cheek teeth. There is patchy increase in opacity of the contents of at least one maxillary sinus, but no evidence of increased bone density. (B) Left latero-30°-dorsal–right lateral radiograph of the right maxillary arcade of the same horse. The increase in opacity of the maxillary sinus is more intense and homogenous in this projection. The root of the caudal radicle of M1 (109) is surrounded and an indistinct radiolucent halo (arrows). There is no evidence of gross osteitis.



Figure 14.30. Maxillary periapical infection of PM4 (208) with sinusitis. Standing lateral radiograph of an 8-year-old pony with painful, antibiotic responsive left facial swelling and recent onset of left-sided nasal discharge. A fluid level can be seen in the rostral compartment of the maxillary sinus, but there is no obvious increase in density of the overlying bone. A small 'cement pearl' (arrowed) lies adjacent to the rostral radicle of PM4 (208). An oblique radiograph confirmed periapical infection of PM4 (208).

Illinois Department of Nuclear Safety. States that achieve an 'agreement state' status with the NCRP then grant licenses to purchase and handle radioisotopes. This license is obtained by describing the purpose, procedure and use of the isotopes as well as the training and experience of the proposed users. Once licensed, users are held to fulfilling the described criteria. I say this because the 'standard' is therefore 'floating.' The most succinct way I can see to put it is that users must comply with the Radiation Protection Act that governs their location.

The technique is particularly indicated in cases in which conventional radiography gives negative or inconclusive results, or if the precise location of a disease process cannot be established. As many dental disorders are associated with pathological changes in adjacent bone, scintigraphic images may provide useful information regarding the exact tooth or teeth involved.[17–19]

Those intending to use nuclear scintigraphy for the investigation of dental disease will be familiar with the general principles of isotope administration, patient management

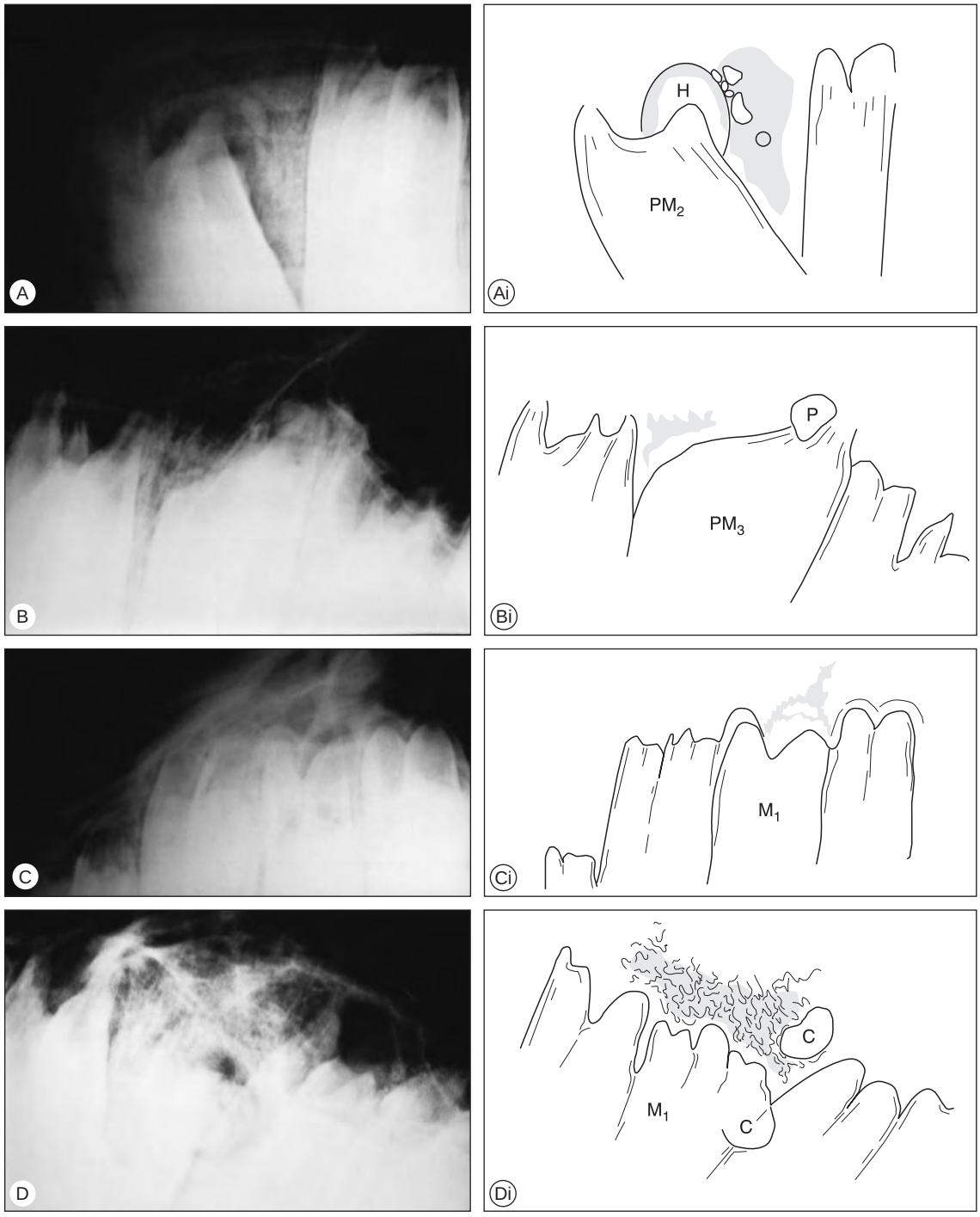


Figure 14.31. Maxillary periapical infections. Close-up images of latero-30°-dorsal–lateral oblique projections of the maxillary cheek teeth of four horses. (A) Five-year-old hunter showing a distinct periapical halo with a sclerotic margin around the caudal radicle of PM2 (107/207), which is more pointed than normal. Fragments of cement can be seen in the caudal part of the halo. The trabecular pattern of the surrounding maxillary bone is dense and coarse. (Ai) Line drawing; H = halo; O = osteitis. (B) Nine-year-old polo pony showing complete absence of the rostral radicle of PM4 (108/208) and an irregular cement 'pearl' superimposed on the caudal radicle. There is no obvious periapical halo or proliferation of maxillary bone and the sinus appears clear. (Bi) Line drawing; P = 'cement pearl.' (C) Three-year-old thoroughbred with an ill-defined and poorly marginated radiolucent halo around the caudal radicle of M1 (109/209). The normal rostral radicle has a narrower, clearly defined halo with a dense margin which represents the dental sac. There is a diffuse increase in radiopacity in the maxillary bone and sinus surrounding this tooth which could be due either to osteitis or localized sinusitis. Note that the roots of unerupted M2 (110/210) are blunt and that the dental sac is represented by a wide periodontal membrane and ill defined lamina dura. (Ci) Line drawing. (D) Eight-year-old pony showing collapse and fragmentation of M1 (109/209) with large masses of cement adjacent to its caudal root. There is marked but well-defined increase in density and trabecular coarsening of the surrounding maxillary bone, but no obvious opacification of the contents of the sinus. Note that the adjacent teeth have migrated to reduce the gap between PM5 (108/208) and M2 (110/210), indicating that the dental lesion is of long standing. (Di) Line drawing; C = cement.

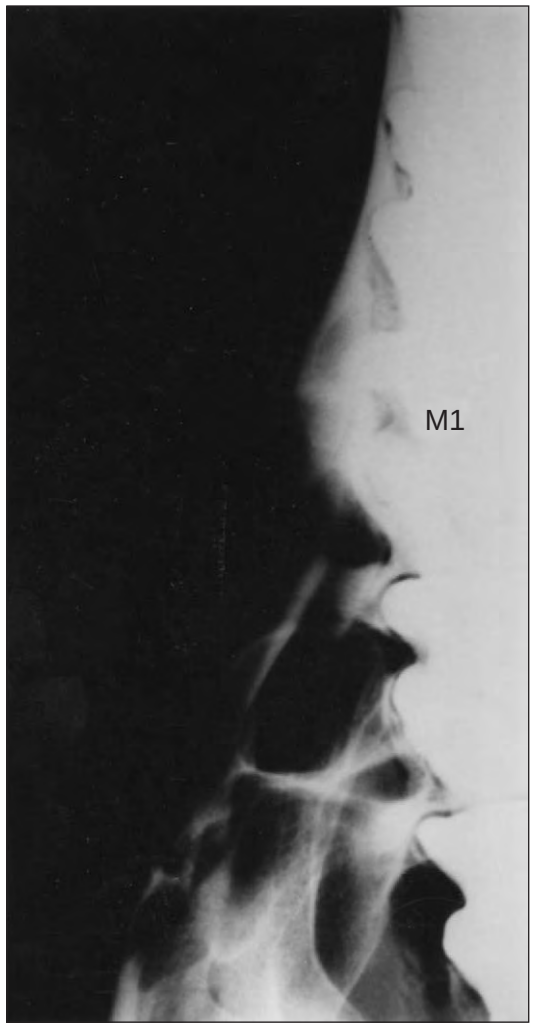


Figure 14.32. Maxillary periapical infection M1 (109). Ventrodorsal radiograph of a 12-year-old hunter with the mandible displaced to the left and the head slightly rotated in that direction, collimated to show the buccal surfaces of the right cheek teeth and maxilla. There is an indistinct radiolucent halo around the rostral radicle of M1 (109) and the overlying and surrounding bone is smoothly thickened. This horse presented with a chronic nasal discharge, quidding and, unusually, facial swelling.

and image acquisition and for details of procedures specifically recommended for dental examinations are referred to a recent comprehensive publication.[19] In this study, ^{99m}Tc-MDP was found to be superior to ^{99m}Tc-HMPAO-labelled leukocytes as a carrier agent and all relevant information was acquired during the bone phase of examinations, with no additional contribution being made by soft-tissue phase images. A series of 12 images was routinely obtained, comprising three ventral, three dorsal and three left and right lateral, centered in turn on the incisors, cheek teeth and temporomandibular joints. Alveolar bone and the ventral border of the mandible were clearly identifiable as areas of increased activity, and the teeth themselves, the nasal bone and the rest of the mandible were seen as areas of relatively low activity. There was a marked age difference in the appearance of the scans of the cheek teeth, with, predictably, much increased intensity in younger animals, in which the periodontal structures were clearly defined, with less obvious distinction between individual teeth in older subjects (Fig. 14.41). Periapical dental infection resulted consistently in very intense focal uptake of ^{99m}Tc-MDP around the affected tooth (Fig. 14.39), but a similar uptake pattern was seen in a mare with a maxillary sequestrum, indicating a potential limitation in the specificity of these studies. Periodontal disease produced a linear pattern of activity over the whole arcade (Fig. 14.42). Lesions confined to dental tissue (pulpitis and infundibular necrosis) did not cause alteration in scintigraphic uptake. Measurements of counts in regions of interest defined over the teeth in right and left dental arcades were compared using dorsal and ventral scans. The results showed a highly significant increase in uptake on the affected side in horses with periapical infections, thus giving an objective method for identification of unilateral disease. This work confirms that scintigraphy makes an important contribution to the identification of periapical dental disease and should be considered routinely when radiographic examinations are inconclusive or fail to confirm clinically suspected disease, but it must be remembered that although sensitivity for lesion detection is high, specifity for defining pathological changes is relatively poor.

Computed tomography (CT)

In computed tomography, a minute X-ray beam scans a selected plane within the patient and the information derived from differential absorption by the individual structures through which it passes is enhanced and displayed by controlled electronic processes. Rapid technological advancement now allows highly sophisticated processing of the images produced, so that the area of interest can be represented in a variety of sectional orientations. Furthermore, by recording minor differences in attenuation numerically, it is possible to make objective measurements of the radiographic density of tissues and the changes associated with disease.[22]

To perform CT on horses, special facilities are required to permit appropriate manipulation of the patient and the scanner must have an aperture large enough to accommodate the part being investigated. Such examinations have been carried out at a few centers for several years and the special requirements for space, environmental control, patient handling and access were reported as early as 1987.[20] Scanning protocols were also discussed. Since that time, refinements in equipment and computer software have led to a vast improvement in the quality of images obtainable. Many advanced imaging centers now offer CT facilities routinely and several reports have been published on its application to the equine head.[21–25]

Computed tomography is particularly appropriate for examination of the equine facial area and dental structures

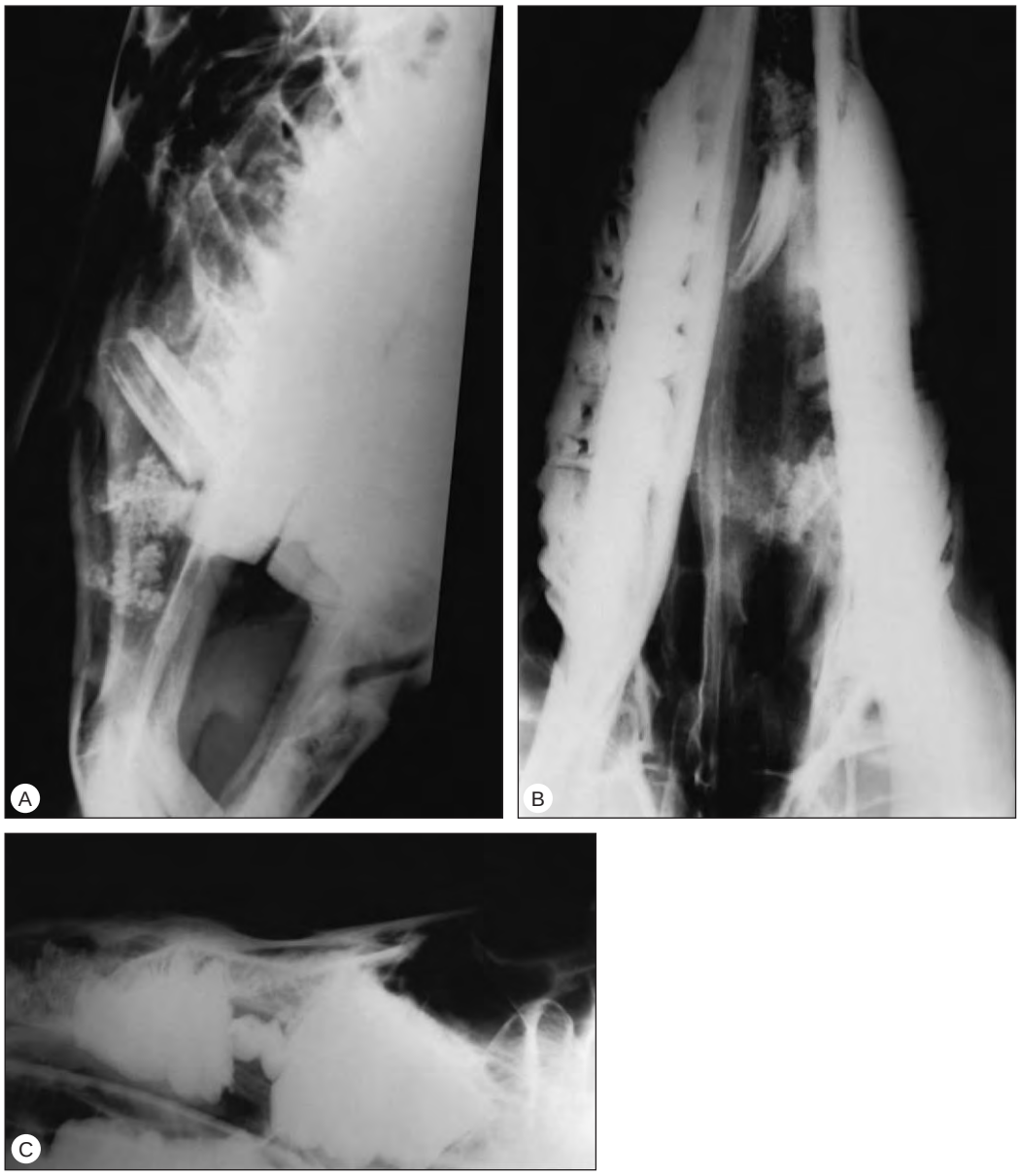


Figure 14.33. Dental rhinitis. (A) Standing lateral, (B) ventrodorsal and (C) right latero-40°-dorsal–left lateral oblique radiographs of the facial area of a 13-year-old pony in good bodily condition with a purulent left nasal discharge of 6 months duration and absence of left maxillary PM4 (208). Part of the missing tooth has been displaced into the left nasal cavity (B) in which there are also multiple large deposits of granular mineralized material. The normal contents of the cavity are disrupted and the nasal septum is displaced to the right. The contents of the maxillary sinus, as shown in (C) appear to be normal.

because of the high inherent radiographic contrast of the areas of interest. In dentistry, the need for clear, unobstructed images of the teeth is an additional powerful indication for this technique. Figures 14.43 and 14.44 show respectively gross changes in the maxillary bone and sinus following extraction and subtle periapical infection.

The main disadvantage of CT is that general anesthesia is essential and it must often be performed in a location which is not suitable for immediate transfer to surgery. The high cost of equipment and maintenance and the need for specialist technical skills for operation and interpretation suggest that, for the foreseeable future, facilities will mainly be confined to specialist centers and investigations undertaken by qualified diagnostic imagers. However, if clinical examination, carefully executed conventional radiography and scintigraphy fail to give the information needed to

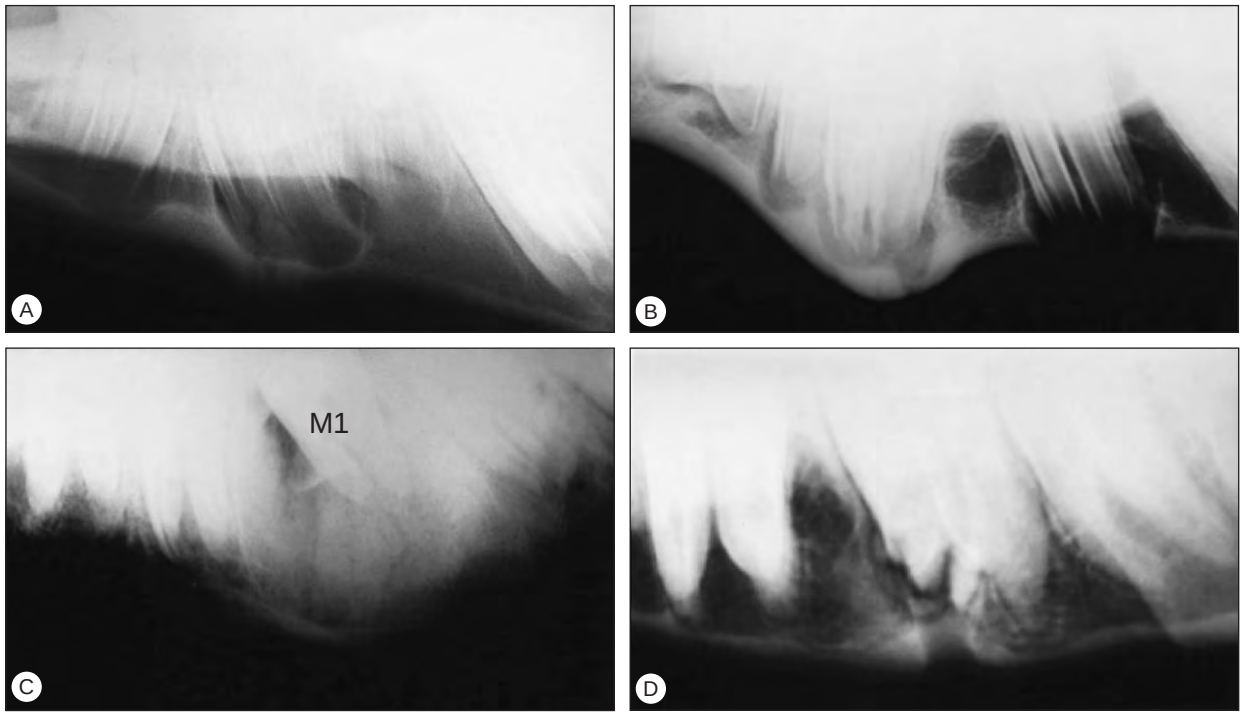


Figure 14.34. Mandibular periapical infections. Close-up images of latero-45°-ventral–lateral oblique radiographs of the mandibles of four horses. (A) Two-year-old thoroughbred with a small ventral mandibular swelling, recently noted, and discharging tract. The dental sac of developing PM3 (307/407) is slightly irregular in outline, the ventral mandibular cortex is thickened, especially rostrally and a linear, irregular radiolucent shadow leads from the rostral radicle to the exterior. The mineralized dental tissue does not appear to be disrupted. The image of the developing germ of PM4 (308/408) is partly superimposed on the caudal aspect of the affected tooth, because the X-ray beam was not exactly perpendicular to the long axis of the mandible. (B) Three-year-old thoroughbred with a hard, ventral mandibular swelling and discharging tract. PM3 (307/407) is deformed and the caudal radicle is large and appears to be duplicated. The apices are irregular in outline and convergent, with ragged borders. The surrounding mandibular bone is grossly thickened and a large radiolucent tract runs through it. Adjacent PMs2 and 4 (306/406 and 308/408) are within normal limits for their stage of development. (C) Seven-year-old hunter with a caudal mandibular swelling of unknown duration suspected of being neoplastic. Both radicles of the root of M1 (309/409) are shrunken, blunted and convergent and the tooth is surrounded by dense floccular bone which appears to be retained by a slightly thickened cortex. (D) Ten-year-old cob with a discharging tract and thickened ventral mandible of 2 years duration. The rostral radicle of PM4 (308/408) is grossly distorted. The apex is absent and the residual reserve crown is thickened and irregular in outline. A large, radiolucent tract passes from the diseased root through the thickened cortex. The area of increased opacity of interdental bone indicates diffuse proliferation due to osteitis.



Figure 14.35. Periodontal disease. Lateral radiograph of an aged horse taken under general anesthesia. Loss and displacement of the mandibular cheek teeth as a result of periodontal disease has led to severe wear abnormalities in the maxillary arcades.

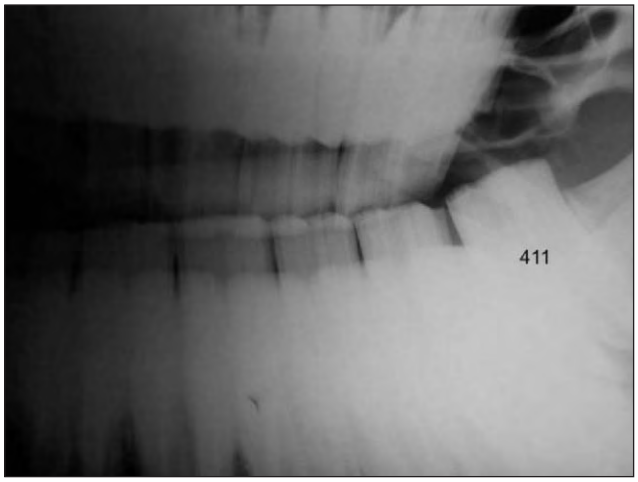


Figure 14.36. Diastema formation. Right lateral-10°-dorsal-left lateral open-mouth oblique radiograph of a 10-year-old horse showing a disterma between left Ms 2 and 3 (310 and 311).

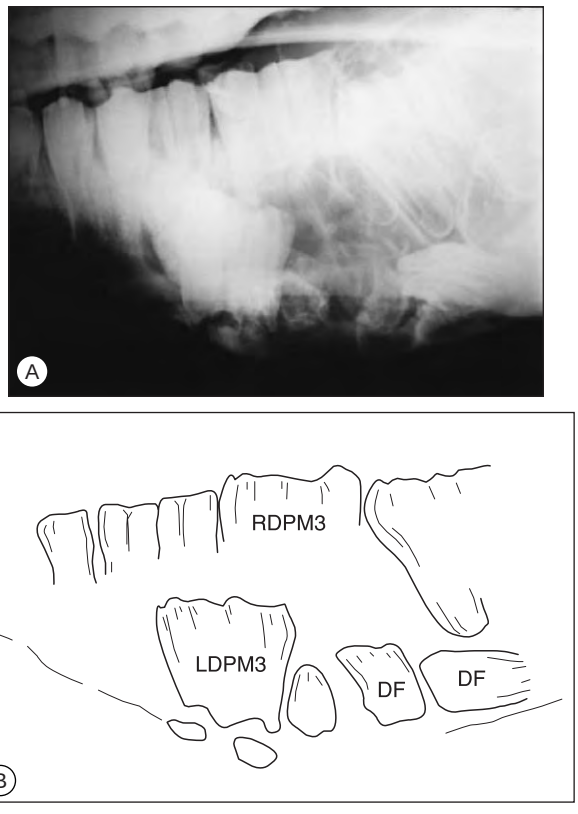


Figure 14.37. Ossifying fibroma. (A) Lateral radiograph of the mandible of a 6-month-old foal with a large, hard swelling of the left caudal horizontal ramus. On the normal right side, DMs 1–3 (84–86) and unerupted M1 (409) can be seen in a normal position. On the affected side, DMs 1 and 2 (74 and 75) are in place, superimposed on their right counterparts. DM3 (76) is normal in shape and size but displaced ventrally. Small, distorted germs of Ms 1 and 2 (409 and 410) appear to lie free in the soft-tissue mass. Several smooth-margined, amorphous clumps of mineralized material can also be seen within the lesion. (B) Line drawing; DF = dental fragments; RDPM3 = right deciduous PM3 (86); LDPM3 = left deciduous PM3 (76).

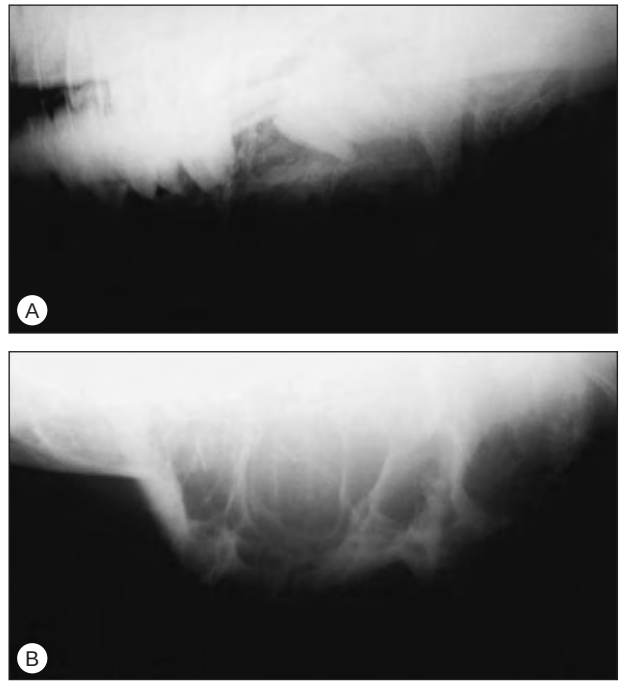


Figure 14.38. Anaplastic sarcoma. Two left latero-45°-ventral–right lateral radiographs of the right hemimandible of an 18-year-old pony with a large, firm, rapidly growing ventral swelling. Oral examination was reported to reveal loosening and displacement of the fourth cheek tooth. (A) (high exposure) shows that the affected tooth is, in fact, M2 (410). The more rostral teeth are in place, but the alveolar bone surrounding them has been destroyed. (B) (low exposure) shows the massive expansile swelling which is composed mainly of soft tissue, but traversed by numerous bony trabeculae. The mandibular cortex is thickened at the rostral site of origin of the mass.

make a properly informed decision on management of a potential dental problem and an appropriate facility can be reached, consideration of referral for CT would be strongly justified.

Magnetic resonance imaging (MRI)

The principles of MRI and its application to the equine head have recently been reported.[25] In this article, emphasis was placed on its contribution to evaluation of neural tissue and most of the lesions illustrated were intracranial. However, with continuing technical advances and further clinical experience, MR imaging of mineralized tissue is likely to become more widely applied and may prove to have an advantage over CT in certain circumstances. Figure 14.44B illustrates how the appearance of subtle changes associated with apical granuloma formation on CT is enhanced on an MR image.

Contexts

Alternative imaging techniques undoubtedly have a place in advancing knowledge of the patho-anatomical and patho-physiological changes associated with equine dental disorders. This knowledge can usefully be applied to

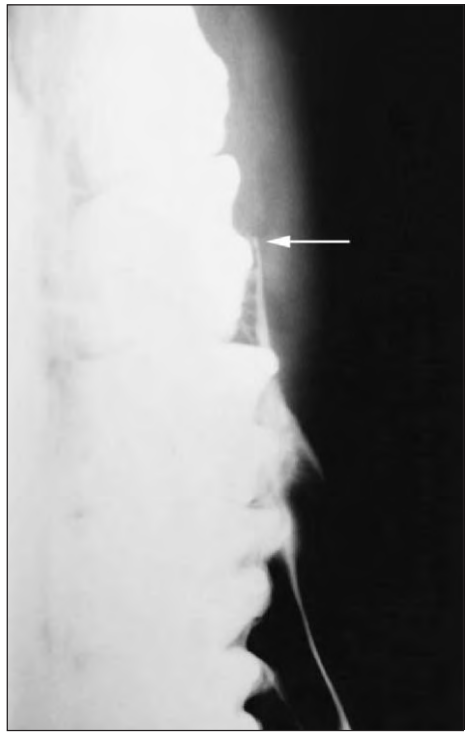


Figure 14.39. Undifferentiated sarcoma. Lesion-orientated oblique radiograph of a 12-year-old pony with a firm 5 cm × 8 cm painless swelling over the left infraorbital canal. Standard projections showed increase in the radiopacity of soft tissue in the affected area but no evidence of dental disease. The radiograph shows the soft-tissue outline of the mass and destruction of the lateral wall of the underlying maxilla (arrowed), which terminates abruptly at the level of mid-PM3 (207). The teeth appear normal.

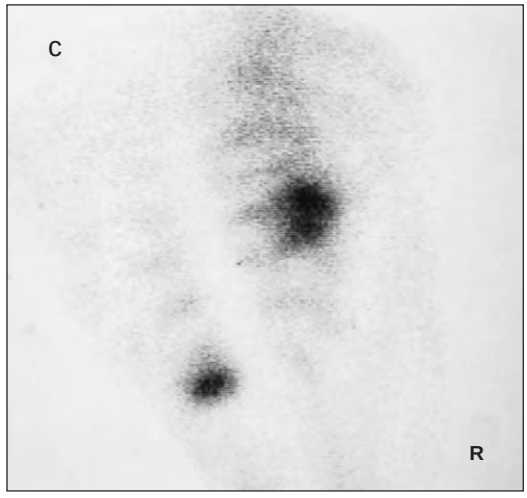


Figure 14.40. Nuclear scintigraphy. Five-year-old Cleveland bay mare presented with facial swelling and oral evidence of periodontal disease around right PMs 3 and 4 (107 and 108) which could not be related conclusively to either tooth. A scintigraphic image of the right side of the head (R = rostral; C = caudal), made 3 hours after injection of ^{99m}Tc-MDP shows localized increase in radionuclide uptake over the root of PM4 (108). Increased uptake has also occurred in the mandible at the level of PM2 (406). This was related to a small mass on the lateral aspect, believed to be associated with clinically insignificant trauma (from Jane Boswell, Royal Veterinary College, London, with permission).

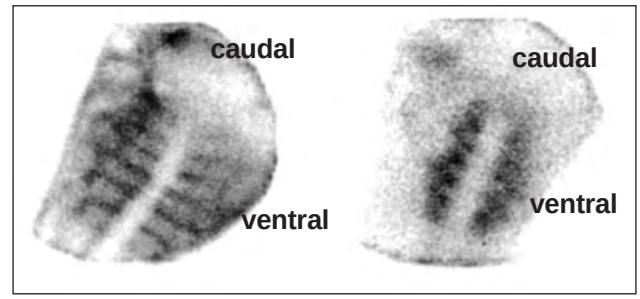


Figure 14.41. Left lateral bone phase scintigram of the teeth in a 3-year-old (left) and an 18-year-old (right). On the left image the alveolar bone is clearly visible surrounding each tooth on its dorsal and caudal aspects. On the right image the dorsal outline of the alveolar bone is lost and the interdental alveolus is shorter and less distinct (from ref. 19; reproduced courtesy of the authors and the Editor of *Equine Veterinary Journal*).

improve the understanding of the disease processes involved and, in certain circumstances, may also be helpful in improving our approach to conventional radiography by suggesting possibilities for new procedures and enhancing interpretive skills.

Alternative imaging techniques also give the option for valuable supplementary investigations in clinical situations in which conventional radiography fails to resolve a diagnostic problem. However, it is likely that for the foreseeable future, the wide availability and relatively economical cost of conventional radiography will ensure that it remains the most widely used diagnostic imaging modality for dentistry in general equine practice. It is for this reason that this chapter has concentrated on providing information aimed at helping to improve technical and interpretational standards.

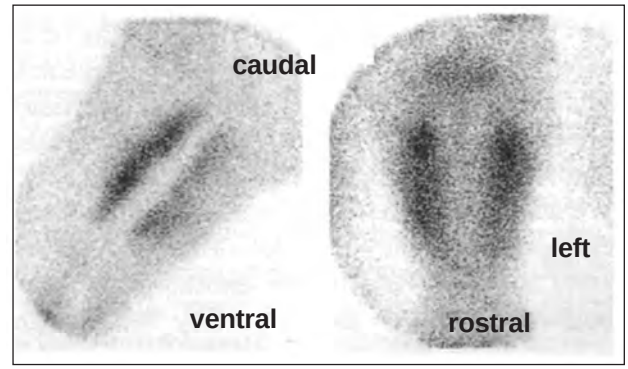


Figure 14.42. Left lateral (left) and dorsal (right) bone phase scintigram of a 25-year-old Welsh pony with a history of weight loss for a number of months. The scintigrams show a linear increase uptake, suggestive of severe generalized periodontal disease. The diagnosis was confirmed on post mortem examination (from ref. 19; reproduced courtesy of the authors and the Editor of *Equine Veterinary Journal*).

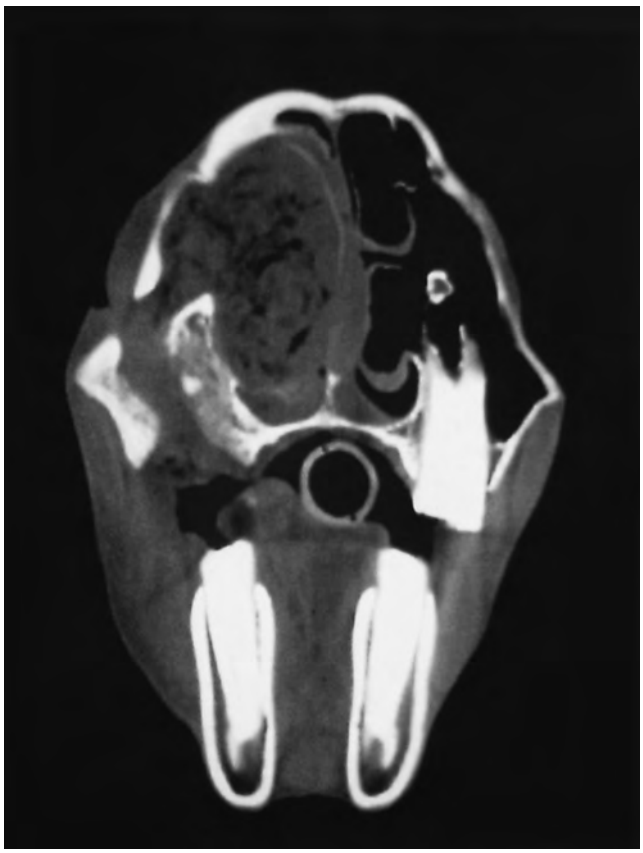


Figure 14.43. Computed tomography. CT image of a transverse scan through the head of a horse at the level of Ms 1 (09s). This animal had shown signs of maxillary sinusitis following extraction of maxillary M1 (109). On the left, relationships between M1 (209), the maxillary sinus, nasal cavity and nasal conchae are normal. On the right, maxillary M1 (109) is absent, the alveolus is open and there is gross thickening of the maxillary bone on either side (osteitis). The right maxillary and conchofrontal sinuses are filled with material of mixed soft-tissue density and gas lucency, representing ingesta which has entered from the oral cavity via the fistula. The nasal septum is displaced to the left and both dorsal and ventral conchae are virtually obliterated. There is a defect in the right maxillary wall and overlying soft-tissue swelling (from Prof.dr K. J. Dik, University of Utrecht, with permission).

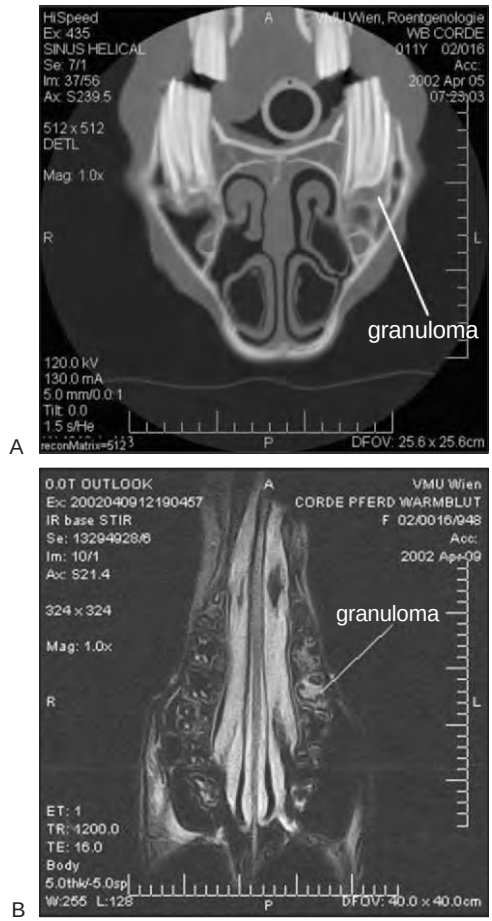


Figure 14.44. Computed tomography and magnetic resonance imaging. Twelve-year-old Austrian warmblood mare. (A) Transverse CT image (inverted) at the level of the fourth premolars (08s) using a bone window. There is opacification of the left maxillary sinus and the alveolus of left PM4 (208) is widened and distorted with obliteration of the lucency which represents the dental pulp in the apical region as compare with 108 on the right side (B) MR image using a fat-suppressed sequence showing hyperintense (light) signals in the crown of left PM4 (208) which surround the darker roots. These changes represent granuloma formation. Note that the vascular nasal conchae appear as hyperintense light images and air in the nasal passages and sinuses gives a hypointense black signal (reproduced by permission of Dr W. Henninger, Clinic for Radiology, Veterinary University, Vienna, Austria).

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REFERENCES

1. Cook WR (1970) Skeletal radiology of the equine head. *Journal of the American Veterinary Radiological Society,* 11, 35–55.
2. Baker GJ (1971) Some aspects of equine dental radiology, *Equine Veterinary Journal,* 3, 46–51.
3. Gibbs C (1974) The equine skull: its radiological interpretation. *Journal of the American Veterinary Radiological Society*, 15, 70–78.

4. Boulton CH (1985) Equine nasal cavity and paranasal sinus disease: a review of 85 cases. *Journal of Equine Veterinary Science,* 5, 268–275.
5. Wyn Jones G (1985) Interpreting radiographs, 6: radiology of the equine head. *Equine Veterinary Journal*, 17, 417–425.
6. Lane JG, Gibbs C, Meynick SE *et al.* (1987) Radiographic examination of the facial, nasal and paranasal sinus regions of the horse: I. Indications and procedures. *Equine Veterinary Journal,* 19, 466–473.
7. Gibbs C and Lane JG (1987) Radiographic investigation of the facial, nasal and paranasal sinus regions of the horse: II. Radiological findings. *Equine Veterinary Journal*, 19, 474–492.
8. Dik KJ and Gunsser I (1990) In: *Atlas of Diagnostic Radiology of the Horse. Part 3: Diseases of the Head, Neck and Thorax*. Schlutersche Verlagsanstalt and Druckerei, Hannover, pp.13–69.
9. O'Brien RT (1996) Intra-oral dental radiography: experimental study in two horses and a llama. *Veterinary Radiology and Ultrasound*, 37, 414–416.
10. Dixon PM (1997) Dental Extraction in horses: indications and preoperative evaluation. *Compendium of Continuing Education for the Practicing Veterinarian*,19, Equine 399–376.
11. O'Brien RT and Biller DS (1998) Dental Imaging. *Veterinary Clinics of North America: Equine Practice,* 14 (2) 259–271.
12. Butler JA, Colles CM, Dyson SJ, Kold SE and Poulos PW (2000) In: *Clinical Radiology of the Horse* (2nd edn). Chap. 8, Blackwell Science, London, pp. 357–384.
13. Tremaine H (2000) Equine dental radiography. *UK Vet*, 5, 8–13.
14. Tremaine H (2001) Equine dental radiology. *UK Vet,* 6, 1–5.
15. Easley J (2002) A new look at dental radiology in Proceedings. *American Association of Equine Practitioners*, 48, 412–420.
16. Barakzai SZ and Dixon PM (2003) A study of open-mouthed oblique radiographic projections for evaluating lesions of the erupted (clinical) crown. *Equine Veterinary Education,* 15, 143–148.
17. Metcalfe MR, Tate LP and Sellett LC (1989) Clinical use of ^{99m}Tc-MDP scintigraphy in the equine maxilla. *Veterinary Radiology*, 30, 80–87.
18. Boswell JC, Schramme, MC, Livesey, LC *et al.* (1988) Use of scintigraphy in the diagnosis of dental disease in four horses. *Equine Veterinary Education*, 11, 494–298.
19. Weller R, Livesey L, Maierl J *et al.* (2001) Comparison of radiography and scintigraphy in the diagnosis of dental disorders in the horse. *Equine Veterinary Journal*, 33, 49–58.
20. Barbee DD, Allen JR and Gavin PR (1987) Computed tomography in horses. *Veterinary Radiology*, 28, 144–151.
21. Dik KJ (1994) Computed tomography of the equine head. *Veterinary Radiology and Ultrasound,* 35, 236.
22. Tietje S, Becker M and Brockenhoff G (1996) Computed tomographic evaluation of head diseases in the horse: fifteen cases. *Equine Veterinary Journal,* 28, 98–105.
23. Morrow KL, Park RD, Spurgeon TL *et al.* (2000) Computed tomographic imaging of the equine head. *Veterinary Radiology and Ultrasound*, 41, 491–497.
24. Henninger H, Frame EM, Willmann M *et al.* (2003) CT of alveolitis and sinusitis in horses. *Veterinary Radiology and Ultrasound*, 44, 269–276.
25. Tucker RL and Farrell E (2001) Computed tomography and magnetic resonance imaging of the equine head. *Veterinary Clinics of North America: Equine Practice*, 17, 131–145.
26. Roberts GD and Graham JP (2001) Computed radiography. *Veterinary Clinics of North America: Equine Practice*, 17, 47–61.
27. British Veterinary Association (2002) *Guidance Notes for the Safe Use of Ionising Radiations in Veterinary Practice. Ionising Radiation Regulations 1999*. British Veterinary Association, London.
28. Morgan JP and Silverman S (1993) In: *Techniques for Veterinary Radiography* (5th edn). Iowa State University Press, Ames, IA, pp. 84–95.
29. Dixon PM and Copeland AN (1993) The radiological appearance of mandibular cheek teeth in ponies of different ages. *Equine Veterinary Education*, 5, 303–307.
30. Kirkland KD, Baker GJ, Marretta SM *et al.* (1996) Effects of ageing on the endodontic system, reserve crowns and roots of mandibular cheek teeth. *American Journal of Veterinary Research*, 57, 31–38.
31. DeBowes RM and Gaughn EM (1998) Congenital dental disease of horses. *Veterinary Clinics of North America: Equine Practice*, 14, 273–289.
32. Edwards GB (1993) Retention of permanent cheek teeth in three horses. *Equine Veterinary Education,* 5, 299–302.
33. Sullins KE and Turner AS (1982) Management of fractures of the equine mandible and premaxilla (incisive bone). *Compendium of Continuing Education for the Veterinary Practitioner,* 4, S480–S489.
34. Stickle R (1998) The equine skull. In: *Textbook of Veterinary Diagnostic Radiology* (3rd edn), ed. DE Thrall, WB Saunders, Philadelphia, pp. 105–112.
35. Richardson JD and Lane JG (1993) Metaplastic conchal calcification secondary to chronic peri-apical infection in seven horses. *Equine Veterinary Education,* 5, 303–307.
36. Pirie RS and Dixon PM (1997) Mandibular tumours in the horse: a review of the literature and seven case reports. *Equine Veterinary Education*, 5, 287–294.
37. Lane JG, Longstaffe JA and Gibbs C (1987) Equine paranasal sinus cysts: a report of fifteen cases. *Equine Veterinary Journal,* 19, 537–544.

15 Equine Dental Equipment, Supplies and Instrumentation

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Introduction

The early part of the twenty-first century has been an exciting time for those interested in diagnosing equine dental abnormalities and performing corrective procedures. The ever-increasing demand for proper dental care from horse owners/trainers and the interest of veterinarians in providing excellent dental care have created a huge market for dental equipment, supplies and instruments. This increased emphasis and interest has stimulated manufacturers to develop a variety of new and innovative instruments as well as to improve existing equipment methods of physical and chemical restraint. The development of new instruments and methods necessitates education on safety issues and the proper use of the equipment. With this new instrumentation, the equine practitioner can now perform more precise corrective procedures with reduced soft-tissue trauma and less physical effort. The patient's safety and comfort as well as the safety and comfort of the operator and assistants should be a priority at all times.

Dental work area and patient restraint

Dental work can be performed in several types of work areas depending on the stable, farm or clinic setting, patient temperament and veterinarian preference. Certain criteria should be met for safe and efficient delivery of dental services.

The work area should be free of obstacles that could injure the patient and/or operator. If working outdoors is necessary, one should seek a shady or sheltered area with access to water (warm in very cold weather) and electricity. The horse should be confined to a space free of dangerous obstacles. Dental procedures can be performed in an open pasture space but confinement to stocks or an area with solid walls has been found to be much safer and more efficient for work (Fig. 15.1). If working in a stable, the horse can be contained in its stall or a central work area. There are several advantages to working in the horse's own stall. Most importantly, the animal is usually more relaxed and less apprehensive in familiar surroundings. The stall should have sturdy walls and a ceiling tall enough that the horse cannot hit its head should it rear. Again, easy access to electricity and water are necessary. The flooring should drain well and have a non-slip surface. Fans or heaters are welcome additions during hot and cold weather, respectively. A slightly darkened area with light coming from over the operator's shoulder allows better visualization of the patient and the dark recesses of the oral cavity (Fig. 15.2).

A disadvantage to working in a stall is the need to transport the dental equipment and supplies from one stall to another. This makes it necessary to have a mobile work station. Some veterinarians perform dental prophylaxis in the stall working simply from an organized bucket and a clipboard (Fig. 15.3). If more equipment is required, a portable table or cart can be used to move the equipment (Fig. 15.4). Horses can be attended to inside a stall either backed into a corner or standing in the stall doorway (Fig. 15.5).

Some practitioners prefer to work in a designated area of the stable. In such cases, stocks, a wash rack or a farrier's room is suitable. A single equipment set-up in a central location decreases the need to move equipment and supplies from stall to stall. Special mobile dental work stations constructed around stocks or horse trailers have been developed in recent years (Fig. 15.6). The disadvantage to any of these centralized work stations is that the horse must be moved into and out of the work area. Some horses become apprehensive when brought into strange surroundings. Some respond better if given sedation prior to being taken to the work area. They can usually be safely moved while the drug is taking effect. This usually allows for a smoother transition to a working plane of sedation. The horse being worked on must be cleared from the work area before the next patient can be examined. This may require a



Figure 15.1. (A) Portable equine dental stocks. (B) Performing dental procedures using portable stocks, speculum and air-powered floats in a pasture (Clay Stubbs, DVM, Johnson City, TX, USA). With this set-up, dentistry can be performed while comfortably sitting in front of the sedated horse.

long waiting period for a heavily sedated animal to become steady enough to transport back to its stall. The use of sedative reversing agents has helped expedite the recovery process, thereby making work more efficient.[1]

When performing dental work in stocks, several equipment modifications will help. The front door of the stocks should not be more than 3 feet high to allow the relaxed sedated horse to drop its head without compressing its trachea on the door. A butt rope or adjustable rear gate will help keep the horse positioned in the front of the stocks. Head support is needed when working on heavily sedated patients and can be provided with a head stand or suspended metal framed dental halter. If a suspended halter is used, a 3-inch long overhead extension on the stocks will keep the horse's head in a suitable working position. Ponies and miniature horses may be treated efficiently by



Figure 15.2. Unsedated horse being floated in the stall doorway. A half-cup blinker had been placed over the horse's eyes to encourage the patient to lower its head (Ed Barnett, DVM, Bloomington, IN, USA).

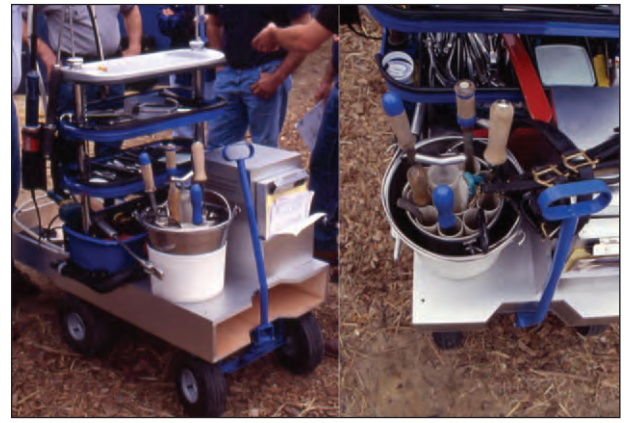


Figure 15.3. Mobile hand cart to transport equine dental equipment from stall to stall.



Figure 15.4. An electric golf cart converted to a mobile equine dental station (TA Banner, DVM, Gainesville, FL, USA).



Figure 15.5. A sedated horse being worked on backed into the corner of its stall. The horse's head is supported in a dental frame suspended from a beam in the ceiling of the stall.

using elevated stocks. If the operator finds himself having to descend to the level of a pony or miniature horse's head, construction worker knee pads make work more comfortable.[2]

Dental head stands vary greatly in cost and complexity, ranging from a padded human crutch to those manufactured specifically for equine dental procedures (Fig. 15.7). Manufactured head stands have several advantages, including rapid adjustment of height, good stability and often, heavy padding as well as a washable cover on which the mandibles can rest.[3] Many stands are designed with attachments for holding equipment and dental instruments (Fig. 15.8 and 15.9).

There are a variety of manufactured rigid dental halters available (Fig. 15.10). In addition, practitioners are very inventive in developing restraint devices and homemade halters, depending on individual preference and need. The full mouth speculum used may determine the type of rigid halter used, as the larger specula will not fit inside some rigid halters. Some mouth specula have been modified to accommodate suspension devices by incorporating a metal arch over the nose piece of the speculum for rope attachment.



Figure 15.6. A custom-designed trailer to perform equine dentistry. This unit contains two sets of stocks. The trailer is self-contained with an electric generator, hot and cold running water, central heating and fans (T Johnson, DVM, Grass Lake, MI, USA).



Figure 15.7. A padded human crutch makes an economical head support stand.



Figure 15.8. Equine dental head support stand. This unit has a wide, stable base and easily adjustable head support. The base pole has hooks and cups to hold dental tools/supplies (T Banner, DVM, Gainesville, FL, USA).



Figure 15.9. A dental head stand that incorporates a wide flat platform with an angle adjustment to provide stability to the sedated horse's head. The padded headpiece can be detached from the stand and used as a head sling when suspended from an overhead support (Cherry and Co. Loogootee, IN, USA).



Figure 15.10. Sedated horse with head suspended by two ropes for greater stability.

However, caution should be exercised when using these devices as damage can occur to the incisor arcades should the horse become excited. Ropes with different types of quick release devices can be used to help steady or restrain the head (Fig. 15.10). Suspending the head with two ropes provides greater stability, but a practitioner working with few assistants may find a single rope with a quick release device more manageable (Fig. 15.11).

Other recommendations on restraint and handling are detailed in Chapters 13 and 16. These chapters should be consulted for more information.

Chemical restraint for dentistry

Commonly used chemical restraint agents for use in equine dentistry and standing oral surgery are classified as tranquilizers, sedative-hypnotics, or opiates. Sedative-hypnotics produce deep depression and have analgesic properties, so they are better suited to dental procedure use than tranquilizers and opiates. However, sedative-hypnotics can be used in combination with tranquilizers and opiates.[4]

The most commonly used sedative-hypnotics stimulate alpha-2 adrenergic receptors, reducing release of various neurotransmitters. Xylazine, detomidine and romifidine are the alpha-2 agonists used most frequently to chemically restrain the standing horse.[5,6] All three of these drugs have a rapid onset of action (1–3 minutes) with duration of action being dose dependent but generally lasting 20–30 minutes for xylazine, 30–45 minutes for romifidine, and 60–120 minutes for detomidine. In addition to their sedative and hypnotic properties, these drugs cause dose-dependent analgesia, muscle relaxation and ataxia. Combining the alpha-2 agonist drugs with opiates or tranquilizers reduces their required doses, thereby minimizing the degree of ataxia that occurs. It is very important to note that horses that appear deeply sedated with these agents can still be startled, even in their relaxed states, and have been known to deliver well-aimed kicks or bites when stimulated.

The alpha-2 agonist drugs have been shown to reduce cardiac output, decrease heart rate and increase central venous pressure. They exert relatively minor effects on respiration but may slightly decrease respiratory rate. No adverse reactions to these drugs have been reported in pregnant mares. However, these drugs have been shown to increase myometrial electrical activity, so caution is advised for their use in mares in the later stages of pregnancy. Other effects of

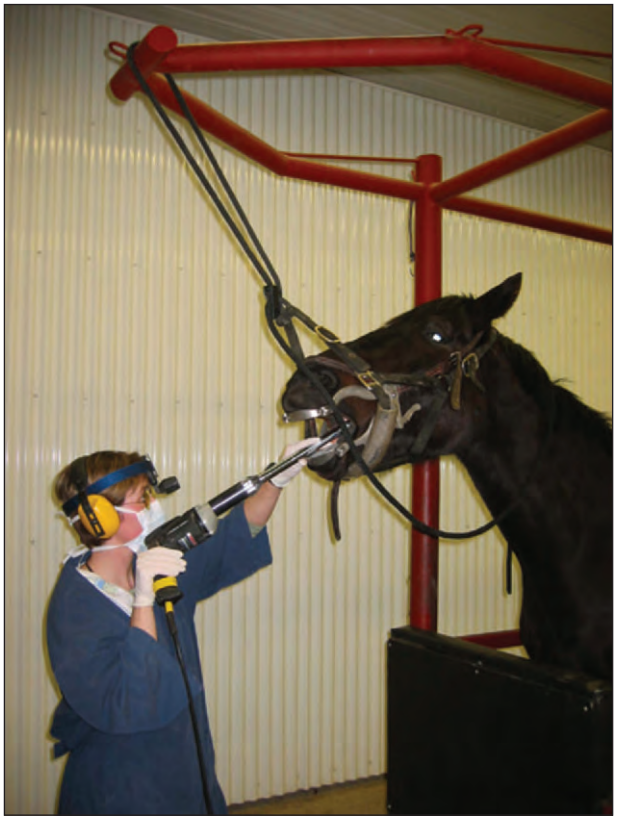


Figure 15.11. Sedated horse suspended by one rope with a quick-release device. Power dentistry is easily performed in this position, allowing excellent visualization of the oral cavity (D & B Equine Enterprises, Calgary, Alberta, Canada).

alpha-2 agonists on the horse include localized or generalized sweating, diuresis, hyperglycemia, hyperthermia, and alterations in gastrointestinal motility.

Reversing the sedative effects of alpha-2 agonists can be achieved with yohimbine or tolazoline. These drugs have been found to be helpful in expediting recovery following heavy sedation. Typically, horses are alert and coordinated within 5 minutes of receiving a slow intravenous dose (30–60 mg) of yohimbine.[1]

Benzodiazepine tranquilizers, such as diazepam, are sedative-hypnotics that produce tranquilization and significant muscle relaxation without analgesia. Diazepam can be used in combination with other sedatives, but ataxia may be a problem. The most beneficial use of diazepam occurs when short-term tongue or jaw relaxation is desired. Chloral hydrate, another sedative-hypnotic, is very predictable in its action. It has historically been widely used in horses but has experienced more limited use in recent years.[7,8]

Opiates can be a valuable component of standing chemical restraint of dental patients. These drugs produce analgesia and variable degrees of sedation or excitement. The increased central nervous system responsiveness demonstrated by a horse in response to administration of certain opiates can be overcome by co-administration of a sedative-hypnotic. Combinations of opiates and alpha-2 agonists used for standing procedures result in good analgesia with a profound sedative effect and minimal ataxia. The mixed agonist/antagonist opiate butorphanol is the most common and reliable drug from this class currently used in equine dentistry.

Numerous drug combinations have been used to pursue the goal of providing better sedation and analgesia and less ataxia than can be achieved with any one drug alone. Each practitioner must work to devise the combination that will work best for individual situations. For example, a deeper degree of sedation than that desired for manual floating is often needed when power instruments are used. Several references are available that can assist practitioners in making decisions on which drugs to use at a particular time.

Equipment for oral examination

Various types of equipment may be employed to conduct a thorough dental examination. As described previously, the horse needs to be prepared using proper physical and/or chemical restraint. Continuing the examination process, the mouth must be held open to allow a complete visual and digital examination. A halter with an oversized nose band will allow the horse to fully open its mouth for inspection and treatment. An adjustable nose band is useful but not essential.

A bucket to hold floats and disinfectant is needed. Steel buckets are often used, but a plastic or rubber bucket will provide protection for expensive, brittle tungsten carbide float blades. If a steel bucket is used, a rubber pad or mat can be placed in its base to help protect float blades. Many instrument companies offer a variety of guards, restrainers or liners for float blade protection (Fig. 15.12).

Some practitioners performing equine dentistry may choose to wear examination gloves for protection. Wearing gloves reduces the number and severity of cuts and abrasions on the hands and fingers caused by sharp enamel projections or the handling of rasping instruments. Most practitioners should carry a hand brush, medicated soap or disinfectant and towel to clean their hands and nails.

Dose syringes are widely used to rinse mouths and are available with either a pistol grip or in a pistol style. The patient's mouth should be rinsed with water (and dilute chlorhexadine as needed) prior to the intra-oral portions of the dental examination in order to facilitate proper visualization of the teeth and associated soft tissues. The rounded blunt end of the nozzle on the large plastic dose syringes helps prevent accidental oral injury during flushing (Fig. 15.13). A high pressure rinse can be obtained with instruments like the Power Flush (Stubbs), which attaches directly to a hose (Fig. 15.14).

Several types of gags and specula can be utilized to visually and manually evaluate the mouth. These instruments work via insertion into the mouth between the cheek teeth arcades. Round, one-sided gags made from PVC or metal (as is the case with spool or coil gags) may be covered with rubber, polyurethane, or neoprene. Dental fracture is a possible



Figure 15.12. A bucket with a rubber bottom and dividers to protect float blades from damage (Jorgensen Laboratories, Loveland, CO, USA).

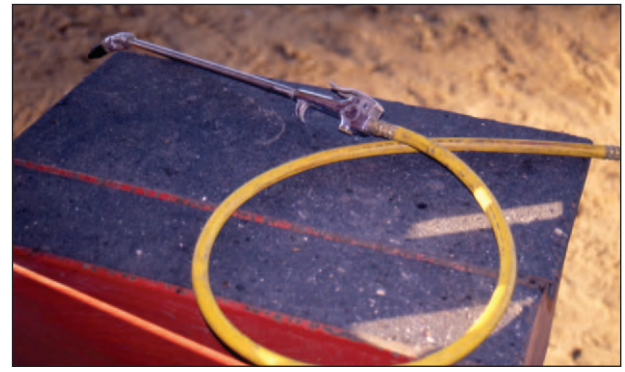


Figure 15.14. Oral irrigation device with a trigger and guarded stray tip. This device attaches to a regular water hose (Clay Stubbs, DVM, Johnson City, TX, USA).

complication resulting from use of metal spool or coil gags because only a small part of a given tooth rests on the hard gag (Fig. 15.15). Wedge-shaped gags may be covered with rubber, polyurethane or neoprene, or may simply consist of metal. Such gags are safer than spool or coil gags as they allow several teeth to contact the wedge simultaneously. Heavily sedated horses may still chew forcibly and often appear painful following the use of a one-sided gag, possibly from stretching of the temporomandibular joint capsule on the side opposite the gag. World Wide Equine, Inc. manufactures a double Jeffrey mouth gag made from polyurethane that may help alleviate this problem. The use of gags should be confined to the treatment of the incisor teeth. However, generally and in practice, their use is limited for cheek teeth dentistry because they push the tongue against the opposite arcade and do not allow unrestricted access to the cheek teeth.

Full mouth specula are especially helpful in performing both complete, detailed dental examinations and precise corrective procedures. These instruments work by keeping the mouth open with the plates on the incisor arcades. There are three categories of full mouth specula: (1) lightweight, collapsible ratchet speculum (McPherson, Haussmann and Series 2000), (2) screw type speculum (Gunther, Stubbs and Butler), (3) oversized, hinged type speculum (Conrad and McAllen) (Fig. 15.16).

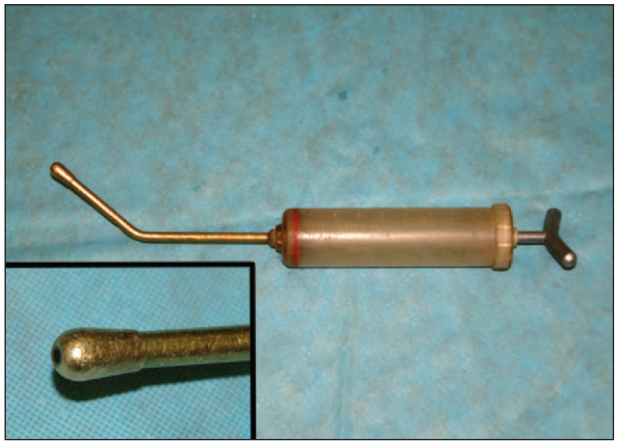


Figure 15.13. A 450 ml drenching dose syringe with a blunt brass tip. This syringe is well suited to safely rinse a horse's mouth (Alberts Equine Dental Supply, Inc., Old Chatham, NY, USA).

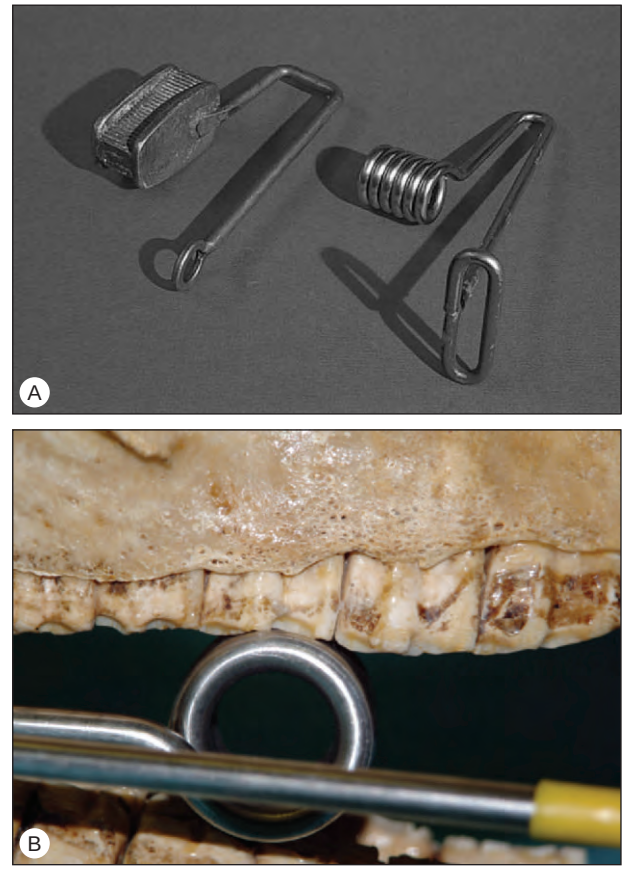


Figure 15.15. (A) Two different dental gags. The Landmesser wedge and the spool or coil gag. (B) This figure illustrates the small area of tooth surface in contact with the spool gag. Solid round gags have caused dental fracture and loosening.



Figure 15.16. Four different equine full mouth specula. From left to right: (1) Meister – no longer manufactured; (2) Stubbs – Stubbs Equine Innovations, Johnson City, TX, USA; (3) McPherson – Alberts Equine Dental Supply, Inc., Old Chatham, NY, USA; and (4) Conrad – Spencer Equine Services, Groveland, FL, USA.

The McPherson or Haussmann types are the most widely used and most economical specula. The Series 2000® is a modification of the ratchet speculum with a greater number of adjustments. With McPherson, Haussmann and Series 2000 specula various sizes and shapes of plates are interchangeable. Gum plates are designed to be used on horses with incisor malocclusions (or on cattle) (Fig. 15.17). Offset incisor plates are available for horses with underbite and overbite (parrot mouth and monkey mouth). A 2.5-inch-diameter plastic pipe, 4 inches long, can be used to cover the incisor plates allowing the speculum to be used as a wedge in the interdental space, thus exposing the incisor teeth. A single threaded bolt opens the mouth with the Stubbs or Gunther screw type speculum. With the Stubbs speculum, the bolt can conveniently be positioned on either side of the mouth (Fig. 15.18). The Stubbs or Gunther specula are best suited for oral surgery when the horse is in lateral recumbency.

Conrad, Meister and McAllen hinged type specula are best suited for oral examination and therapeutic procedures in a heavily sedated horse. Because of their large size and weight these specula can be dangerous in horses that are not adequately sedated and/or well restrained.

Safety is of utmost importance when a full mouth speculum is in use. A horse that swings its head suddenly can cause serious injury to anyone in the vicinity. Each person working around a dental patient wearing a speculum should be constantly mindful of the horse's demeanor, stability and activity. The examiner's hand should be placed on the bridge of the horse's nose as much as possible to steady its head.

To best visualize the oral cavity, an appropriate light source must be utilized in addition to a full mouth speculum. Illumination is especially important during performance of corrective procedures with motorized instruments. Hand-held flashlights or lamps mounted on the floor or ceiling can be used. The Spec Lite® attaches to the incisor plates of most specula and allows for continuous illumination of the mouth (Fig. 15.19). Its use minimizes the amount of head movement the operator must make to maintain visualization of the oral cavity.

Head-mounted lights are most useful for oral cavity illumination. Numerous types of head lights are available (Fig. 15.20). Some have been adapted especially for equine dentistry, but many are lights intended for a variety of other



Figure 15.17. McPherson speculum with upper gum plate. This speculum is configured in this manner to fit the mouth of a parrot-mouthed horse with an incisor overjet.

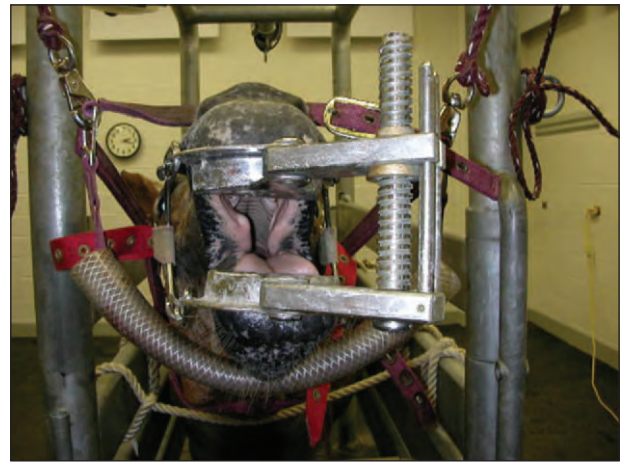


Figure 15.18. The Stubbs full mouth speculum uses a single threaded bolt to hold the mouth open. The threaded bolt can be swung to either side for greater access to the mouth (Stubbs Equine Innovations, Johnson City, TX, USA).

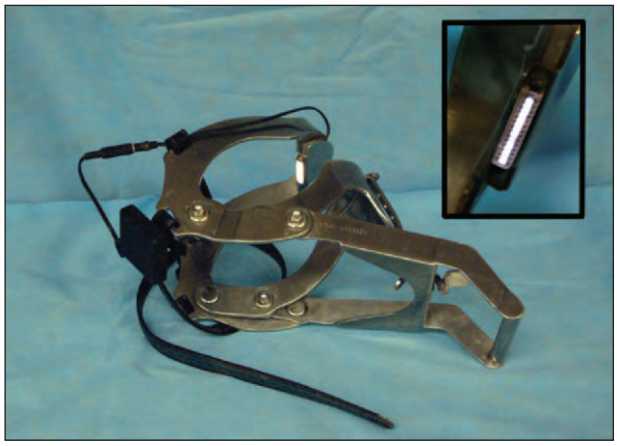


Figure 15.19. A battery-powered light (Spec Lite®) attached to the upper incisor plate of a Conrad speculum. This light allows continuous unobstructed illumination of the oral cavity (Rena's Equine Dental Instruments, Reno, NV, USA).



Figure 15.21. An innovative use of one battery-powered lamp. This detachable light source can be used on a head lamp, basket speculum or long, rigid-handled, intra-oral mirror (Stubbs Equine Innovations, Johnson City, TX, USA).

activities. These head lights range in price from the high teens to several hundreds of dollars. Stubbs Equine Innovations offers a multipurpose light that can be used as a head light, but can also be used to illuminate the Stubbs Arcade Speculum or Stubbs Intraoral Mirror (Fig. 15.21).

Once the oral cavity is adequately illuminated, instruments such as picks, probes, mirrors and retractors can be used to continue the oral examination and further investigate any potential problems. Dental picks are useful in clearing debris from and probing diastema and crown defects. They are also helpful in extracting tooth and root fragments in aged horses (Fig. 15.22). Some practitioners have adapted a wire coat hanger as a useful and inexpensive dental pick or probe. Additionally, an insemination pipette, curved by being held over a flame just long enough to make the plastic bendable, is a useful dental probe and flushing device. Special calibrated human or small animal periodontal probes can be modified on a long handle and can be used to explore and measure periodontal pockets.

Using a mirror in conjunction with dental picks and/or probes is helpful in allowing the practitioner to visualize identified abnormalities in the mouth (Fig. 15.23). Mirror fogging can be a problem when working in the oral cavity, particularly in cold weather. Warming the mirror or using an antifog wipe or spray designed for reading glasses can be helpful in eliminating this problem. Most mirrors will need to be set at a 35°–45° angle to their handles to allow adequate visualization around the cheek teeth.

At various points during the dental examination, the practitioner may find it helpful to have an assistant retract



Figure 15.20. Four different head lights that can be used when performing equine dentistry. From left to right: (1) a fiberoptic surgical head lamp (Stubbs Equine Innovations, Johnson City, TX, USA); (2) a small portable battery light with head strap (Cherry and Co., Loogoote, IN, USA); (3) an electric halogen head lamp (Pelican Products, Torrance, CA, USA); and (4) a battery-powered lamp with rigid head frame (Light-Tech, Inc., Sebring, FL, USA).

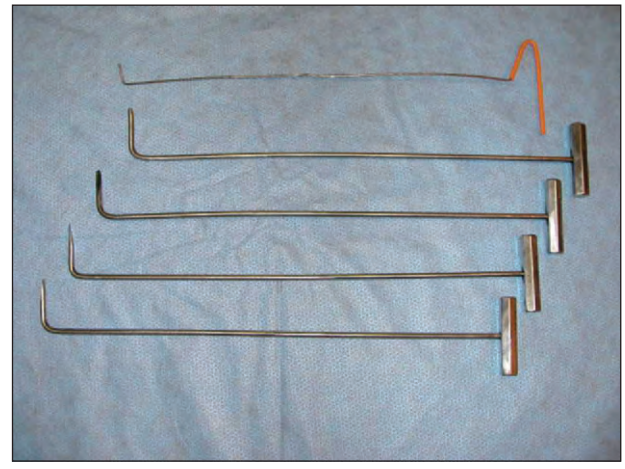


Figure 15.22. A variety of dental picks, probes and fragment elevators. The dental pick at the top was made from a wire coat hanger. The four lower fragment elevators have blades positioned to enable one to approach root fragments from different angles (World Wide Equine, Inc., Glenns Ferry, ID, USA).

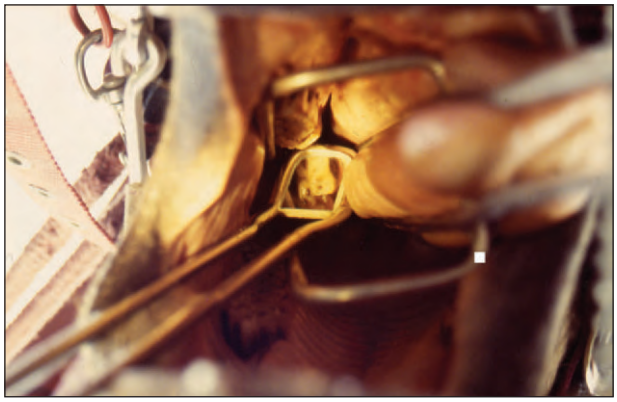


Figure 15.23. Using a dental basket retractor and intra-oral mirror to examine the occlusal surface of the upper dental arcade (Stubbs Equine Innovations, Johnson City, TX, USA).

the tongue and/or cheeks of the patient. An abdominal retractor or a specially designed equine dental basket retractor may be utilized to provide more adequate oral visualization (Fig. 15.24).

Once potential dental abnormalities are identified, imaging modalities such as radiographs and ultrasound may be utilized to gain more information about the problem at hand. A digital camera and/or video equipment can be used to identify and record lesions and document the performance of various procedures. This documentation is a useful record of care and can be used to educate clients and other veterinarians.

Dental floating equipment

Manual floats

Manual floats continue to be widely used in equine dentistry. The variety of blades, heads, shafts, and handles currently available to practitioners is extremely useful in performing prophylactic procedures. The most durable and aggressive float blades are made from solid tungsten carbide. While these blades historically have been relatively expensive, they are so effective and efficient that many practitioners now use them. Most of these blades originally consisted of a tungsten carbide rasp blade bonded to a plate of stainless steel, variably sized to fit the desired float head. More recently, tungsten blades have been directly bonded to the float head or shaft, resulting in a slimmer float design. Carbide blades cut in only one direction, so care must be taken to ensure that they are properly set on the float to cut either on the push or pull, depending on the desired use.

Tungsten carbide blades range in classification from ultra-fine to coarse, with each manufacturer having individual scales of aggressiveness (Fig. 15.25). The finer (less aggressive) blades stay sharp longer and can be resharpened more times than the coarser (more aggressive) blades. Most blade manufacturers offer an economical sharpening service to practitioners. In general, the fine and medium blades are best for general floating, while the coarse blades are reserved for reducing large overgrowths. Though these blades are sharp, the teeth of the blades are brittle, so one should handle them with care. Since different parts of the blade act as the cutting area when used in different handles, blades can be switched to different handles as they become dull in order to get additional use before they need to be resharpened. It is, however, important to recognize when a blade is completely spent and needs to be sharpened or replaced.

Other blade designs are available in addition to tungsten carbide, including carbide grit blades, steel file (Dick) blades, and Jupiter Speedy Cut float blades (Fig. 15.26). Carbide grit blades come in various sizes of grits or coarseness and cut in both directions, i.e. on the push and the pull. The Dick blades have been discontinued by most companies because the steel blades are inferior to carbide products in durability. The Jupiter blades are made of either stainless steel or tungsten carbide and are also designed to cut in both directions.

The standard float blade is 3 inches long by 1 inch wide and has square corners. Changes in the size and shape of



Figure 15.24. The basket retractor with an attached light positioned between the upper and lower dental arcades on the specimen (Stubbs Equine Innovations, Johnson City, TX, USA).

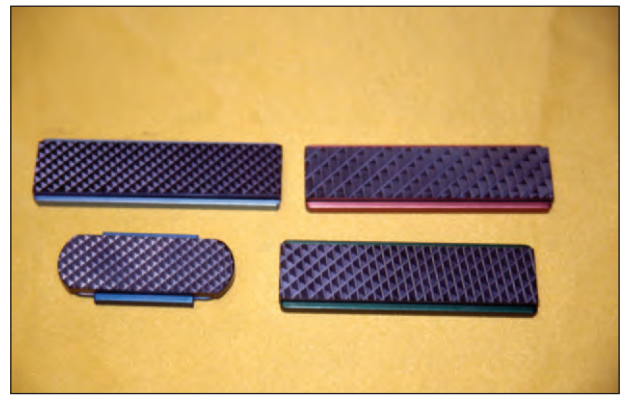


Figure 15.25. Four tungsten carbide float blades in fine, medium and coarse grades. Two different blade configurations: (1) standard box float blade and (2) short, rounded corner blade (Alberts Equine Dental Supply, Inc., Old Chatham, NY, USA).

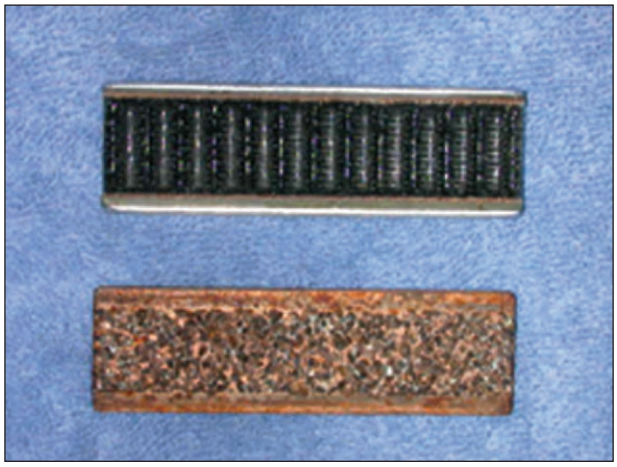


Figure 15.26. The Jupiter Speedy Cut blade (top) and carbide chip blade (bottom) fit most float heads and cut in both directions (on push or pull stroke) (Jupiter Veterinary Products, Harrisburg, PA, USA).



Figure 15.28. An innovation in solid tungsten carbide float head and blade design. These convex blades are very useful in reducing dental overgrowths (Kruuse Worldwide, Marslev, Denmark).

blades have recently been introduced by several manufacturers. Capps manufactures a trough float head that holds triangular tungsten carbide blades (Fig. 15.27). Kruuse has recently modified the shapes of their tungsten carbide float blades, making work on the table surfaces of the dental arcades more efficient (Fig. 15.28).

Manual dental floats are constructed in a variety of configurations. Float heads should be made to cover the sharp corners of the blade and fit the area of the mouth to be floated. Float head weight and thickness will vary depending on intended use and operator preference. Float shafts may be round, three-quarter round or flat. Round shafts slide through the operator's fingers more easily than flat shafts. Flat shafts allow the operator to more accurately assess the blade's angulation in relation to the tooth being floated. Three-quarter round shafts combine the advantages of both the round and flat shafts. Float handles may be constructed of wood, plastic, metal, rubber or molded acrylic and can be padded or unpadded. They may be configured in either a pistol grip or a shaft grip and should be an appropriate size for the hand of the operator.

Different shapes and lengths of floats are employed to reduce sharp enamel points on the various teeth, create bit seats, and reduce more major overgrowths. This is discussed in detail in Chapter 16. A minimal set of floats should consist of: (1) a long, straight float, (2) an angled float, (3) a short, straight float and, (4) a back upper molar float (Fig. 15.29).

Practitioners may want to consider a variety of specialized floats for their dental equipment selection. A long offset float may be used to float the lower dental arcades. A float consisting of a 9–15 inch shaft, offset head and short blade box is used to create and/or 'polish' bit seats (Fig. 15.30). Some manufacturers offer smaller floats made specifically for miniature horses (Fig. 15.31), and extra-long handles can



Figure 15.27. A set of Capps® floats with a triangular carbide blade that stacks in a trough-shaped float head (World Wide Equine, Inc., Glenns Ferry, ID, USA).



Figure 15.29. Four basic floats used to perform routine dental prophylaxis. From top to bottom: (1) round medium-length shaft, 20° downward-angled, box head float with rubber handle, (2) flat long-shaft, 10° upward-angled, bonded head float with neoprene handle, (3) round, short-shaft, straight, box head float with rubber handle, and (4) round, long-shaft, straight box head float with rubber handle.

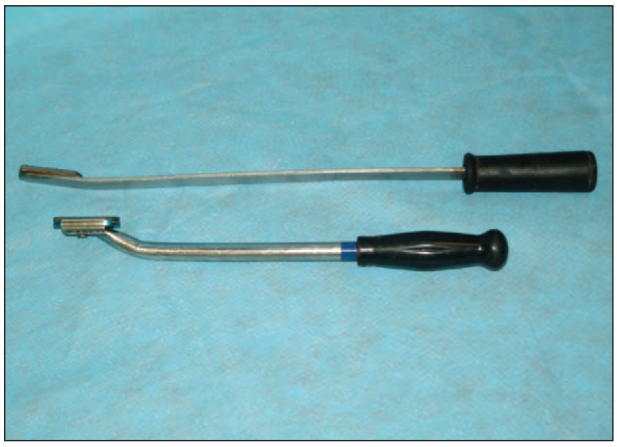


Figure 15.30. Two very useful dental floats: (top) flat, long-shaft, 10° upward-angled, bonded head float with a neoprene handle to reach the caudal edges of the molars, and (bottom) a round, medium-length shaft, offset open box head float with a rubber handle to round bit seats on the rostral premolars (FXA Float®, Alberts Equine Dental Supply, Inc., Old Chatham, NY, USA).



Figure 15.32. A steel S-shaped double end file and large carbide grit table float.

be purchased for use in large/draft horses. Carbide-chip S-floats and steel files are often used to polish bit seats, smooth incisors and reduce canines (Fig. 15.32). A long, wide S-float, also called a table float, can be used to reduce sharp areas in horses with wave mouth or other types of uneven arcades. These floats are often sold with grit on both sides of each end. This can be a disadvantage if the float is used to smooth the buccal and caudal aspects of the upper 10s and 11s as the 'outside' gritted area will damage the mucosa. Practitioners who prefer to use the S-float may want to consider purchasing floats with grit on only one side of each end (one end being convex and the other end being concave).

Power instruments

Motorized instruments were first used in equine dentistry in the 1930s. These instruments are now generally classified as reciprocating, rotary cable, rotary drum or rotary disk, and may be electric, battery or pneumatic powered. Variable speed control is helpful to prevent excessive crown reduction and minimize soft tissue damage. Regardless of burr type, rotary instruments should be used at low speeds (<8000 rpm).[9] Many reciprocating floats are electric or battery powered and are modifications of woodwork power saws, varying in stroke length and strokes per minute (Fig. 15.33). Several manufacturers (Stubbs, Olsen, and Silk and Carbide Products) make pneumatic reciprocating floats that make short strokes at high speed. Some operators have developed hand problems caused by the vibration of pneumatic floats and it is suggested that antivibration gloves be worn when performing corrective procedures with these floats. Most of these instruments have a thin carbide blade bonded to the shaft, similar to those used on some manual floats (Fig. 15.34). Long-stroke reciprocating floats are also available and are used to reduce excess crowns and sharp enamel points. Long-stroke reciprocating floats should not be used on the

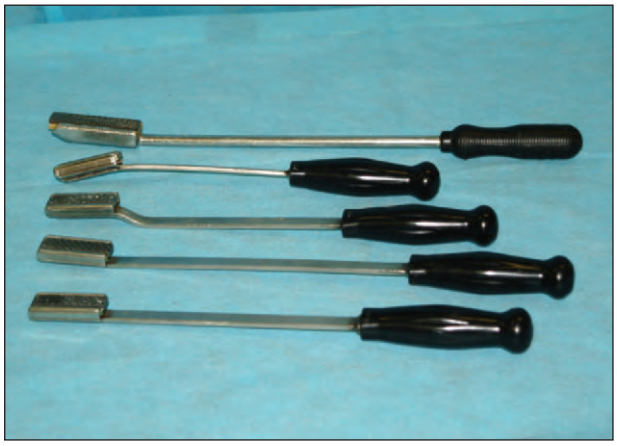


Figure 15.31. The top float is made for use in a normal-sized horse. The bottom four floats are of reduced size (both in shaft length and diameter – box head and blade dimensions) and are used on ponies or miniature horses.

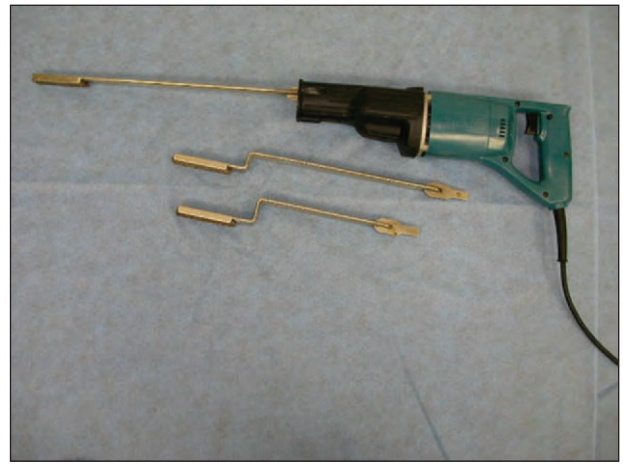


Figure 15.33. An electric-powered long-stroke reciprocating float with interchangeable shafts and float heads (Rena's Equine Dental Instruments, Reno, NV, USA).

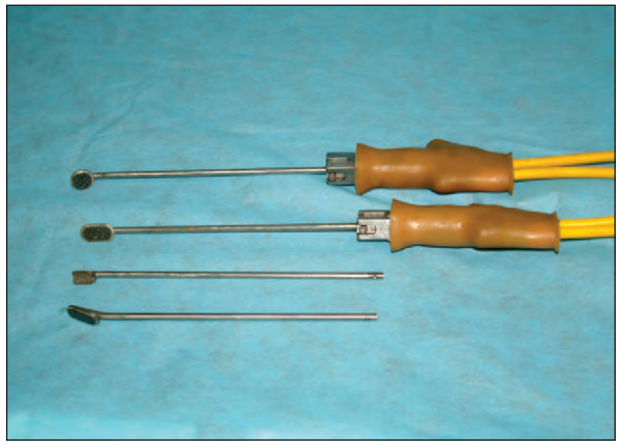


Figure 15.34. Hand pieces from air-powered, high-speed, short-stroke reciprocating floats with interchangeable shafts. These float heads have slimline, bonded blades (Stubbs Equine Innovations, Johnson City, TX, USA).



Figure 15.36. Rotary dental grinder constructed from a Suhmer motor with clutch, a variety of guards, vacuum system to remove dental dust and various style heads and burrs (Carbide Products Co., Torrance, CA, USA).

third molars due to risk of iatrogenic mandibular or soft-tissue trauma. A polymer lubricant used with reciprocating pneumatic instruments will reduce heat produced by friction and airborne dust.

Rotary cable floats are quite effective in removing sharp enamel points and reducing overgrowths (Fig. 15.35). However, the Dremel®, a type of rotary cable grinder, should not be used for equine dental work due to the electric shock hazard of working in a damp or wet environment (i.e., the mouth). Dremel motors are not endorsed by the manufacturer for equine dental work.[10] Rotary cable floats are used in the front of the mouth to reduce incisors and canines and to create bit seats. An assortment of guards, the most simple of which are those fashioned from PVC pipe, are available to help prevent soft-tissue trauma with the use of these floats. Several manufacturers have various lengths of guards and extensions to facilitate good control and minimize soft-tissue damage in all areas of the oral cavity. Some power instruments have built-in vacuum systems that reduce the operator's exposure to dental dust and improve visibility (Fig. 15.36). Other units may have irrigation systems that reduce dust and decrease the risk of thermal damage to the teeth. Some units contain both a light source and an irrigation system (Fig. 15.37). A built-in clutch makes these units safer for the horse, reducing soft-tissue damage and decreasing the incidence of cable breakage. Solid tungsten rotary-powered burrs are available in a variety of cutting teeth and degrees of coarseness. A fine cross-cut burr will not tend to jump off the tooth during rasping, as is often the case with spiral-cut burrs. Burrs or grinding drums coated with fine carbide grit or diamond chips are available in a variety of shapes.

Disk burr instruments have become increasingly popular as they are less apt than other power instruments to damage



Figure 15.35. A flex-shaft with variable-speed foot pedal, Dremel® motor tool modified to perform dental corrective procedures. The white PVC pipe guard is homemade. The stainless steel guard and extended shaft burrs have been designed for corrective procedures on incisors, canines and premolars (Dremel®, Racine, WI, USA; Harlton's Equine Dental Specialties, Columbus, OH, USA).

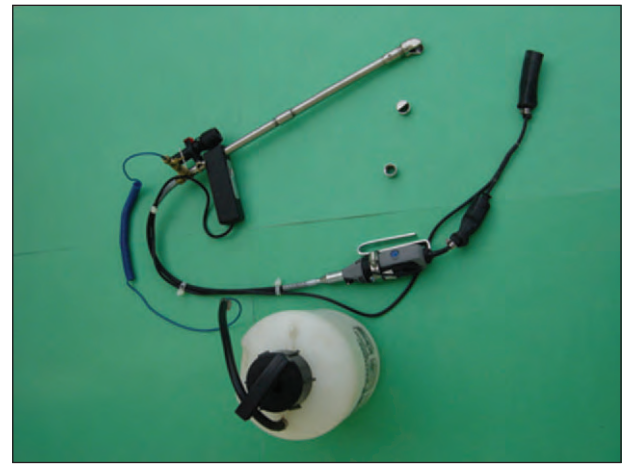


Figure 15.37. Variable-speed flexible-shaft tooth grinder with long shaft and compact, guarded head. This unit has a built-in water irrigation unit and light (Jupiter Veterinary Products, Harrisburg, PA, USA).

the soft tissues inside the oral cavity. Additionally, it appears to be easier for an operator to master the use of disk type motorized instruments. These instruments are manufactured with various lengths of shafts and run from fixed electric or battery-powered drills or flexible shaft motors. Instrument head design and thickness varies between manufacturers. The cutting surface of the disk is made from solid tungsten carbide, fine carbide grit or diamond dust. Examples of these instruments include the Eisenhut (SwissFloat)®, the PowerFloat® and the Horsepower® hand piece. The Eisenhut consists of a hand-held electric drill motor with a 4 cm circular stainless steel, carbide or diamond disk that rotates horizontally at the end of a shaft most commonly 65 cm in length. This instrument is useful in reducing caudal mandibular overgrowths even in large horses due in large part to the length of its shaft.[10] The PowerFloat® is comprised a 2.5 cm tungsten carbide chip disk that rotates horizontally at the end of a 45 cm long shaft (Fig. 15.38). This low-profile power instrument can be used for numerous corrective procedures including caudal 11 overgrowths in small ponies. The Horsepower hand piece is modified to fit the Dremel® motor.

The use of power instruments in equine dentistry has generated controversy, but when used correctly, they allow one to perform precise corrective procedures with minimal soft-tissue trauma. Since more tooth can be removed with less physical exertion on the part of the operator, excessive crown removal and even pulpar exposure has occurred in some cases. Operators should be especially observant when these instruments are being used.[9–11]

Safety of the operator, his assistant(s) and the equine patient during motorized instrument use can be optimized in several ways. Protective eyewear and an air filter mask reduce the chance of debris and tooth dust getting in the eyes and/or being inhaled. Ear protection should be considered if loud electric motors are used close to the operator's head or noisy air compressors are in operation nearby. When using power floats or any electric-powered instrument for dental procedures, a ground fault circuit interrupter should be used to reduce the chance of electric shock (Fig. 15.39). Battery powered motorized instruments reduce the possibility of electrical shock, but they require additional batteries and chargers for continuous operation. Pneumatic instruments reduce the chance of electrical shock but require a noisy compressor motor or bottled compressed gas for operation.



Figure 15.38. Battery and electric-powered disk burr (PowerFloat®). The carbide chip or diamond grit rotary disk head is guarded by a stainless steel lip (D & B Equine Enterprises, Inc., Calgary, Alberta, Canada).



Figure 15.39. An extension cord with a ground fault circuit interrupter (GFI) incorporated in the plug. A GFI should be used whenever electric tools are used for dentistry to prevent the possibility of electric shock.

It is important to minimize the heat produced by power instruments so that thermal dental pulp trauma does not occur. This is accomplished by using only clean sharp blades, burrs or disks, working on the same area of tooth less than 10 consecutive seconds and irrigating the tooth with water while burring. Water cooling eliminates all potentially pathogenic thermal effects of motorized instrument use.[11] A scanning electron microscopic study of the effects of three different types of rasps (solid carbide, tungsten chip, and electrically driven rotating burr) on the occlusal surface of equine cheek teeth revealed significant physical damage to the teeth by all three types of rasps.[12] The use of all three rasps caused amputation of odontoblast processes, predisposing the teeth to bacterial infection. The authors of this study suggest that the damage sustained by the dentin may cause pain to the patient. They offer the following advice to preserve the integrity of the pulp: (1) use light, intermittent cutting, (2) use an efficient cooling system and high speeds of rotation, (3) avoid desiccating the dentin and (4) do not apply irritating chemicals (i.e. mouthwashes) to freshly cut dentin. Dixon suggests that post-procedure oral pain is due to damage of the dentin and deep exposure of odontoblast processes leading to pulpar inflammation.[13]

Special dental equipment

Molar cutters and chippers

The use of percussion instruments to reduce overgrowths has occurred since early in the nineteenth century. Chisels and mallets or chisels with sliding captive bolt hammers have also been used to reduce enamel points as well as rostral and caudal hooks. The Equi-Chip® guarded chisel with a sliding hammer is a refinement of earlier devices. This instrument is no longer widely used, since power tools have proven to be more efficient, precise and safe in removing these types of overgrowths (Fig. 15.40).

Molar cutters come in various sizes and configurations and are best suited for removing isolated tall teeth (step mouth) or selected tall hooks. They have largely been replaced by the previously described powered instruments that can more safely and controllably reduce major overgrowths. The blades of molar cutters should be parallel to one another when making contact with the sides of the tooth to be fractured. Compound action cutters with a D-head are usually required to reduce maxillary teeth. C- or D-head simple action molar cutters can be used to reduce mandibular cheek tooth elongations. Simple joint B- or C-head cutters have been used to reduce rear lower molar (311, 411) hooks (Fig. 15.41). Care should be taken to precisely place the cutter on the tooth as iatrogenic tooth fracture and pulp exposure can result from improper use of these instruments.

Deciduous tooth instruments

Various forceps, elevators and dental picks are available to remove retained, displaced or broken deciduous cheek tooth remnants (caps). A set of deciduous tooth instruments should consist of: (1) short-handled molar forceps and (2) dental elevators or a modified screwdriver. The use of these instruments is outlined in Chapter 16.



Figure 15.40. The Equi-Chip® guarded dental chisel with a captive bolt, sliding hammer built into the shaft. The chisel head is guarded and designed to cut a rear tooth elongation on the pull or a front tooth elongation on the push (Alberts Equine Dental Supply, Inc., Old Chatham, NY USA).

Canine tooth instruments

Instruments have been developed to reduce the crown of sharp canine teeth as well as to scale and buff older canine teeth with tartar accumulation. A full set of canine tooth instruments consists of: (1) a straight-handled, fine-grit, tungsten carbide dental rasp, (2) a small dental scaler, (3) wire-bristle toothbrush or nylon-bristle nail brush and (3) Oral Cleansing Gel® (Addison Biological Laboratory, Fayette, MS). Care of canine teeth and other use of instruments are outlined in Chapter 16.

Dental extraction equipment

Removal of cheek teeth, wolf teeth, canines and incisors requires special equipment and supplies (Fig. 15.42). Special operating room support is required in certain circumstances. Equipment and techniques for tooth removal are outlined in Chapter 18.

Periodontic, endodontic and dental restorative materials

Areas of growing interest in equine dentistry include periodontal disease and infundibular decay.[9] Specialized equipment, instruments and medication are available to treat equine periodontal disease. Pacific Equine Dental Institute, Inc. has adapted and modified human and small-animal dental equipment for use in the equine patient. The Equine Dental System® (Pacific Equine Dental Institute, Inc., El Dorado Hill, CA) is a self-contained, high-pressure (up to 200 psi) water delivery spray unit that can be used to evacuate deep periodontal pockets. Additionally, this system contains a Prophy Air Abrasion Unit and a baking soda/chlorhexidine delivery system to clean out periodontal pockets and areas of infundibular decay. With this system, the equine practitioner can now provide the same level of periodontal care that is provided in human dentistry.[14,15]

As in human dentistry, endodontics, orthodontics and crown restorative techniques are now being utilized in equine dentistry. The equipment and materials needed for such detailed techniques are outlined in Chapters 17, 19 and 20.



Figure 15.41. Compound and simple action molar cutters. These instruments should be used with caution when reducing tall teeth.



Figure 15.42. Various forceps, elevators and a mallet used to remove wolf teeth (L Scrutchfield, DVM, College Station, TX).

Conclusion

Many dental vendors and manufacturers can provide the equine practitioner with high quality instruments and supplies. Details of suppliers are given as an appendix. As equine veterinary dentistry progresses and evolutes, equipment and supply needs will continue to change. Continuing education and instruction on the use of supplies are ongoing processes. The equine practitioner should take advantage of current literature as well as participate in dental seminars and wet labs to stay abreast of the latest developments.

REFERENCES

1. Allen T, Johnson T and Miller RO (2003) Safety issues and restraint procedures. In: *Manual of Equine Dentistry,* ed. T Allen, Mosby, St Louis, pp. 43–48.
2. Schultze W, Allen T, Mitz C and Johnson T (2003) Dental tools and equipment. In: *Manual of Equine Dentistry,* ed. T Allen, Mosby, St Louis, pp.17–41.
3. Pence P (2002) Dental equipment in equine dentistry. In: *Equine Dentistry: A Practical Guide,* ed. P Pence, Lippincott, Williams and Wilkins, Philadelphia, pp. 25–52.
4. LeBlanc PH (1991) Chemical restraint for surgery in the standing horse. In: *Veterinary Clinics of North America: Equine Practice,* 7(3), 521–533.
5. Kannegieter NJ (1993) The use of romifidine as a sedative in the horse. *Australian Equine Veterinarian,* 11(2) (insert).
6. Baker GJ and Kirkland K (1995) Sedation for dental prophylaxis in the horse: a comparison between detomadine and xylazine. In: *Proceedings of 42nd Annual Convention of American Association of Equine Practitioners,* p. 40.
7. Thurman JC and Benson GJ (1987) Injectable anesthetics and anesthetic adjuncts. In: *Veterinary Clinics of North America: Equine Practice,* 3, 15.
8. Muir WW (1981) Drugs to produce standing chemical restraint in horses. In: *Veterinary Clinics of North America: Large Animal Practice,* 3, 17.
9. Greene S (2001) Equine dental advances. *Equine Practice,* 17(2), 319–334.
10. Dacre KJP, Dacre IT and Dixon PM (2002) Motorized equine dental equipment. *Equine Veterinary Education,* 4(5), 337–340.
11. Baker GJ and Allen ML (2002) The use of power equipment in equine dentistry. In: *Proceedings of 48th Annual Convention of American Association of Equine Practitioners,* pp. 438–441.
12. Kempson SA, Davidson ME and Dacre IT (2003) The effect of three types of rasps on the occlusal surface of equine cheek teeth: a scanning electron study. *Journal of Veterinary Dentistry,* 19–27.
13. Dixon PM (2002) The gross, histological and ultrastructural anatomy of equine teeth and their relationship to disease. In: *Proceedings 48th Annual Convention of American Association of Equine Practitioners,* pp. 421–437.
14. Pence P and Basile T (2002) Dental infection in equine dentistry. In: *Equine Dentistry: A Practical Guide,* ed. P Pence, Lippincott, Williams and Wilkins, Philadelphia, pp. 209–229.
15. Allen T (2003) Other procedures. In: *Manual of Equine Dentistry,* ed. T Allen, Mosby, St Louis, pp. 157–173.

16 Corrective Dental Procedures

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Introduction

Corrective dental procedures have been practiced on equine patients for hundreds of years. They have been shown to be an important part of a horse health care program. Corrective procedures are often performed: (1) to relieve discomfort associated with oral soft tissue injuries caused by sharp enamel points, (2) to improve mastication and digestion of feedstuffs, (3) to alleviate stresses on abnormally worn teeth, and (4) to prevent discomfort associated with the bit. The benefits of dental floating on feed digestibility in the horse have been critically evaluated.[1–3] Improved athletic performance and responsiveness to the bit have been documented in 20 horses post dental floating.[4] The analysis of videotapes of the mastication cycle reveals a small rostrocaudal component to the otherwise elliptical chewing cycle.[5] This rostrocaudal mobility of the mandible also occurs as the horse raises and lowers its head in collection. Floating has been shown to increase the rostrocaudal mobility of the mandible. It has been suggested that measurement of rostrocaudal mobility of the mandible can be used as a guide to determine whether dental correction is necessary. After dental floating, the measurement can be used to ensure that irregular occlusal surfaces have been corrected.[6]

Taking a moment to educate trainers and owners about the value of a thorough dental examination, types of pathology and indicated dental corrective procedures is time well spent. Dental forms or charts should be used to record abnormalities, corrective procedures performed and any planned treatment. Dental forms will also help in itemizing the bill and in providing an estimate of professional fees before procedures are performed (see Chapter 13).

There are four distinct levels of equine dental care. The first is referred to as dental prophylaxis. Historically, this level of dentistry has been labeled 'floating teeth.' It involves an oral examination and routine dental maintenance procedures such as reduction of sharp enamel points. This level of dentistry may also involve reduction of small abnormal enamel projections (hooks, beaks, small waves and transverse ridges) to better balance the mouth and allow comfortable mastication and even dental wear. The second level of dentistry is often referred to as performance dentistry. This includes dental prophylaxis in addition to procedures developed to aid the horse's comfort in accommodating the bit or other equipment (tongue tie, hackamore, nose band, martingale). Such procedures may include creating bit seats, removal or reduction of wolf teeth, smoothing canine crowns and balancing the molar and incisor table heights and angles. Corrective dentistry describes the third level of equine dental care and involves procedures devised to reduce excess dental crown and associated pathologies. Dental overgrowths may involve a portion of a tooth (hooks, abnormal transverse ridges), the entire tooth (step, ramp), several teeth (wave) or the entire arcade (shear mouth). This level usually requires a more thorough examination and precise planning for correction. Oral dental surgery, orthodontics and endodontics, the fourth level of equine dentistry, will be covered elsewhere in this text.

It is not always possible to assign an equine patient to a level of dental care prior to making clinical contact. A dental history and physical examination along with a complete oral examination and evaluation of lateral jaw excursion will usually establish the level of care the horse requires. Occasionally, the veterinarian may be involved in what appears to be a routine prophylaxis when a decayed, loose or broken tooth is encountered. This may move the horse into a higher level of care.

Dental corrective procedures should be undertaken only after a complete dental examination. For effective oral examinations and to carry out many dental procedures, a full mouth speculum is required. The veterinary practitioner should develop a routine of examination, recording findings and outlining a plan of treatment. Maintenance and/or corrective procedures should be carried out in a systematic and efficient manner. Any procedure should be well planned with proper equipment and instrumentation available. Horses requiring special diagnostic and therapeutic oral procedures should be referred to veterinarians with the equipment and expertise to properly diagnose and treat such problems. In correcting dental abnormalities, it has been found most effective to work from the front of the mouth caudally and then move to the evaluation and correction of incisor abnormalities.

Two approaches to dentistry have become standard over the past few years. Both involve examination and dental corrections carried out in a standing, sedated equine patient.

At all times the practitioner must be cognizant of patient fright, pain and discomfort that may result in a flight reaction. This can increase the risk of injury to the veterinarian, his assistant and the patient. In some cases, general anesthesia may be required. The less involved type of standing restraint has been described as 'performing dentistry by feel.' This type of dentistry is performed with the horse's head at the level of the operator's waist or chest. This technique often requires minimal sedation and works well for most horses with normal dentition that need only minimal routine corrective procedures. The horse's head can be periodically elevated and the oral cavity visually evaluated during the procedure.[7–10] The second method commonly employed is 'visual dentistry.'[7,11] Working in the horse's mouth visually requires the patient to be well restrained and heavily sedated. The animal's head must be elevated and supported at a height that allows visualization of the mouth while in a comfortable ergonomic body position.[12] Visual dentistry allows for a more thorough dental examination and precise correction of dental abnormalities. Both methods have their place in practice but visual dentistry has many advantages over dentistry by feel, especially in horses with dental pathology or severe abnormalities. Working with dental instruments, including power equipment, requires strength, dexterity and mastery of technique. The visual method allows better access to the mouth and lowers the learning curve on the use of equipment.

Proper equipment, a good working environment and proper patient restraint and positioning are important factors for safe equine dentistry. Instruments that are specifically designed to be used in the horse's mouth are necessary. Any electrical device should be used with a ground fault circuit interpreter plug (GFI). The GFI should be placed between the electrical outlet and the extension cord. If power tools are used in the rostral aspect of the mouth, all persons in the immediate area should wear safety goggles or glasses with side shields to protect the eyes from flying dental debris. If loud motors are in the work area, earplugs or other hearing protection should be worn. Grinding equipment aerosolizes dental dust, and a face mask should be worn with a filter efficiency of 95 per cent. Vacuum devices and/or fans are often used to remove dental dust from the working area. Fluid irrigation is used to reduce thermal damage to teeth and eliminate aerosolized dental dust. Veterinarians and all assistants should be dressed properly. Loose clothing, jewelry and drawstrings can get caught in moving parts.

Some practitioners prefer to perform dental work with the horse in its stall. This requires that equipment be moved around in the barn. The stall should be free of obstacles, have a high ceiling and have good footing. Electricity and water should be located nearby. Good ventilation and some control of the climate makes work more enjoyable for the veterinarian and patient. Working in one designated area of the barn such as a wash rack, farrier's room or stocks simplifies equipment handling and patient restraint. The disadvantage to this situation is that some horses become apprehensive when moved into a strange area. If the horse is to be sedated, it must be awakened in order to move it back to its stall before the next patient is brought into the work area.

Dental corrective procedures such as floating teeth were once considered fairly innocuous. With the development of better quality and more efficient equipment to instrument the mouth and reduce dental crowns, dental correction can be overdone and have severe detrimental effects on the patient. Rasping teeth has been shown to amputate odontoblast processes, leave deep grooves in the surface of the dentin and/or chip the enamel surface and peripheral cement. Modern motorized dental tools remove greater amounts of dental tissue thereby increasing the risk of crown damage. It has recently been speculated that horses may suffer dental pain after corrective procedures.[13] A fine-toothed burr should be used with a light intermittent cutting stroke to reduce dental crowns. Fine-toothed rasps or fine carbide chip burrs cause less damage in reduction. An efficient cooling system and frequently cleaned high-speed burrs may reduce injury to the dentin and pulp.[14,15]

This chapter is divided into five sections: (1) dental prophylaxis, (2) performance dentistry, (3) special concerns in treatment of geriatric patients, miniature horses and draft breeds, (4) correction of cheek teeth and incisor overgrowths and associated pathology, (5) complications of dental corrective procedures. The dental equipment and instruments needed to carry out these topics have been covered previously in this text. Dental surgeries and cheek teeth, incisor and canine extraction procedures will be discussed in separate chapters.

Dental prophylaxis

In veterinary medicine the concept of prophylaxis, that is, the ability to use a practice that will prevent the development of subsequent serious disease, is the foundation of any health maintenance program. Dental prophylaxis, the examination of the oral cavity and the use of corrective procedures to arrest disease processes before clinical signs are seen, has been reaffirmed as an important part of a patient's health care program. Historically, 'floating' was a term that originated in the masonry and/or carpentry professions to describe leveling or smoothing out of plaster. In equine veterinary practice, floating involves the use of files, burrs or chisels to remove the enamel points from the buccal aspects of the upper and lingual edges of the lower cheek teeth. Smoothing makes these areas that contact soft tissue less irritating, thus providing more comfortable mastication and bitting for the horse.[16–18] Floating may be the first dental procedure performed and can make the mouth more comfortable when using a full mouth speculum; or, it may be preceded by cutting, grinding or extracting teeth to provide a dental arcade that can be properly rasped. Hand floating by feel with minimal sedation will be described in detail. However, many practitioners use power tools in routine floating.

Since each type of equipment requires varied techniques, it is recommended that one works closely with practitioners who have experience with the specific tools being used. Manufacturer recommendations on the use of particular tools should be followed very closely.

Equine dental floating should be approached in a sequential fashion, beginning with the upper premolars, working toward the rear of the mouth and covering all surfaces. A full set of floating instruments is needed to reach the various areas of the mouth. The upper buccal aspect of the central four cheek teeth is the easiest point of the arcades to float. The most appropriate tool to reach this area is a straight head float. The practitioner can introduce the upper molar float to the horse by allowing the animal to sniff the float and to observe and feel the float's action on the outside of the cheek *before* inserting the instrument into the mouth. The initial strokes should be light and short, progressing along the length of the dental arcade. As the horse becomes more receptive to the tool, the stroke can be lengthened and more pressure applied to the head of the float. The position of the float head should be at a 45° angle to the buccal cusps (Fig. 16.1). Hand position, which influences float head position, should be adjusted according to feel and sound. The high-pitched rough sound of sharp enamel points being rasped will soften and smooth as floating continues. The 45° angulation of the float head should not be rigidly maintained or two sharp angles could be left on the buccal edge of the tooth. The float should be rotated slightly along the longitudinal plane to slightly round the buccal tooth edges. This procedure should be performed on both upper dental arcades before proceeding to the next area (Fig. 16.2).

The upper caudal molars (110, 111, 210, 211) should be floated next using a long-shafted straight float with an upward tilted or obtuse 10° head (back molar float) (Fig. 16.3). The instrument is placed in the buccal space and eased to the back of the mouth. With short oscillating strokes on the pull, the float head is pressed against the buccal aspect of the last two molars. If rough sharp edges are present, the first few strokes are made by placing the head of the float against the tooth and pulling vigorously (Fig. 16.4).



Figure 16.2. A different technique for floating the left maxillary arcade. The left wrist is laid on the horse's nose with the fingers hooked over the shaft of the float, lifting it up. Wrist or hand placed on the horse's nose helps control head movement.

The last area to be floated on the upper arcade involves the second upper premolars (106 and 206). The instruments of choice to use on these teeth are a short-shafted upper premolar float with a 20° angled head or a 9-inch offset head molar roller float (Alberts Dental, Old Chatham, NY) (Fig. 16.5). This float is worked back into the mouth along the buccal aspect of premolars 2 and 3 (Fig. 16.6). Horses with hooks or performance horses that are bitted require special considerations and this will be addressed later in this chapter.

A 15–17-inch straight float with a 3-inch head or a carbide chip table rasp (Olsen and Silk Abrasives, Salem, MA) can be used to float the lower arcade (Fig. 16.7). This instrument is introduced along the lingual aspect of the lower molar table with a mouth speculum in place (Figs 16.8 and 16.9). Horses have a tendency to raise their heads when the lower arcades are floated. A dental halter used as a



Figure 16.1. Floating the right maxillary arcade, the left hand lifts the shaft of the float up and out. Contact with the cheek brings the float blade into proper position.



Figure 16.3. A straight float frequently misses the caudal aspect of upper molar 3 (111, 211). (Figure used with permission from AAEP, 1996 Proceedings.)



Figure 16.4. A back upper molar float has an obtuse head that angles upward at 10°. It makes good contact with the caudal aspect of upper molar 3 (111, 211). This is an important area to float especially in the performance horse. (Figure used with permission from AAEP, 1996 Proceedings.)



Figure 16.7. Young horses may be floated without using a full mouth speculum. This demonstrates using the left hand as a dental wedge with the hand in the interdental space and over the shaft of the float.



Figure 16.5. To float the upper premolars, a 20° angle head float works well. The left fingers are hooked over the nose band and the shaft of the float is lifted up and outward with the thumb.

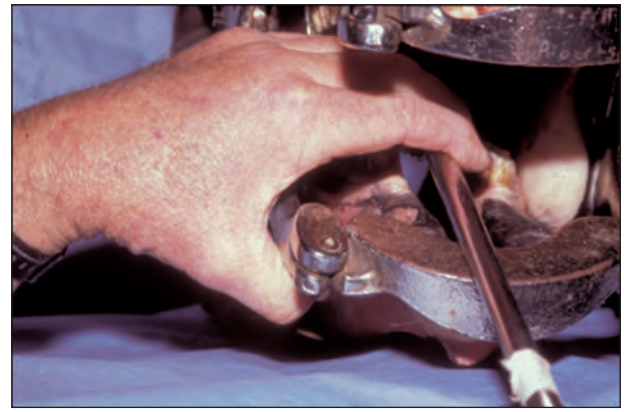


Figure 16.8. When floating the lower arcades with a full mouth speculum in place, the left thumb is worked under the arm of the speculum and the fingers of the left hand placed over the shaft of the float. This technique gives good stabilization to the float and increases the downward pressure that can be applied. (Figure used with permission from AAEP, 1996 Proceedings.)

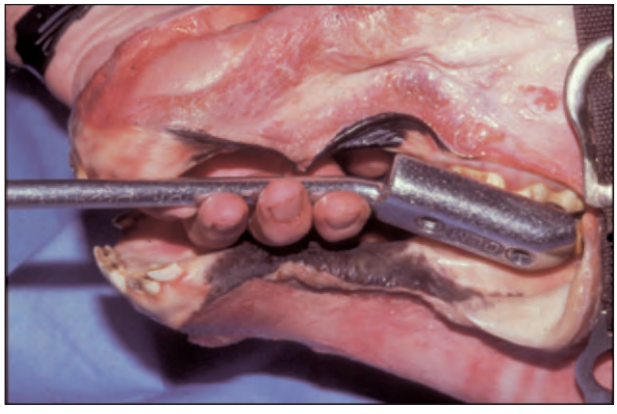


Figure 16.6. Floating the left maxillary arcade, the left hand is across the interdental space with the fingers pushing the shaft of the float up and outward.



Figure 16.9. Lower second premolars (309, 409) are often short in relation to the remainder of the lower arcade. A long straight float has a tendency to pass over these teeth without removing sharp points or edges. A curved or concave molar table rasp can be used to reach these sharp points.



Figure 16.10. Horse restrained for hand floating by 'feel.' The head is held down in a comfortable position with a metal-framed dental halter used as a martingale.

martingale is helpful in holding the head in proper position (Fig. 16.10). Tongue retraction should never be used as a method of restraint when floating. The hyoid apparatus can be fractured with serious complications ensuing. A back molar float can then be used to round the caudal edges of the last lower molar.

With a full mouth speculum in place, the entire upper and lower molar arcades should be digitally and visually inspected. Finally, any areas of asymmetry or overlooked sharp points can be addressed. The speculum is removed and lateral jaw excursion re-evaluated for balance, symmetry and incisor contact. Depending on the horse's age and use, other corrective procedures may need to be considered.

Various types of power dental tools have been manufactured to make the process of dental floating more efficient and less stressful for the horse and the operator. These products have been outlined in Chapter 15. Each type of instrument needs to be handled in the manner intended by the manufacturer. The manufacturer's operator's manual should be consulted for specific details.

Performance horse dentistry

In additional to regular dental prophylaxis, several areas of the dental arcade are of particular interest to veterinarians working with performance horses. Since the horse was first domesticated, reins and bits have been used to send cues from the rider to his mount. Today's performance horses are involved in a wide variety of disciplines. In most endeavors, the equine athlete wears a bridle and bit for control. Oral and dental problems often lead to bad habits and vices such as resisting the bridle, poll sensitivity and head shaking. Over 100 years ago Merillat, in his thesis on horse dentition, summarized the importance of dental care in the performance horse as follows:

> In drivers, runners and saddle horses enamel points are the greatest sources of annoyance. The expert reinsmen will properly recognize their presence by the horse's behavior in harness. Lugging, side reining, ptyalism and tenderness about the seat of the bit are manifestations of pain from the bridle are symptoms of these points. The aim in dressing the teeth of a horse should be to simply blunt the enamel points along the course of the arcades and to "round up" the first superior and inferior molars as smooth as an ivory ball.[19]

Floating the teeth to remove sharp points has been shown in a recent clinical study to have a positive effect on the trainer's perception of the horse's response to the bit. This effect was enhanced by rounding the premolars in what has been referred to as bit seating.[19] Recent studies on the position of the bit in the horse's mouth and surgical correction of bit-induced bar injuries have shed new light on bitting problems.[20,21] Performance horse dentistry entails: (1) normal dental prophylaxis, (2) creating bit seats, (3) care of wolf teeth, (4) deciduous teeth management, and (5) rounding and smoothing canine teeth. Types of bits and contact points on the horse can be reviewed in Chapter 2.

When evaluating performance horses, the veterinarian must keep in mind that subtle points and hooks or a difficult-to-detect loose or painful tooth may cause great personality and performance changes in the elite equine athlete.[22] An important consideration when working on the performance horse's mouth is to remove any sharp or protruding edges from teeth that could make contact with the tender soft tissues of the mouth. A good test for detection of sharp points is to position the fingers just in front of the masseter muscles on both sides of the cheeks at the level of the upper molar arcade. Place firm pressure on the cheeks, pressing them into the teeth and moving the fingers forward. Press the commissures of the lips back against the rostral edges of premolar 2. If the horse flinches or tosses its head, the animal is feeling pain from sharp enamel points.[23]

A regular dental floating usually resolves problems. However, special consideration should be given to balancing the right and left dental arcades. The exposed crown length on both sides of the upper and lower arcades should be equal. If unevenness is discovered, the longer table surfaces should be reduced with a carbide float, table rasp or one of a variety of power instruments.

The rostral edges of the upper and lower second premolars (106, 206, 306, 406) should be rounded to provide a smooth surface against which the cheeks can rest when bit pressure is placed on them. This procedure, termed 'creating

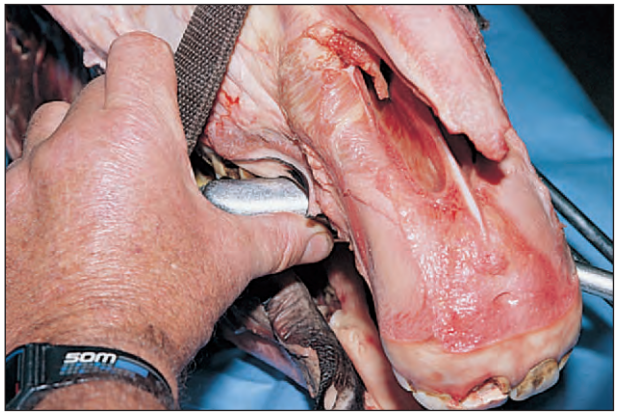


Figure 16.11. In creating a bit seat on 106, the long-handled angled rasp is used to float in a palatal to buccal direction. With the left fingers hooked over the nose band of the halter, the left thumb is used to stabilize and position the float blade against the rostral surface of the tooth.



Figure 16.13. In creating a 206 bit seat, the tooth is floated in a palatal to buccal direction with the left hand in the interdental space, holding the float shaft in position.

bit seats' is performed in an attempt to make the performance horse as comfortable as possible for a reasonable period of time as the bit pulls or pushes soft tissue against the premolar teeth. These teeth should be shaped like the end of an index finger or a marble. There are differences of opinion about the need for creating bit seats, the degree to which the teeth should be beveled and the smoothness required.[17,18,24,25]

To shape the upper teeth (106, 206), several cuts are made. The first is the outside (buccal) cut. The instrument of choice for this cut is a 9-inch float with an offset head. When floating begins, the handle is held on the ipsilateral side of the tooth and is subsequently worked across to the opposite or contralateral side of the face between the open incisors (Fig. 16.11). This rolls the head of the float from the buccal aspect of the second premolar to its rostral buccal and then rostral aspect. This cut is followed by an inside cut to the same tooth with a 9-inch float. The float is positioned at a 30°–45° angle to the lingual surface (Fig. 16.12). Strokes should be made on the lingual edge of the tooth in order to round the rostral aspect. The objectives of the inside and outside cuts are to produce a narrow edge shaped much like a boat hull which can be blunted with a crosscut. The sharp edge is removed using an offset straight head. The float is introduced from the contralateral side, placed at a 45° angle to the point and short vibrating strokes are made (Fig. 16.13). Two fingers may be placed on the outside of the cheeks to serve as guides (similar to how a person holds a pool cue). To observe float placement, the commissure of the lips should be retracted. The last cut made is the fan cut. The float head is placed at a 30°–45° angle to the rostral point of premolar 2, and using a fanning action, short sequential strokes are made (Fig. 16.14). Strokes are initially made on the buccal aspect of premolar 2. A half-circle is rolled or cut starting buccally and is continued around the rostral and lingual surfaces of the tooth. This blends the three prior cuts. The last cut is easier to perform using a full mouth speculum.



Figure 16.12. After the first stage of bit seating, the rostral surface of the tooth is floated in a buccal to palatal direction. The fingers are still hooked over the nose band of the halter but the shaft of the float is held against the tooth by the left thumb.

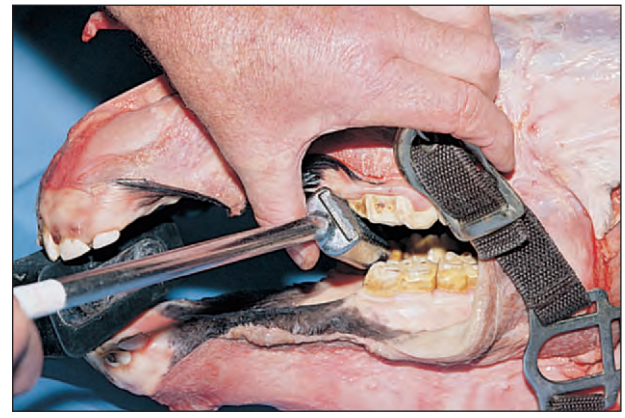


Figure 16.14. After the first stage of 206 bit seating, the fingers of the left hand are hooked over the nose band of the halter while the left thumb stabilizes and pushes the float blade into the tooth, moving the float in a buccal-to-palatal direction.



Figure 16.15. Bit seats can be created with a full mouth speculum in place using a short-blade, offset head, molar roller type float on the upper 106 and 206.



Figure 16.17. Dremel® type instruments with tungsten carbide burrs can be used to create upper and lower bit seats. The cheeks and tongue must be guarded or retracted to prevent soft-tissue trauma.

The float is inserted into the mouth over the speculum incisor plate (Fig. 16.15).

The final step in performance horse floating entails floating the lower first cheek teeth and shaping the lower bit seats. These lower teeth, like the upper second premolars, need to be rounded like a marble and shaped symmetrically relative to one another. The final shape desired depends on the bit being used. The bit seats are formed with the same cuts used on the upper arcade (Fig. 16.16).

The lower cut may be adjusted depending on the bit used. For a snaffle bit, some advocate that the tooth should be rounded to one-third its length, starting at the level of the gingiva on the rostral aspect. For a curb bit, it is thought to be advantageous to start the rounding process halfway up the second premolar, extending it distally. Horses with a loose gingival fold that can be rolled over the occlusal surface benefit from a steeper angle being created on the front cheek teeth. Ideally, the bit should lie on the tongue and not against these teeth. The bit seats and the rest of the arcade should be evaluated for sharpness, symmetry and balance. Sharp points on the caudal molars should be reduced as they can cause nose band pain.

Finally, lateral jaw excursion should be evaluated. The occlusal surfaces of the upper and lower arcades should be reduced to the same distance on the tooth. If one surface is taken out of contact, ramps or hooks tend to form in that area. The tooth crown should not be reduced to a level that would cause excess damage to the sensitive dentin tubules or would expose the pulp chamber. If power tools are used, thermal damage should be controlled by cooling the area with water or by using gentle intermittent tooth contact (Fig. 16.17). As previously described, power tools are best used when performing dentistry by sight (Fig. 16.18). If indicated, the incisor teeth should be addressed appropriately, as will be discussed later in this chapter.

'Wolf tooth' is the common term used to describe the first upper premolar (105, 205).[26] These teeth come in various shapes and sizes. The appearance of the exposed crown is not necessarily a reflection of the size or shape of the root (Fig. 16.19). The number and position of these teeth are



Figure 16.16. Lower bit seats can be formed on 306 and 406 with the full mouth speculum in place. A short-blade, offset head, molar roller type float is inserted beneath the cheek plates of the speculum.



Figure 16.18. Power tools can be used to float the teeth using the visual method of dentistry. The horse must be sedated and restrained with its head suspended in a stand to allow the operator good visualization of the mouth and a comfortable working position.



Figure 16.19. Wolf teeth extracted from several horses. Notice the variation in crown, root size and shape. The lower root fragments were not broken but had resorbed as the permanent 106 or 206 crown had erupted.

quite variable. Forty to eighty per cent of domestic horses erupt at least one upper wolf tooth.[27] Wolf teeth usually erupt at 6–18 months of age but this may be quite variable. In some 2–3 year-old horses, wolf teeth are shed concurrently with the second premolar caps. The larger erupting permanent second premolar tooth often causes root resorption of a wolf tooth that is positioned close to the deciduous second premolar. This probably accounts for the high percentage (80–90 per cent) of horses under 2 years old with wolf teeth and the lower percentage (15–25 per cent) found in adults, even in groups of horses having had no previous dental work. Wolf teeth are usually positioned just rostral to the upper PM2s (Fig. 16.20), but they can be positioned on the buccal side of the first cheek teeth or up to 1 inch rostrally to these teeth (Figs 16.21, 16.22). Double wolf teeth have been seen as well as teeth displaced into the interdental space (Fig. 16.23). Unerupted wolf teeth, referred to as 'blind wolf teeth,' can be detected as firm nodules under the



Figure 16.20. Normal upper wolf teeth in a 6-year-old gelding. Notice the newly erupted canine teeth with sharp crowns positioned rostral in the diastema.

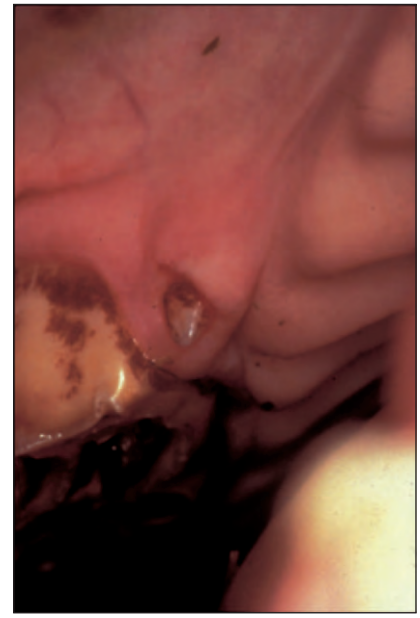


Figure 16.21. A sharp upper right wolf tooth (105) positioned 1 cm rostral to the second upper premolar (106).

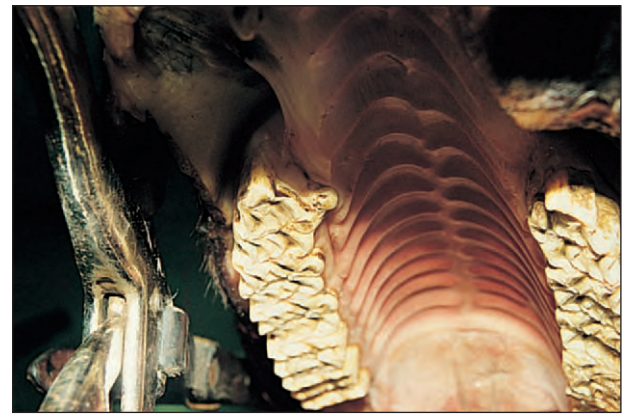


Figure 16.22. Upper wolf teeth (105, 205) erupted palatal to the upper second premolars (106, 206). These palatally displaced wolf teeth can present a problem with extraction because of their close proximity to the palatine artery.



Figure 16.23. Equine skull demonstrating a set of double wolf teeth.

buccal mucosa rostral to the first cheek tooth. These are often painful and at times are covered with ulcerated mucosa.

The role of wolf teeth in causing oral discomfort has been widely debated.[28–30] Tradition and client/trainer pressure are the greatest indications for extraction of these vestigial teeth.[30] Certainly, most wolf teeth cause no problem to the horse but cause concern to the trainer for several reasons. It is difficult, if not impossible, to properly round the rostral edge of PM2 to accommodate bitting with a wolf tooth in place. Displaced or sharp-crowned wolf teeth can cause buccal pain and ulceration when bitting pressure is placed on the cheeks. Some wolf teeth do become loose or diseased and have been shown to be a cause of head shaking or bitting problems.

Some veterinarians advocate floating or grinding the wolf tooth crown, incorporating it into the bit seat. This has potential to loosen the tooth or expose the pulp chamber. Both conditions could be detrimental in the long term and lead to eventual extraction. It is however, customary practice to extract wolf teeth in young performance horses. In most cases, these single-rooted teeth can easily be extracted from the socket in total with proper restraint, equipment and practice. Horses should be sedated and given analgesia or a local anesthetic before these teeth are removed.[29] Usually, only minimal physical restraint is required.

Various dental elevators, gouges and extraction forceps have been developed to loosen and extract wolf teeth. Bone rongeurs and small curettes may be needed to remove root fragments. A complete set of wolf tooth instruments to allow uncomplicated tooth removal would include a Burgess or Musgrave elevator set (Capps Manufacturing, Clatonia, NE), a gouge dental elevator no. 34, a half moon wolf tooth elevator, wolf tooth extraction forceps, fragment forceps (Alberts Dental, Old Chatham, NY), dental extraction forceps no. 62, and a bone rongeur (Mead or Bane) (Fig. 16.24).

A cylinder type punch extractor can be placed over the crown of the tooth to excise the mucosa and allow the root elevator to be properly seated. A curved root elevator should be introduced into the alveolus and gentle pressure placed on the root circumferentially. When the elevator has reached the bottom of the dental socket, pressure on the tooth and downward retraction of the elevator will usually extract the tooth from its socket (Fig. 16.25). If a crown is broken the alveolus should be palpated for loose retained fragments. If remaining slivers are detected they should be removed with a curette or small rongeur. Small root fragments firmly embedded in the alveolus can be left in place with no detrimental effects. Loosened wolf tooth root fragments have been known to emerge at the visible surface of the socket 7–14 days after partial tooth extraction. They can become a source of inflammation and irritation and should be removed. Some wolf teeth in 2- and 3-year-olds have resorbed roots that may give a false impression that the tooth was broken during extraction.

Care should be taken when extracting wolf teeth to avoid having a sharp elevator slip under the palate. The palatine artery can be accidentally lacerated, leading to profuse hemorrhage. If this should occur, pressure applied to the area with a sponge or towel will usually control the hemorrhage. Attempts at ligating the palatine artery can cause more harm as it is buried in a groove in the palatine bone, making the artery difficult to reach.

Blind or unerupted wolf teeth can be evaluated radiographically if one is uncertain about their presence or position. The best way to remove them is to place a Burgess or cone type wolf tooth elevator over the mucosa at the most

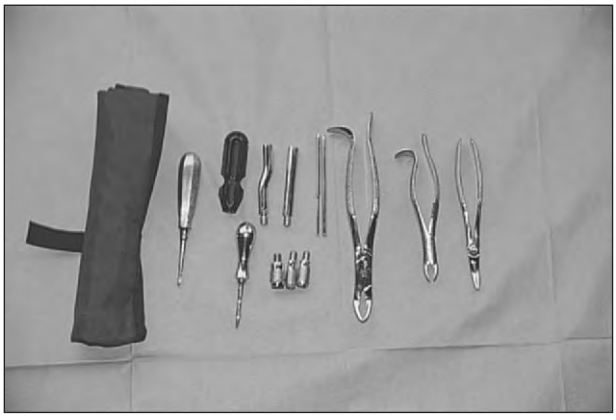


Figure 16.24. A set of wolf tooth instruments including a nylon storage bag, small bone curette, cylinder- or Burgess-type elevator, gouge dental elevator, wolf tooth forceps, dental extraction forceps and fragment forceps.

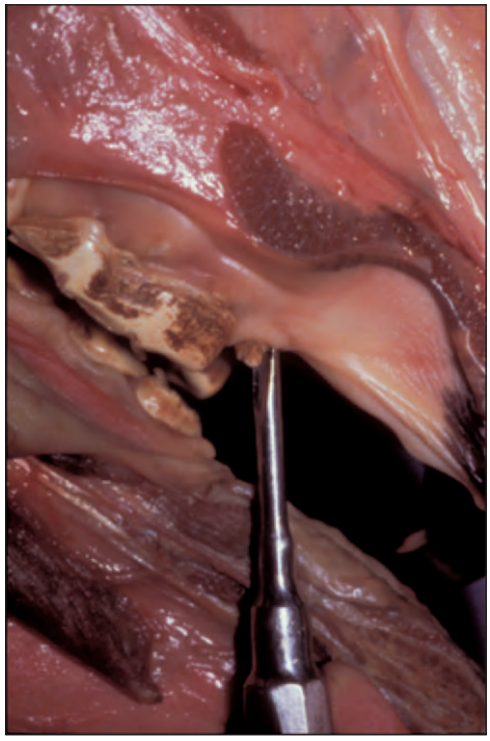


Figure 16.25. A dental root elevator can be used to loosen and elevate most wolf teeth.

rostral aspect of the tooth crown. The cone is pushed through the mucosa over the crown of the tooth and the entire piece of tissue is pried from the bone (Fig. 16.26A–D). These unerupted teeth are usually small and completely covered with cementum.

Rarely, a wolf tooth will be encountered that is quite large and looks as if it has become molarized like the other cheek teeth.[31] These should be evaluated radiographically and, if unopposed, they need to be shortened or extracted. These may prove to be supernumerary teeth in some cases.

Lower first premolars (305–405) are occasionally detected in the mandibles rostral to the first cheek teeth. These are usually quite small and may only be a small tooth sliver detected soon after the deciduous teeth have been shed. However, they can be large with sharp crowns. Lower first premolars have caused problems in bitted horses. Their presence should always be noted during an oral examination on a performance horse. They can be difficult to see on the oral examination because they may be partially covered by a loose fold of buccal mucosa at the lip commissures. Digital palpation just rostral to the first lower cheek tooth is the most accurate way to detect these short-crowned teeth. Unerupted lower wolf teeth are rare and can only be detected radiographically (Fig. 16.27). These teeth can be elevated using the same techniques as for the uppers. The empty alveolus should be packed with gel foam to prevent feed from accumulating in the open dental socket, causing alveolar osteitis (dry socket).

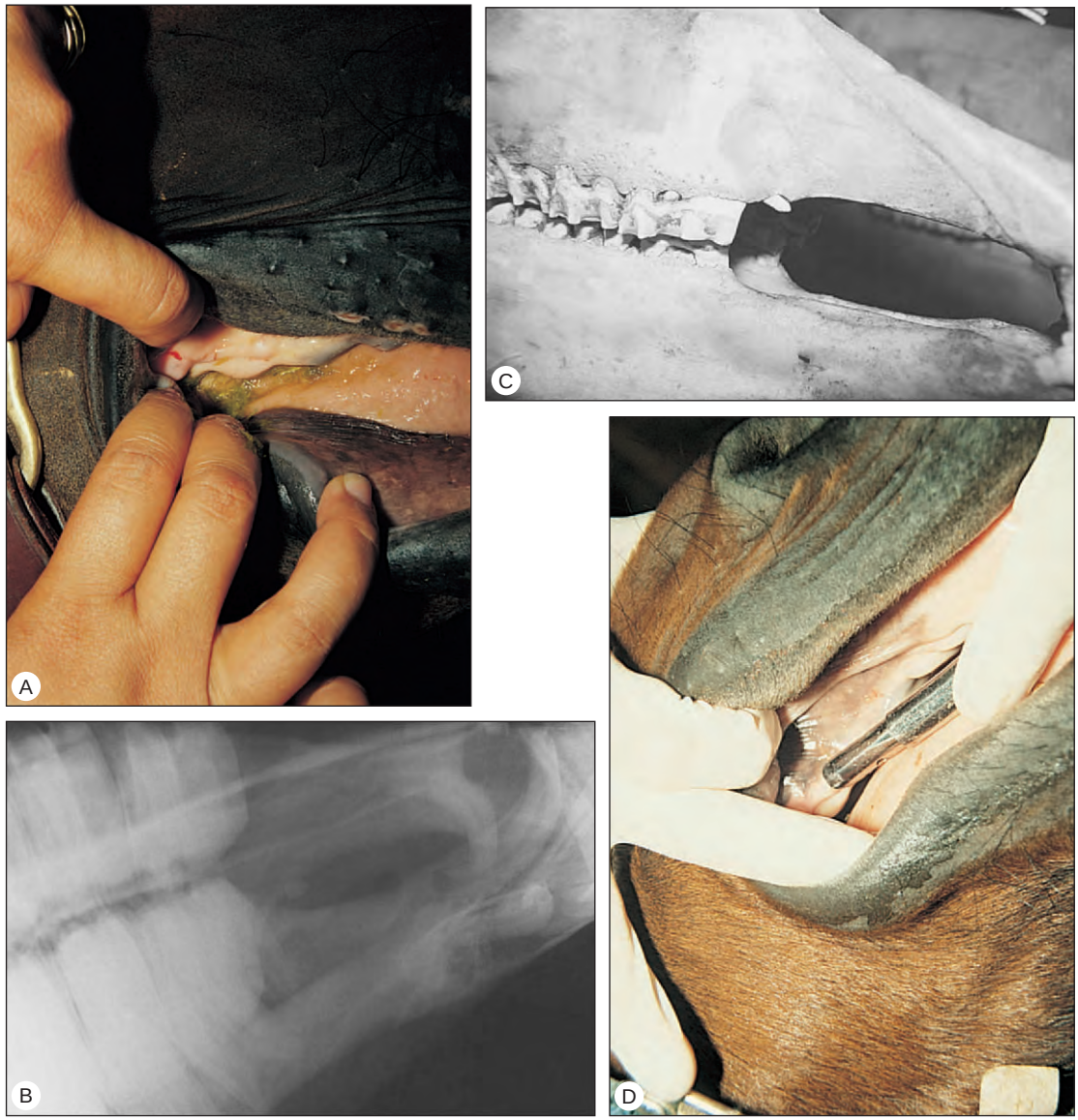


Figure 16.26. (A) An unerupted 'blind' wolf tooth under the oral mucosal rostral to 106. (B) Radiograph of skull demonstrating an unerupted wolf tooth (a radiopaque nodule in the rostral maxilla). (C) A cadaver specimen showing a tilted and rostrally migrated blind wolf tooth. (D) A Burgess type elevator being used to remove a blind wolf tooth.



Figure 16.27. A radiograph demonstrating an unerupted or 'blind' wolf tooth (305). This horse exhibited severe bitting problems that resolved when the tooth was removed.

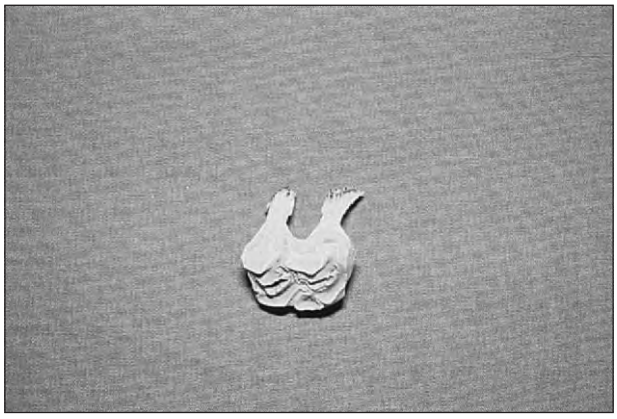


Figure 16.29. Deciduous premolar 3 (507) cap with long buccal root slivers. Broken cap slivers can cause young horses considerable irritation.

In the 2.5- to 4-year-old horse, deciduous premolars and incisors are replaced by permanent teeth (Fig. 16.28). The alveolar walls adjust to this change in dentition through bone production and resorption to provide a new socket for the embedded portion of the developing permanent hypsodont tooth. As the developing permanent teeth push to the surface, they press on the roots of the worn deciduous teeth, gradually cutting off their nutrition. The worn crowns of the deciduous teeth (caps) become loose and eventually die. Subsequently, they are either displaced or are shed into the mouth. These wafer-thin portions of deciduous teeth crowns can have a variable number of root slivers (Fig. 16.29). The caps appear much like a table with four legs laying over the top of the permanent tooth. Gingivitis and periodontal disease can result if these root slivers are broken off and remain in the subgingival space after the cap is shed.

The eruption pattern of permanent molarized dentition follows a sequence that predisposes to entrapment (impaction) of deciduous PM3 and PM4 (Fig. 16.30). Delayed shedding of deciduous premolars can predispose to gingivitis, periodontal irritation or infection (Fig. 16.31A,B). Retained, split or displaced deciduous premolars can be distracting to the training process of a young horse. Additionally, retained deciduous premolars may cause inadequate mastication, anorexia, and malocclusion. In some cases they have been recognized as a factor in dorsal displacement of the soft palate.[32] Radiographs may be necessary to diagnose retained deciduous premolars. If one cap has shed, all other caps in that corresponding quadruplet should be removed. Impacted caps manifested as bony enlargements or eruption bumps on the ventral mandibular ramus or maxilla rostral to the facial crest can cause lingual displacement of permanent teeth or can delay their eruption. Facial swellings are only cosmetic problems in most cases. However, they can become pathological if eruption is severely inhibited or blood-borne bacteria inhabit the inflamed dental pulp. This can lead to anachoretic pulpitis and facial swelling with a draining tract on the mandible or maxilla.[33] Caps should be evaluated by palpation and visual inspection, and in some instances radiographic interpretation may be required. Occasionally, caps may extend above the occlusal surface of the adjacent teeth but cannot be extracted without using excessive force.



Figure 16.28. A cadaver skull from a 2-year-old horse demonstrating 606 and 607 caps. The upper permanent teeth can be seen just erupting out of the dental socket. The opposing 706 cap has been shed.



Figure 16.30. Enlarged mandible on a 5-year-old horse with an impacted premolar 4 (408). This horse developed a periapical abscess that was evident on radiography.

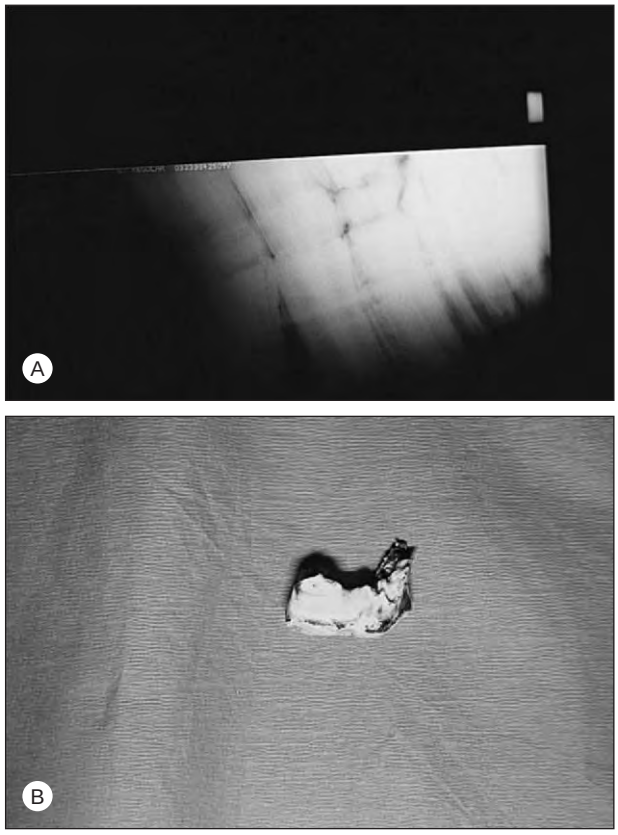


Figure 16.31. (A) Radiograph of an impacted lower premolar 4 (308) trapped beneath a 708 cap. This tooth has become wedged between the first molar and third premolar (309, 307). This horse had a painful enlargement on the affected mandible that resolved 60 days after the cap was removed. (B) Entrapped cap (708) after removal. Notice the flattened crown on the distal occlusal surface where it had been wedged under the first lower molar.

These caps should be floated down level with adjacent occlusal surfaces and evaluated for extraction 6–8 weeks later.

Various forceps, elevators and dental picks are available to aid in the diagnosis and treatment of retained deciduous teeth (Fig. 16.32A,B). These include equine molar forceps (17 inch), Reynolds cap extractors (upper and lower), molar forceps (11 inch), a 7-inch and an 11-inch modified screwdriver, a no. 34 gouge dental elevator and dental extraction forceps no. 69.

To remove the cap of deciduous PM2 and PM3, small wolf tooth extraction forceps work well. On the PM4 cap, open head molar extraction forceps possess a better angle with which to clamp the cap. The forceps are clamped firmly on the base of the cap and pulled lingually across the arcade and the tooth extracted. Care should be taken not to place the forceps below the level of the gums as the palatine vessels along the upper arcade could be disrupted upon clamping, resulting in severe hemorrhage. Rolling the cap toward the lingual surface will reduce breakage of the buccal roots, which can leave slivers of the cap behind. With this method, only the lingual cap roots may break. The residual lingual slivers can be easily removed by the horse with its tongue or by the veterinarian with a root elevator. If slivers do exist on either the lingual or buccal sides of the premolars, they can be worked out of the gum with a dental pick or can be plucked out with a set of closed head rongeurs. When caps are removed, the underlying permanent tooth will erupt and should be in wear in 3–4 months (Fig. 16.33). Sharp enamel edges will be present on these teeth in 3–6 months and the horse should be rechecked and floated at this time.

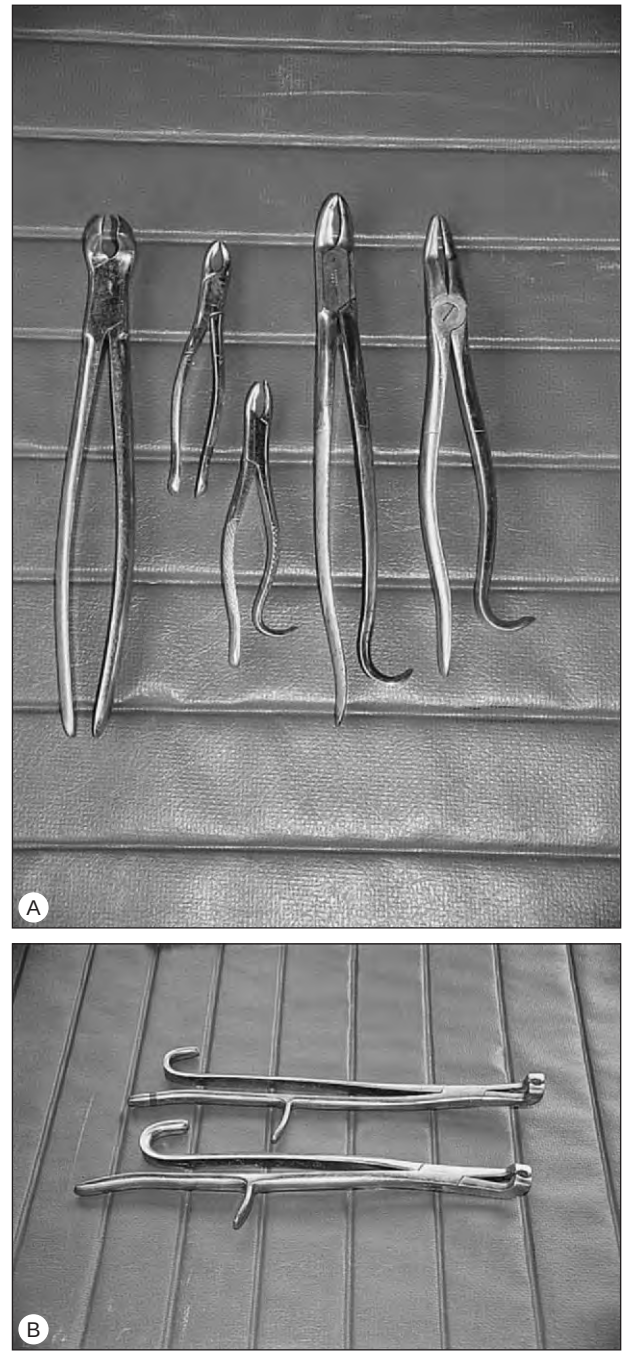


Figure 16.32. (A) Set of deciduous premolar extraction instruments includes a premolar extractor, closed head cap forceps, open head cap forceps, liatard style forceps and wolf tooth extraction forceps. (B) Reynolds premolar cap extraction set.



Figure 16.33. Erupting 106 just after the cap (506) had been shed. Notice the small wolf tooth (105) just rostral to the eruption site. Wolf teeth often become loose and at times avulsed from the socket soon after premolar 2 cap shedding.

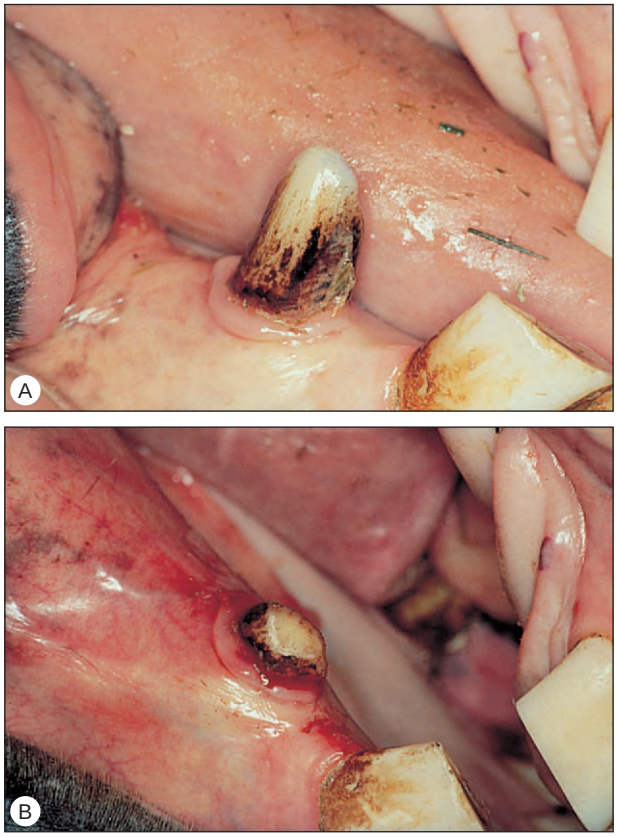


Figure 16.34. (A) Tall sharp canine tooth (404) in a 9-year-old gelding. (B) The same 404 canine tooth after crown reduction and buffing.

Retained deciduous incisor teeth may be another source of discomfort in the 2–5-year-old horse. Head tossing while eating or rubbing the incisors on the stable wall and/or feed box can result from retained incisor caps or root slivers. Incisor caps are easily removed with small extraction forceps. Retained root slivers may need to be elevated with a root elevator while the horse is sedated. Uneven eruption of permanent incisors has been reported as a predisposing factor in incisor malalignment and uneven wear. If the incisor table is not level, it should be filed or burred to make an even surface for upper and lower incisor tooth contact (see Chapter 17).

Canine teeth (104, 204, 304, 404) are usually present in most male horses over 5 years of age. These teeth normally cause few if any problems. In a study of 400 horses, five presented with bitting or head carriage problems related to canine teeth. In four cases with displaced, supernumerary or fractured canines, the teeth were extracted with resolution of clinical signs.[34] It is suggested that displaced or supernumerary canines may be successfully treated by simply reducing or grinding down the clinical crown. Some mares have small rudimentary canines that generally do not cause problems unless they become loose or accumulate tartar. Long or sharp canines in a stallion or gelding may interfere with bitting, the mechanism of which has not been determined.

Tall or sharp canine teeth need to be cut and blunted before performing corrective dental procedures, to reduce the likelihood of injuring one's hands and/or wrists (Fig. 16.34A,B). Another benefit of reducing and smoothing the canines is decreasing the chances of the horse being injured by catching a canine on some fixed object. Reducing the canines to near the gum line may increase the horse's comfort by alleviating constriction of the tongue between tall canines and the bit. In older horses, the crown enamel can become worn and pitted allowing tartar accumulation and gingivitis. By polishing or grinding down the canines, the potential for these problems may be reduced.

Erupting canine teeth in 4–6-year-old horses can cause subgingival pain and bit irritation that has been manifested by head shaking or other bad habits. This problem was reported by Percivall over 100 years ago:

> I was requested to give my opinion concerning a horse, then in his fifth year, who had fed so sparingly for the last fortnight, and so rapidly declined in condition in consequence, that his owner, a veterinary surgeon, was under no light apprehensions about his life. He had himself examined his mouth without having discovered any defect or disease, though another veterinary surgeon was of the opinion that the difficulty or inability manifested in mastication, and the consequent cudding, arose from the preternatural bluntness of the surfaces of the molar teeth, which were, in consequence, filed but without beneficial result. It was after this that I saw the horse, and I confess that I was, at my first examination, quite as much at a loss to offer any satisfactory interpretation as others had been. While meditating, however, after my inspection, on the apparently extraordinary nature of the case, it struck me that I had not seen the tusks. I went back into the stable and discovered two little tumors, red and hard, in the situation of the inferior tusks, which, when pressed,

gave the animal insufferable pain. I instantly took out my pocket knife and made crucial incisions through them both, down to the coming teeth, from which moment the horse recovered his appetite and, by degrees, his wonted condition.[35]

Special concerns in the treatment of geriatric patients, miniature horses and draft breeds

Several types of horses deserve special consideration when performing dental procedures. This section will cover the very old horse (geriatrics), the very small horse (miniatures and ponies) and the very large horse (draft breeds).

Horses are considered old or 'geriatric' after the age of 20.[36–38] Several studies have shown that abnormal wear patterns and periodontal disease are present in 50–90 per cent of geriatric horses.[39–41] Periodontal disease is most often associated with other dental abnormalities such as overgrown, displaced, absent or fractured teeth.[42–44] If a horse lives long enough, by the very nature of hyposodontia, they will eventually suffer from severe dental attrition and disease. Recently, studies have been undertaken to look at disease conditions affecting this age group.[45,46] While many old horses suffer from dental disease, most aged horses seen by veterinarians have abnormal conditions affecting various body systems (Fig. 16.35). In one population of geriatric horses, 54 per cent had gastrointestinal problems, 24 per cent had musculoskeletal afflictions and 16 per cent experienced respiratory tract problems. Body systems affected less commonly in this age group included 11 per cent endocrine, 11 per cent ocular, 7 per cent cardiovascular, 6 per cent integument, 7 per cent reproduction, 4 per cent nervous, 3 per cent urinary and 2 per cent hemolymphatic. Dental disease was recorded in only 8 per cent of the horses in this study group, but all horses did not benefit from a complete oral examination. Wave mouth was the most common dental abnormality diagnosed (50 per cent). This diagnosis was made during a physical examination after referral for another problem such as signs of colic (41 per cent), respiratory tract disease (23 per cent), musculoskeletal disease (10 per cent), esophageal obstruction (8 per cent) and other health problems (10 per cent).[45] This data is presented to emphasize the importance of a complete medical history and physical examination prior to dental correction on all geriatric horses. Appropriate laboratory work-up including hematology, serum chemistry profile, endocrine testing and evaluation of feces for parasite infestation may be indicated. Results of testing should be evaluated and underlying medical conditions addressed at the time of dental correction.

Special consideration must be given to restraining old horses prior to performing any dental procedures. Muscle wasting, degenerative joint disease, laminitis and cardiac disease can complicate sedation and/or restraint. All abnormal wear patterns of the cheek teeth and incisors are exaggerated in old horses. Loss of reserve crown makes teeth more susceptible to loosening or avulsion when stressed with floats or power instruments. Waves, steps and other abnormal wear patterns seen in the geriatric horse may not be due to long or tall teeth but to excessive wear and shortening of the opposite or adjacent teeth in the dental arcade (Fig. 16.36). Many times the tall teeth are actually of normal height (Fig. 16.37). Dental attrition can predispose to deep periodontal pockets that can lead to loosened or fractured teeth (Fig. 16.38). Deep periodontal pockets can also be an initiating factor in osteomyelitis or paranasal sinus disease. Most old horses with sinus disease secondary to dental abnormalities do not have a problem with a dental infection but probably have a deep periodontal pocket that has resulted in the formation of an oral antral fistula. This fistula may allow food access to the sinus cavity or more commonly, the submucosal space just beneath the sinus lining (Fig. 16.39).

Old horses may accumulate tartar on their teeth. The lower canine teeth, lower incisors and buccal aspects of



Figure 16.35. Geriatric horses can have a multitude of physical problems. A thorough medical history and complete physical examination is often warranted before corrective dental procedures are performed.



Figure 16.36. Upper right dental arcade of a 26-year-old quarter-horse. The central cheek teeth (107, 108, 109, 110) are worn smooth. An enlarged area of cementum has formed around the dental roots of these smooth teeth. This mare made an audible squeaking sound when eating.



Figure 16.37. Upper arcade of a 26-year-old Arabian mare. The arcade is irregular with several smooth teeth present. The gums appear healthy with no signs of periodontal disease.

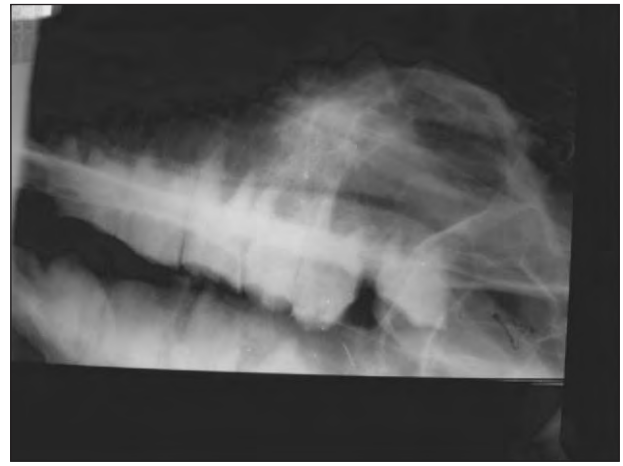


Figure 16.39. Skull radiograph of a 28-year-old Arabian mare with chronic right nasal discharge. A deep periodontal pocket had formed between 110 and 111. An abscess developed just under the sinus mucosa. Reduction of elongated 410 and packing the fistula with dental acrylic after the sinus and abscess were lavaged, controlled the problem for several years.

lower premolars are especially susceptible to gingivitis and periodontal disease secondary to tartar accumulation (Fig. 16.40A,B). This is most often seen in older horses with abnormal mastication due to molar or incisor abnormalities. Tartar should be removed from these teeth and the gingival sulcus thoroughly cleaned. Dental brushing and increasing roughage in the horse's diet along with correcting abnormal incisor and/or molar wear problems often alleviates tartar accumulation.

Dental disease may contribute to a decrease in digestibility of nutrients and result in large roughage fibers entering the esophagus and gastrointestinal tract. This may predispose



Figure 16.38. A common example of irregular dental wear in an old horse. Lower PM2 and PM3 (306, 307) are worn close to the gum and smooth while the upper PM2 (206) has become elongated. A central wave has formed involving 308 and 309 while the upper teeth (208, 209) have been worn to the root. This type of mouth cannot be corrected but regular prophylaxis can help prevent this problem.

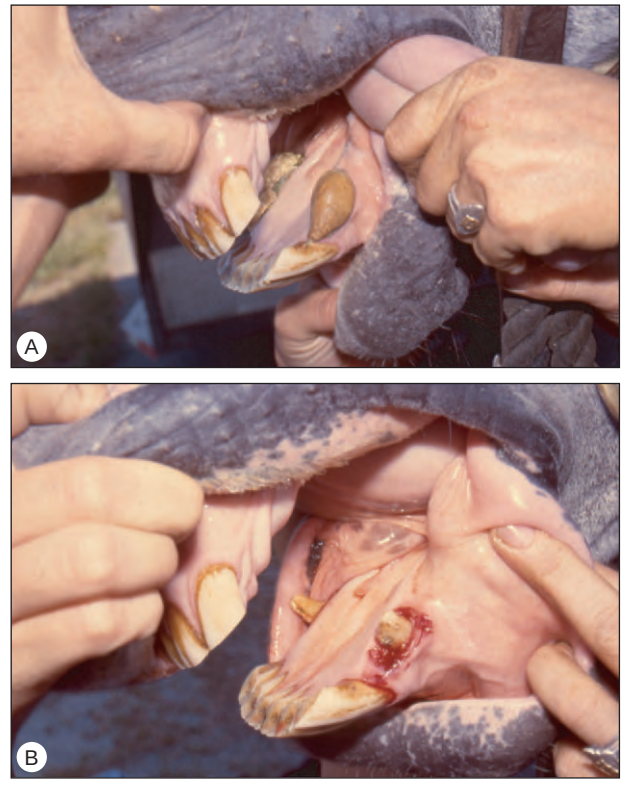


Figure 16.40. (A) Lower canine (304) with a large ball of tartar attached to the crown. (B) Lower canine (304) after tartar scaled from the tooth. Notice the severe gingival recession around the base of the tooth. Frequent dental brushing has helped control the problem.

old horses on rough forage to choke or an intestinal tract obstruction (Fig. 16.41). Nutritional requirements of geriatric horses are complex. Poor dentition severely compromises the ability of old horses to prehend, masticate, digest and absorb nutrients from normal horse diets (grass, hay and oats). Dietary management must be addressed with owners of old horses.[47] Specially formulated senior feeds have been found to improve digestion. One study revealed that old horses in poor body condition fed a specially formulated senior feed, showed significant improvement in weight gain, body condition score and total plasma protein concentration compared with those variables in similar horses fed sweet feed (Fig. 16.42).[48]

In addition to geriatric horses, ponies and miniature horses warrant special consideration. In recent years, small ponies and miniature horses have become popular as companion animals. Intensive inbreeding to reduce body size and refine the head from a large draft type to a light horse type has led to an increased incidence of dental problems (Fig. 16.43). In genetic studies of other animal species, it has been shown that teeth diminish in size more slowly than the jaws.[32] The teeth of a 250 lb miniature horse are close to the same size as those of a 1000 lb quarterhorse.[49] Disproportionately large tooth size in relationship to head size seen in miniature horses can encourage tooth overcrowding and lead to dental maleruptions and malocclusions (Fig. 16.44A–C). This predisposes the small horse to a higher incidence of dental disease and abnormalities of wear. For this reason, early and frequent oral examinations and interceptive dental corrective procedures are more important in smaller animals.

The miniature's small stature and reduced head size makes the oral cavity more difficult to evaluate. Many of these pet equines are poorly trained and require sedation to be restrained. Careful calculations of body weight and sedative medication dosing at a lower mg/kg level are important. Restraint at a level that allows the examiner/operator to be comfortable requires either elevating the horse or lowering the veterinarian. These horses can be sedated and restrained at ground level but this requires the practitioner to work on his knees. Knee pads offer protection and a solid bar or skirt



Figure 16.42. Processed horse feeds can improve digestibility in geriatric horses. At the top is finely chopped grass hay; on the bottom is extruded complete senior horse feed.



Figure 16.41. Wads of quidded grass found in the pasture where a 26-year-old gelding with severe dental attrition grazed.



Figure 16.43. Very large and very small horses can present special problems to the equine practitioner performing dental corrective procedures.

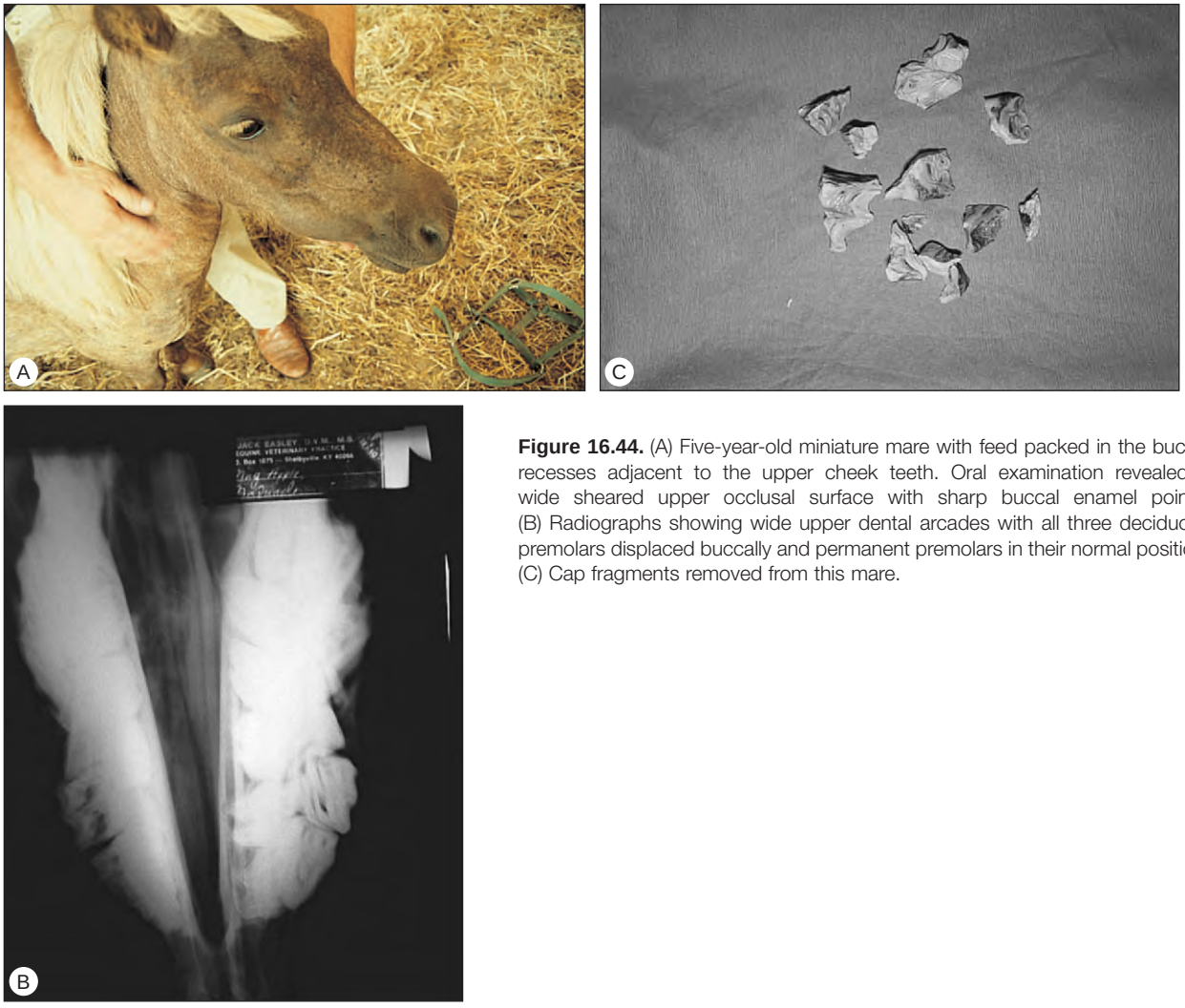


Figure 16.44. (A) Five-year-old miniature mare with feed packed in the buccal recesses adjacent to the upper cheek teeth. Oral examination revealed a wide sheared upper occlusal surface with sharp buccal enamel points. (B) Radiographs showing wide upper dental arcades with all three deciduous premolars displaced buccally and permanent premolars in their normal position. (C) Cap fragments removed from this mare.

in front of the horse will provide a barrier between the operator and the horse. Elevated stocks or loading dock can bring the animal's head to a more comfortable working height.

Special dental instruments may be required to work in the small oral cavity of miniatures/ponies. Most McPherson type speculums work well for these small animals. However, extra holes may need to be punched in the poll strap and small incisor plates may need to be used for a secure fit. Several dental equipment companies manufacture small speculums and dental wedges for miniature horses (Alberts Dental, Old Chatham, NY; Harlton's Equine Specialties, Columbus, OH; Worldwide Equine, Glenns Ferry, ID). Small hand-held floats have been designed with a slimline or low-profile head and short shafts (Alberts Dental, Old Chatham, NY; Harlton's Equine Specialties, Columbus, OH; Worldwide Equine, Glenns Ferry, ID). Power reciprocating slimline tools and small rotary instruments work extremely well in small mouths (Equi-Dental Technologies, Sparks, NV; Jupiter Veterinary Products, Harrisburg, PA; Carbide Products, Torrance, CA; Stubbs Equine Innovations, Johnson City, TX).

Interceptive orthodontics are more often employed in miniature horses because they experience a higher incidence of dental overcrowding (see Chapter 17). It has been found that early removal of deciduous teeth can result in destruction of permanent tooth buds. This information can be used to the horse's advantage if there is severe overcrowding and no room for a full set of permanent teeth in the dental arcade.[49] Malformed permanent teeth and severe early dental decay are other sequelae of premature deciduous tooth removal, most commonly seen affecting the upper premolars (Fig. 16.45).

Finally, draft and large warmblood breeds present special dental considerations due to their weight and size. Large horses weighing over 2000 lb usually require less medication for sedation on a mg/kg basis than medium-sized animals. A head support stand or a metal-framed dental halter suspended from a sturdy beam is necessary to support the additional weight of the heavy horse's head. A sedated draft horse can become unsteady or stumble, which can be extremely dangerous for the veterinarian or his assistant(s).

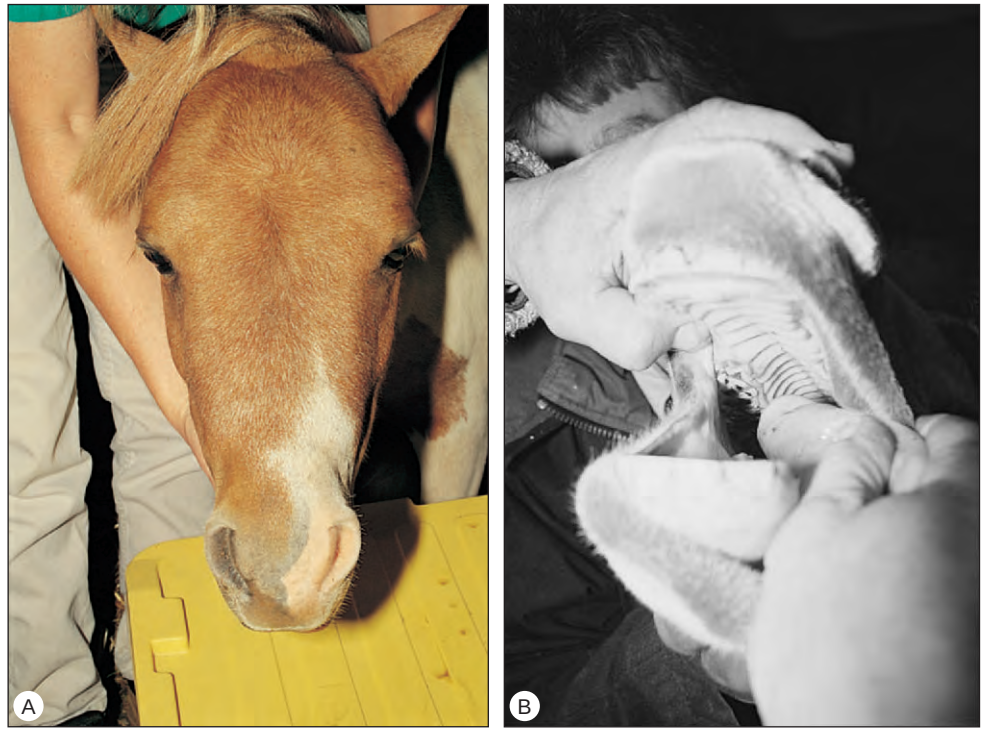


Figure 16.45. (A) Severe facial swelling in a 3-year-old miniature horse. This horse experienced severe respiratory distress when exercised. (B) Oral examination revealed a partially erupted permanent second premolar crown (106) rostral to the deciduous tooth (506).

Sturdy stocks are recommended as a safe area for restraint. The operator may need a platform on which to stand in order to maintain an ergonomic working posture. Large horses may overpower a regular McPherson type speculum and the larger MacAllen or Conrad speculums (Harlton's Equine Specialties, Columbus, OH) will give the operator more security and safety when placing hands and arms into the caudal recesses of the mouth. The Series 2000 speculum (Worldwide Equine, Glenns Ferry, ID) is heavy and can be purchased with a draft horse poll strap. If hand floating, long heavy instruments are required to reach the caudal aspect of the cheek teeth. Special 24-inch-long shafts with bonded or interchangeable blades are available from at least one instrument manufacturer (Alberts Dental, Old Chatham, NY). Power equipment is especially convenient for working in large, deep mouths. The disk burr floats with long shafts have been found to be extremely versatile and efficient for working on draft horses (B & D Equine Enterprises, Calgary, Canada; Worldwide Equine, Glenns Ferry, ID; Eisenhuit-Vet AG, Sandweg, Switzerland).

In large breeds, deciduous tooth shedding may be delayed up to several months compared to the light breeds which will further postpone cap removal. The incidence of certain congenital craniofacial deformities (wry nose, parrot mouth) has been over-represented in draft breed horses.[50] There appears to be a disproportionately large number of draft horses with molar arcade malocclusions leading to hook formation on the upper 06s and lower 11s. Clydesdale horses have a distinct 'mustache' appearance of the upper lip and many have large, even molarized, wolf teeth (105, 205). These large wolf teeth often require removal for correction of bitting problems. Preoperative radiographs and local infiltration of appropriate anesthetic agents have been helpful in planning and successfully carrying out extractions.

Correction of cheek teeth and incisor overgrowths and associated pathology

The process of reducing dental protuberances to adjust the dental arcades has been practiced for centuries. Percussion type instruments described as molar cutters or chisels have been used for at least 100 years. Abnormal wear patterns develop secondary to poor dental occlusion or altered masticatory patterns. It is beneficial to explore the cause of the wear abnormality before corrective action is instituted. It should be determined whether the wear abnormality is an overgrowth of the tooth crown or an attrition or lack of tooth crown that is rendering the table surface uneven. The classic types of cheek teeth abnormalities of wear are described as step mouth (tall teeth), hooks, wave mouth, exaggerated transverse ridges and shear mouth. Common abnormalities of incisor wear include elongated teeth

secondary to overjet, diagonal bite, smile or frown bite and isolated tall teeth.[9,17]

Balancing a horse's mouth entails more than simply reducing the crown on tall teeth. Dental arcade balancing or equilibration allows the jaw to move symmetrically through the full range of mastication. The molar arcades, oral soft tissues, muscles of mastication and temporomandibular articulation should function as a unit. Factors such as head conformation, facial asymmetry, previous trauma, dental attrition and craniofacial deformity (congenital or developmental) determine how close to ideal dental balancing can be achieved. It must be kept in mind that changing the crown shape of a tooth changes the way the tooth functions in the arcade. With even a small alternation of the dental table, all associated structures of mastication (e.g. teeth, bone, muscles, tongue, palate) must adjust. Indiscriminate use of instruments in the mouth by individuals untrained in the principles of dental anatomy, physiology and pathophysiology can cause harm to the dental apparatus. Corrective procedures dealing with the occlusal surfaces of teeth should be conservative until one has a thorough working knowledge of not only anatomy and mastication but tools specifically developed for correction. Principles in treating all dental elongations are the same: Reduce the tall tooth to take the damaged or worn surface out of occlusion and allow for less restricted rostrocaudal and lateral jaw motion.[6]

Hyposodont teeth out of occlusion with teeth in the opposite dental arcade will become tall or protuberant from lack of crown wear or attrition. Congenital or developmental conditions resulting in unopposed teeth include supernumerary teeth or the absence of a tooth or several teeth in a molar or incisor arcade. Acquired conditions with this same result include teeth that have been surgically removed from one arcade or severe crown damage or fracture that has occurred; the unopposed tooth/teeth become affected.[42,51]

Unopposed teeth become longer over time and cause pronounced negative effects on mastication (Fig. 16.46). This condition is often referred to as 'step mouth.' Long crowns can reach the soft tissues of the opposite jaw and lead to ulceration, osteomyelitis or sinus empyema. It is important to detect unopposed teeth early and keep the table surfaces even. This is easy to do with carbide rasps during regular dental check-ups. If the teeth are not attended to on a regular basis, great difficulty may be encountered in attempting to reduce tall teeth with only a rasp. Many power dental instruments available today are quite efficient in reducing tall teeth crowns. These instruments should be used with caution so as not to overheat the tooth or abrade the soft tissues of the mouth. Some tall teeth, especially those in the caudal recesses of the oral cavity, may be difficult to reach with some power tools. Molar cutters and obstetrical wire may be useful in correcting some caudal elongations.

Equipment selection and placement are critical when using molar cutters to reduce tall teeth. Cutters should be sized to fit the tooth to be fractured, with blades set parallel to each other when pressure is placed on the buccal and lingual edges of the crown. The cutter blades should be placed parallel to the normal occlusal surface of the dental arcade. The tooth should be fractured with quick pressure taking care not to twist the cutter. The objective is to cleanly fracture the tooth crown without damaging the remaining tooth. Problems seen after molars have been cut include fissure fractures down the crown and associated periodontal pockets, tooth extractions and pulp exposure. Molar cutters have either simple or compound action. A simple cutter with C- or D-head works well for most mandibular cheek teeth. A compound D-head cutter is more effective in fracturing maxillary cheek teeth (Fig. 16.47). The upper molars are wide and the upper incisor teeth and speculum plates limit access to a long, straight-handled cutter. An open head cutter with an offset 20° angle works well to reach some maxillary cheek teeth. After cutting, the affected tooth should be palpated and probed to ensure the intact section has not been loosened or fractured. The table surface should be smoothed and leveled with a rasp.

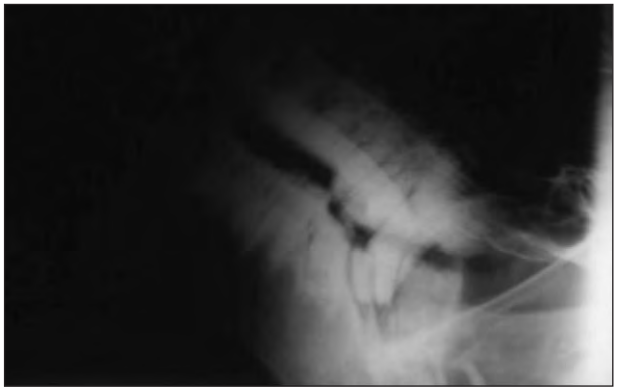


Figure 16.46. Skull radiograph of a thin 18-year-old horse showing an elongated upper last molar opposite a missing lower last molar. This horse experienced severe difficulty masticating any type of dry roughage. The horse's condition improved after the elongated tooth crown was reduced.



Figure 16.47. A set of four equine molar cutters. From left to right: a simple A-head cutter, simple B-head cutter, simple C-head cutter and a compound D-head cutter (Alberts Dental, Old Chatham, NY).



Figure 16.48. Large upper premolar 2 hooks (106, 206) in a 21-year-old thin Arabian gelding. The hook on 206 had formed a callous hole in the left lower mandible just rostral to 306. This horse also had two caudal lower hooks on 311 and 411. These hooks were reduced with a rotary disk dental grinder.

Dental hooks, if present, are usually located on the rostral or caudal aspects of the molar arcades (Fig. 16.48). They are typically the result of a malocclusion of the upper and lower jaws and can be associated with congenital or developmental disorders. Rostral or caudal displacement of the maxillary arcade or a disparity in length of cheek tooth rows will result in a hook. Hooks grow and develop at a variable rate but do so in proportion to the eruption rate of the involved tooth. Most teeth that develop hooks are in partial occlusion, and supereruption is seldom a factor in the rate of hook formation. The length and table surface of premolar and molar hooks increase over time. Hooks alter mastication and place abnormal forces on the teeth and jaws (Fig. 16.49).

It is best to keep hooks from forming by paying close attention to occlusion on a regular basis and correcting protuberant enamel points or beaks when they are small. Not all horses have routine dental care and some will develop large hooks over time. The position, size and extent of the hook should be assessed, as should its mechanical effect on periodontal structures of the affected tooth and opposing teeth. Additionally, the pattern of mastication should be taken into account. Some hooks are bilateral and symmetric to all four molar quadrants. Large hooks can have a detrimental effect on the alignment of the incisor tables from abnormal forces placed on the jaws. Small hooks that consist mostly of enamel can be reduced with a carbide float. Large hooks that consume a greater portion of the table surface contain a high percentage of dentin and are much more difficult to rasp. Hooks can be narrow but quite long as is the case in horses with slight malocclusions. Some hooks comprise almost the entire tooth. This type of hook is more common in horses with missing or extra teeth in a dental row. The hook should be reduced to the level of the normal molar table surface. This reduction may require more work than most veterinarians are willing or able to employ with most hand tools. Percussion instruments, both cutters and chippers, have been used successfully to reduce hooks. These instruments should be used with great caution and precision as teeth have been broken, loosened and/or repelled as a result. The most efficient and safest way to remove hooks is with the use of motorized dental grinders. These instruments use high-speed rotary burrs made of tungsten carbide or diamond grit to grind down the tall crown surface of the tooth.[24,25]



Figure 16.49. Twelve-year-old mare with a broad hook on 206. This hook was opposite a missing lower 406.

Front hooks in the upper or lower arcade are usually reduced without difficulty. The cheeks and lips should be protected from the burr and visualization is aided by a good head light. Air or water should be used to reduce the amount of heat and dental dust generated when burring. Rear hooks are usually associated with a ramp or wave in the back of the mouth. It is helpful to level the arcade rostral to the hook before correction is attempted. The majority of rear hooks can be reduced with a solid carbide blade mounted on a long-handled, straight float. The blade should be set to cut on the pull stroke. The float is pushed to the back of the mouth until it rests on the top of the hook. A pull stroke is used to rasp the crown of the tooth. Small thin caudal hooks can be removed with an Equi-Chip (Alberts Dental, Old Chatham, NY). Several motorized instruments with 18–24-inch-long guarded heads have been successfully used to remove back hooks. Once the hooks are reduced, forces placed on the jaws and the pattern of mastication will change. Incisor occlusion and lateral jaw excursion should be evaluated before and after corrective procedures. A gradual sloping at the end of the arcades is referred to as a ramp. The principles and methods of therapy are the same as for hooks. Special precautions should be taken if molar cutters are used to reduce rear hooks.

'Wave mouth' is the term used to describe an undulating pattern usually involving the central portion of the dental tables. This condition is seen in horses of any age. Waves usually involve elongated lower 08s and 09s with correspondingly worn, cupped-out or decayed upper 08s and 09s (Fig. 16.38). Waves can also form as a result of missing,

misplaced, deviated or rotated teeth in the dental arcade.[42] It is important to assess the cause of a wave in order to develop a plan for dental management. Long teeth are seldom an isolated event in the mouth but affect the pattern of mastication and wear of all other teeth. It is important to note how many teeth are involved in the protuberant area; rarely is only a single tooth overgrown. The usual rate of dental eruption can be increased if the involved tooth is completely out of occlusion with the opposing teeth. Completely unopposed teeth have been seen to erupt at a rate of 0.5–2.0 cm a year, two to four times the normal rate of eruption. The most common wave seen is the slowly progressing condition of aged horses. Infundibular decay or central crown attrition reduces the upper cheek teeth and the wave may become quite tall as the upper cheek teeth wear down to the root and eventually become smooth.

Slight wave formations of the dental table can be corrected with a float or rasp (Fig. 16.50). The horse's mouth must be held open with either a speculum or wedge to gain access to the table surface of the arcade. The use of tungsten carbide blades makes small wave reduction easier, while power floats and/or grinders are often necessary to reduce extremely tall waves. When reducing a wave it is important not to take down the entire molar table but only the portion involved in the elongation. Keep in mind that by reducing the crown height of the involved teeth, this portion of the dental arcade is being taken out of occlusion. Thus, the masticatory forces are increased on the adjacent teeth. Dental waves are easy to manage if the patient is seen on a regular basis and the crown height is maintained at a normal level.

Exaggerated transverse ridges are actually tall wedges of enamel running buccolingually across the occlusal surface of the tooth (Fig. 16.51). These ridges should be reduced to aid in therapy of the defect that occurs in the opposing arcade. A table float or most any power tool can be used to reduce the elevated portion of the ridge. These should not be confused with regular transverse ridges seen in young horses (3–8 years of age).[52] The regular ridges serve a purpose by increasing the surface area of the teeth and are a normal feature in young horses. Normal ridges are not a continuation of the sharp enamel points that form on the buccal cingula of the upper cheek teeth. These ridges can be slightly contoured but no attempt should be made to reduce or flatten the table surface as this can damage the tooth and reduce its longevity. Excessive reduction of the table surface has been known to bring the molar arcades completely out of occlusion. Overzealous reduction of transverse ridges contributes to the unfortunate need for excessive and repeated incisor reductions.

'Shear mouth' occurs when the occlusal table surfaces of the molar arcades are worn at an extremely steep angle. When dental occlusion is symmetric through a full range of jaw motion, the molar tables should wear at an even 10°–15° slope. When masticatory excursion is limited on one or both sides, the teeth wear at an abnormally steep angle (Fig. 16.52). Horses with loose or painful teeth, jaw

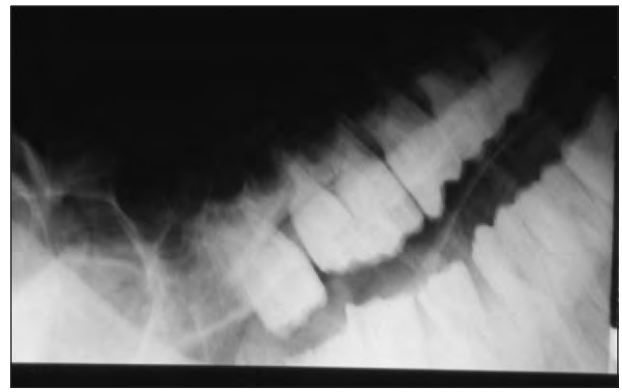


Figure 16.51. Skull radiograph of a 22-year-old horse with exaggerated transverse ridges on the caudal aspect of upper premolar 4 and the caudal aspect of lower molar 2. These ridges have formed opposite diastema and periodontal pockets between lower premolar 4 and molar 1 and upper molar 2 and molar 3.



Figure 16.50. Table rasp or table float used to smooth the junction of the occlusal and lingual surfaces of the maxillary teeth. This same type of carbide chip rasp can be used to reduce transverse ridges.



Figure 16.52. A thin 16-year-old thoroughbred mare with shear mouth. The right dental arcades have worn at a 60° angle and the left at a 45° angle. This mare had no lateral jaw excursion and a slight incisor diagonal bite. Three corrective procedures over a 9-month period improved this mare's condition dramatically.

malalignment or temporomandibular joint problems that limit jaw motion in one direction develop shear mouth on the affected side. Quite often horses with shear mouth will also exhibit masseter and temporalis muscle atrophy on one or both sides.

Before correcting the abnormal wear pattern of shear mouth, the equine practitioner should attempt to identify and correct the underlying cause. Any attempt at correction of the molar table angle abnormality should only be addressed after certain factors are considered: (1) The condition has been present for an extended period of time and the muscles, ligaments and joints have remodeled to accommodate changed chewing patterns. (2) Steep table angles may be accompanied by a long outer buccal edge of the upper arcade (up to 4 cm) and a very short palatal edge that may progress up into the gum line. (A corresponding long sharp edge will usually form on the lower arcade.) (3) The tall, scissor-like conformation of the dental arcades may prevent opening the mouth wide enough to allow visualization or instrumentation in the caudal portion of the mouth.

Correction of shear mouth should be attempted in stages, working on the horse's mouth every 1–3 months for three to six visits. The scissor blade wear pattern on the cheek teeth prevents the operator from establishing a normal table angle even if the tall portion of the crown is reduced to a more normal height. Working from the front of the mouth caudally, the molar tables can be contoured. Over time, the muscles and joints adjust with mastication and many affected horses enjoy more normal occlusion and comfortable masticatory function. This condition is irreversible in some horses and associated dental pathology may be severe. Most of these horses must be managed through dietary adjustments.

Abnormalities of incisors cause difficult mastication and decreased performance. The incisors are easy to observe and can be evaluated with less difficulty than the cheek teeth. Incisor abnormalities have been separated into five classes (Fig. 16.53)[53,54]:

1. excessively long incisor arcades
2. smile bite
3. frown bite
4. diagonal bite
5. stepped or irregular bite.

Most abnormalities can be corrected or at least greatly improved with relatively simple procedures and basic equipment. When realigning the occlusal surfaces of the incisor arcade, it is important to keep in mind the relationship between the incisor and molar arcades and the temporomandibular joints. Before the incisor tables can be properly balanced, the molar tables should be floated and wear abnormalities corrected. Horses are usually more sensitive in the incisor tooth area. Since incisor tooth procedures are the last to be performed, the horse may require sedation or resedation to complete the task of incisor reduction. For minor incisor work a twitch may be used to restrain the horse for a short period of time.

The technique to correct overlying long incisors involves reducing the exposed crown height of the long teeth. A simple logical method for determining how much incisor should be removed has been proposed. Based on this work using trigonometry and measuring lateral jaw excursion and incisor elevation, a fairly accurate estimate of incisor reduction can be determined.[55] Another method often used is estimating the distance in the interocclusal space. This has been defined as the distance between the occlusal surfaces of the upper and lower cheek teeth arcades. To estimate the interocclusal space, the sedated horse's head is elevated and the cheek retracted. Using a penlight or other transillumination device, the distance between the cheek teeth arcades can be estimated.[17]

Long incisor reductions have been performed using Dremel® type grinding tools with solid tungsten, carbide grit or diamond grit burrs (Fig. 16.54). Diamond cut-off wheels, nippers and forceps have been used to remove large amounts of incisor crown, but these tools can prove dangerous to the horse and operator if not used properly and cautiously (Fig. 16.55). Teeth have been fractured or avulsed from the socket in an attempt to score and nip off the crown (Fig. 16.56). Rotary grinders with carbide burrs are the

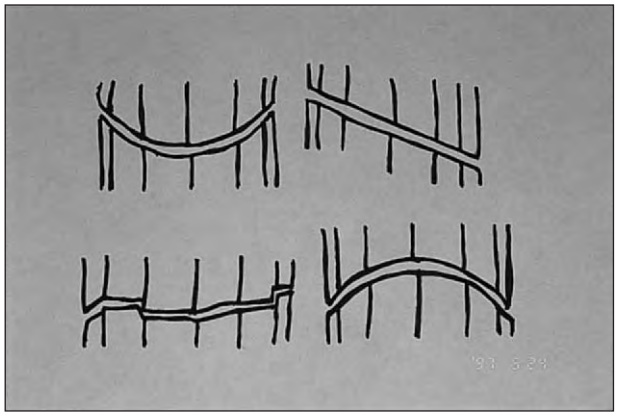


Figure 16.53. Four different incisor abnormalities are shown: grinning, tilted or diagonal, stepped or irregular and frowning.



Figure 16.54. A solid tungsten carbide burr works well for reducing or leveling the incisor arcades.



Figure 16.55. A diamond cut-off wheel is very effective in reducing incisors. Prior to cutting, careful calculation as to the amount of incisor to remove must be made. Care must be taken when using cut-off wheels as they can injure the patient or operator.



Figure 16.57. An example of a titled or diagonal incisor table. This condition occurs when the horse chews only in one direction. This type of abnormality may have to be corrected in stages to keep from bringing the incisor arcades completely out of occlusion.

preferred method for reducing and smoothing incisors. The table surface should be ground down the occlusal surface in thin (1–2 mm) layers and then checked for molar table contact (Fig. 16.57).[16]

Smile bite can be corrected by reducing the corner teeth in the lower arcade (303, 403). Leveling the upper incisors should only be performed if it is determined that reducing them will not create a gap between the upper central (101, 201) and lower central (301, 401) incisors. Frowning incisors are treated in the opposite manner by reducing the corners of the upper arcade (103, 203). Diagonal (or slightly tilted) arcades can be corrected by shortening the upper or lower long or tall incisors (Fig. 16.57). It may be impossible to completely level the more severely tilted incisors without creating a gap between the upper and lower arcades. Incremental shortening of the tall teeth should be performed every 4–6 months until the incisor occlusal surfaces are level from side to side. Stepped (irregular bite) incisors may be locked with the horse unable to move the mandible laterally without opening his mouth, thus reducing cheek tooth occlusion and limiting mastication. The incisors can be leveled from side to side by reducing the tall areas as much as possible with either a fine tungsten carbide float or a carbide burr.



Figure 16.56. A commercially available dental kit with water irrigation. This dental set is ideally suited for reducing premolar 2 hooks, forming bit seats, reducing tall canine teeth and correcting abnormally worn incisor arcades (Equi-Dent Technologies, Inc., Sparks, NV).

The treatment of horses suffering from infundibular decay is controversial. Through the years the belief that cemental hypoplasia and infundibular caries could be diagnosed from an oral evaluation of the tooth has lead some to advocate filling these defects.[56,57] (Refer to Chapter 6 to better understand the etiology and progression of infundibular decay.) To date, the only reasonable management tool is to reduce the tall teeth or wave in the opposite arcade in order to decrease stress on the decayed tooth. With the development of new bonding agents and adhesives, composite restoration has been advocated for fractured crowns, pulp exposure and caries.[58]

Dental overgrowth has been associated with 62.5 per cent of horses with diastema and is attributed to abnormal occlusal movements caused by painful periodontal disease.[59] Becker described treating diastema by enlarging the space between the teeth to reduce food trapping.[60] The type of diastema he dealt with has been recently defined as a 'valve' (or closed) diastema (Fig. 16.51).[44] In this pathological situation, food material is able to enter the triangular defect, bounded rostrally and caudally by tooth, distally by gingiva or the periodontal defect and proximally by the occlusal surface of the dental arcade. Egress of feed material from this space is impeded by the valve effect and the enlarging

abnormal wedge or transverse ridge that often forms on the opposing cheek tooth. Quality regular dental care, appropriate crown reductions and necessary extractions should be the first phase of therapy. Many conditions respond positively with repeated removal of dental associated overgrowths.[59] Removal of foreign material (plant awns, impacted or decayed feed, calculus) in the interproximal spaces and gingival sulci will speed healing in many cases. Flushing dental pockets with a syringe and infusion catheter or an elongated water pick has been described.[16,61] Special long-handled air abrasion units have been developed to deliver water and medical grade baking soda under pressure, to flush periodontal pockets. In cases where reimpaction of feed is likely, placement of a perioceutic within the sulcus and/or dental impression material in the larger interproximal spaces has shown good results.[62,63] Special right-angle burrs have been developed to treat valve diastema. They have been used successfully to grind the dental crown on each side of the valve, opening an occlusal space so the area can self-clean (Fig. 16.39).

Complications of dental corrective procedures

Dental corrective procedures should not be attempted by persons unfamiliar with the possible damage improper equipment and/or technique might cause. Simple tooth floating is not an innocuous procedure but can lead to iatrogenic damage to the horse and its dentition. Coarse float blades chip and break the coronal structures of the tooth and open dentin tubules.[13] Horses chewing on floats or dental spools have been known to fracture or loosen teeth. Floats and other sharp instruments in the mouth can cause soft-tissue damage and can lead to cellulitis and septicemia. Sharp root elevators and dose syringe tips have been known to lacerate the roof of the mouth and cause severe hemorrhage. Power tools must be grounded and have GFI plugs to prevent possible electric shock to the horse and/or operator. Disinfecting agents used on equipment and for flushing the mouth must be prepared in the proper dilution to avoid caustic irritation. Careless or improper use of molar cutters or chippers can lead to tooth fracture or extraction (Fig. 16.58). Overzealous grinding of teeth to reduce elongations or even in forming bits seats and blunting canine crowns can cause complications such as open pulp chambers, tooth decay or tooth loss (Figs 16.59, 16.60). Reducing canine teeth to gum level can lead to tongue lolling in some performance horses (Fig. 16.61A-E). Only a licensed veterinarian should administer an intravenous sedative or analgesic to a horse. Interarterial injections have resulted in severe convulsive reactions or death in some cases. Perivascular medication can cause jugular vein phlebitis or thrombosis, which can end the career of an elite equine athlete. Improperly restrained horses (whether sedated or unsedated) have been known to injure themselves, the operators and other persons

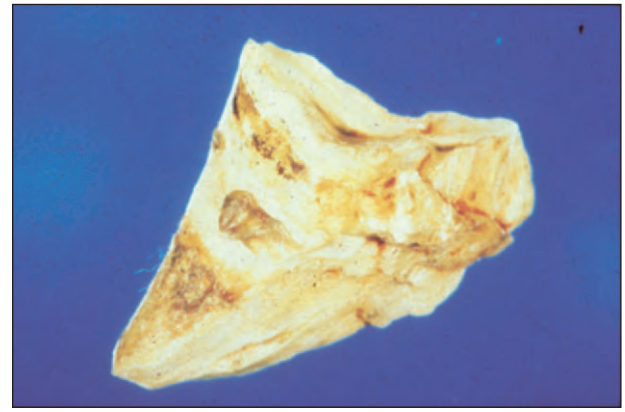


Figure 16.58. This caudal portion of a lower third molar was fractured with a molar cutter while attempting to reduce a caudal lower hook. This improper type of hook reduction may result in an infected lower molar. Surgical removal of the tooth may be required to resolve the infection.



Figure 16.59. Fractured upper incisor tooth (101). A hoof nipper was used to perform incisor reduction. This resulted in a fractured dental crown with pulp exposure. Vital pulpotomy or pulpectomy and crown capping may be required to save the tooth.



Figure 16.60. Lower left canine tooth (304) three years after a crown reduction. The pulp cavity had been inadvertently exposed. The tooth gradually turned dark in color and the exposed pulp chamber blackened. This is a non-viable tooth.

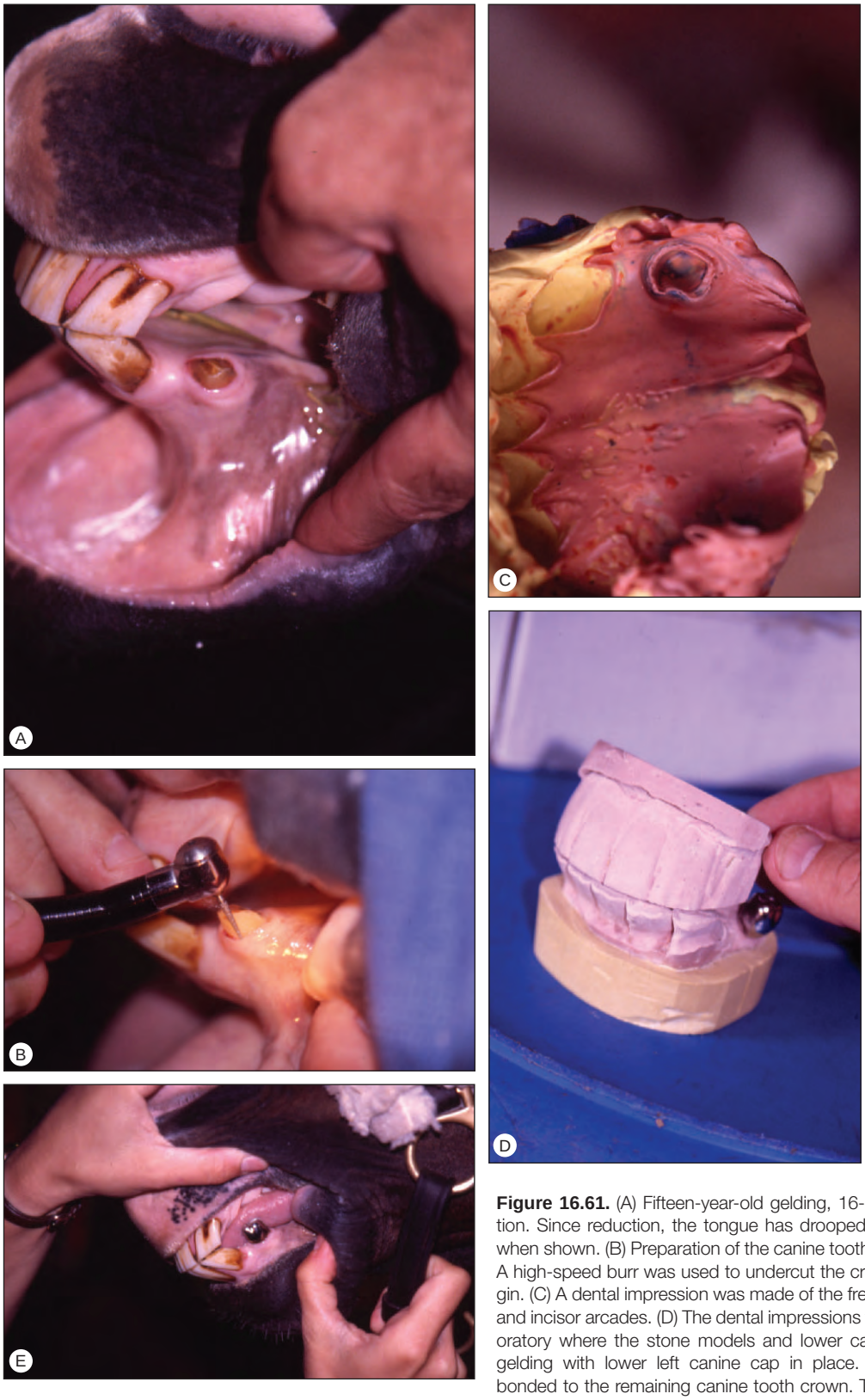


Figure 16.61. (A) Fifteen-year-old gelding, 16-month post canine tooth reduction. Since reduction, the tongue has drooped from the left side of the mouth when shown. (B) Preparation of the canine tooth and gingival sulcus for capping. A high-speed burr was used to undercut the crown just below the gingival margin. (C) A dental impression was made of the freshly prepared lower canine teeth and incisor arcades. (D) The dental impressions were sent to a human dental laboratory where the stone models and lower caps were fabricated. (E) Arabian gelding with lower left canine cap in place. These caps were permanently bonded to the remaining canine tooth crown. This horse won a national championship equitation class the following year and has since been retired with the caps still in place.

in the work area. Iatrogenic jaw fracture has been seen following the use of a full mouth speculum.

Post-dental-procedure pain is experienced by some horses, especially if aggressive procedures are performed with power equipment. Affected horses do not eat well for a few days to a few weeks after dentistry. Some veterinarians advocate giving prophylactic non-steroidal anti-inflammatory medication to all dental patients to help prevent this problem.[64] This painful condition has been blamed on temporomandibular joint pain, readjustment of the masticatory muscles after speculum use, loose teeth post procedure, leaving the mouth not in balance, thus overloading isolated teeth in occlusion, or exposed dental tubule pain.[13] These problems are enumerated to emphasize the importance that only veterinarians and veterinary technicians (under direct supervision of a veterinarian) should perform equine dental procedures. The potential for iatrogenic damage must be kept foremost, as the equine practitioner's job as steward of the horse must be 'to do no harm.'

Timetable for routine dental examination

The following timetable was prepared by Easley.[65]

Birth. Examine for (a) congential defects of the lips or palate, (b) tongue motion and strength, (c) dental malocclusions and (d) body system abnormalities. Recommended procedures are to provide genetic and orthodontic consultation and perform corrective surgery if necessary. Look for other problem signs such as underdeveloped carpal or tarsal bones, ruptured extensor tendons and hernias.

6–8 months. Examine for (a) incisor and premolar occlusion (all incisors should have erupted), (b) missing teeth, (c) sharp enamel points or hooks and (d) ulcers on the tongue and buccal mucosa. Recommended procedures are to provide orthodontic consultation and float teeth if necessary.

16–24 months. Examine for (a) upper and lower wolf teeth eruption, (b) points and hooks on premolars and (c) bit lesions. Recommended procedures are to float teeth and round off the rostral corners of the second premolars. Extract wolf teeth.

2–3 years. Examine (a) upper and lower wolf teeth or blind wolf teeth, (b) deciduous tooth eruption – central incisors and premolars, (c) bit injuries at the corners of the mouth and interdental spaces, and (d) points or hooks on molars and premolars. Recommended procedures are to float outside of upper and inside of lower cheek teeth, remove caps if present and ready for removal, and extract wolf teeth. Rostral corners of upper and lower second premolars should be rounded.

3–4 years. Examine (a) corners of the mouth and interdental space for bit injuries, (b) incisors for retained deciduous teeth or supernumerary teeth, (c) molars and premolars for points and retained third premolars (second cheek teeth), (d) size and shape of the lower jaw, and (e) presence of blind wolf teeth. Recommended procedures are to remove caps if present, float teeth and remove wolf teeth.

4–5 years. Examine (a) incisors for eruption, (b) canine teeth for sharp edges or eruption delays, (c) molar arcade for proper eruption and alignment of fourth premolars, (d) presence of upper rostral and lower caudal cheek teeth hooks from malocclusion, and (e) points or sharp edges on cheek teeth. Recommended procedures are to remove deciduous teeth if ready, grind or rasp hooks if present, float teeth and remove mucosa over canines if gingival eruption cysts are present.

5 years and older. Examine (a) mouth visually and digitally, especially noting hooks and uneven wear, (b) canines for sharp edges and tartar, (c) for oral decay or gingivitis, (d) incisors for even wear and (e) evaluate lateral jaw excursion. Recommended procedures are to float teeth, remove hooks, correct abnormal wear patterns and level or shorten the incisors if indicated.

Educate owners and trainers of the need for routine dental examinations. Indicated corrective procedures should be performed before starting any horse in training.

Summary

A thorough visual and manual examination of the patient must be performed to identify any abnormalities. Sedating the patient and the use of a full mouth speculum facilitates both the examination and corrective procedures. The use of proper dental instruments makes it much easier for both the patient and veterinarian. A dental form can be used to maintain a record of what procedures were done and what will have to be done in the future, and to itemize the charges.

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REFERENCES

1. Ralston SL, Foster DL, Devers T *et al.* (2001) Effect of dental correction on feed digestibility in horses. *Equine Veterinary Journal,* 33, 390–393.
2. Gatta D, Krusic L, Casini L *et al.* (1995) Influence of correcting teeth on digestibility of two types of diets in pregnant mares. In: *Proceedings, 14th Symposium Equine Nutrition and Physiology Society*, pp. 326–331.
3. Krusic L, Easley J and Pagan JD (1995) Influence of correcting teeth on daily food consumption and glucose availability in horses. In: *Proceedings, Symposium Horse Diseases*, pp. 53–68.

4. Wileweki KA and Ruben L (1999) Bit seats: a dental procedure for enhancing performance on show horses. *Equine Practice*, 21, 16.
5. Bonen SJ (2001) Three-dimensional kinematics of equine temporomandibular joint. Masters Thesis, Dept of Mechanical Engineering, Michigan State University, East Lansing, MI.
6. Carmalt JL, Townsend HGG and Allen AL (2003) Effect of dental floating on the rostrocaudal mobility of the mandible of horses. *Journal of the American Veterinary Medical Association,* 5, 666–669.
7. Allen T, Jeffery D and Moriarity LA (2003) Routine procedures. In: *Manual of Equine Dentistry*, ed. T Allen, Mosby, St Louis, pp. 109–156.
8. Wharton I (2000) Equine dentistry working on the un-sedated horse. Academy of Equine Dentistry, Idaho.
9. Easley J (1998) Dental corrective procedures. *Veterinary Clinics of North America: Equine Practice*, 14(2), 411–432.
10. Rucker BA (2003) My approach to motorized equine dentistry using the Powerfloat. In: *Operator's Manual, Powerfloat*, D & B Equine Enterprises, Calgary, Canada.
11. Rach D (2003) *Operator's Manual*, D & B Equine Enterprises, Calgary, Canada.
12. DeForge (2002) Physical ergonomics in veterinary dentistry. *Journal of Veterinary Dentistry,* 19, 196–200.
13. Kempson SA, Davidson MEB and Dacre IT (2003) The effect of three types of rasps on the occlusal surface of equine cheek teeth: a scanning electron microscopic study. *Journal of Veterinary Dentistry,* 20(1), 19–27.
14. Baker GJ and Allen ML (2002) The use of power equipment in equine dentistry. *Proceedings, American Association of Equine Practitioners,* 48, 438–441.
15. Dacre KJP, Dacre IT and Dixon PM (2002) Motorized equine dental equipment. *Equine Veterinary Education,* 14, 263–266.
16. Judd RC (2002) Equine dental prophylaxis using a pneumatic system. *Compendium on Continuing Education for the Practicing Veterinarian,* 24(1), 62–70.
17. Scrutchfield WL (1999) Dental prophylaxis. In: *Equine Dentistry*, ed GJ Baker and J Easley, WB Saunders, London, pp. 185–205.
18. La Flure S (2000) Whole body restoration through whole mouth dental equilibration. Academy of Equine Dentistry, Idaho.
19. Merillat LA (1905) Animal dentistry and diseases of the mouth. In: *Veterinary Surgery*, Vol. 1, Alxander Eger, Chicago, p. 189.
20. Engelke E and Gasse H (2003) An anatomical study of the rostral part of the equine oral cavity in respect to position and size of a snaffle bit. *Equine Veterinary Education,* 5(3), 200–205.
21. Johnson TJ (2002) Surgical removal of mandibular periostitis (bone spurs) caused by bit damage. In: *Proceedings, American Association of Equine Practitioners*, 48, 463–466.
22. Easley J (2003) Oral and dental disease. In: *Equine Sports Medicine,* ed. Kaneps, in press.
23. Fisher D and Easley KJ (1994) Floating: making equine dentistry a practice profit center. *Large Animal Veterinarian,* 49, 16–22.
24. Dixon PM (2000) Removal of equine dental overgrowths. *Equine Veterinary Education,* 12(2), 68–81.
25. Pence P and Wileuska K (2002) Mature horse dentistry. In: *Equine Dentistry – A Practical Guide*, ed. P Pence, Lippincott, Williams and Wilkins, p. 141.
26. Sesson S (1921) Digestive system of the horse. In: *The Anatomy of Domestic Animals* (2nd edn), WB Saunders, Philadelphia, p. 395.
27. Nickel R, Schummer A and Seiferle E (1979) *The Viscera of Domestic Animals* (2nd edn), Paul Parey, Berlin.
28. Lane JG (1994) A review of dental disorders of the horse, their treatment and possible fresh approaches to management. *Equine Veterinary Education,* 6, 13–21.
29. Grove T (1991) Extractions and simple oral surgeries in equines. In: *Proceedings of Eastern States Veterinary Conference,* Vol. 5, p. 337.
30. Gaughn EM (1998) Dental surgery in horses. *Veterinary Clinics of North America: Equine Practice*, 14(2), 387–388.
31. Colyer JF (1990) *Variations and Diseases of the Teeth of Animals* (rev. edn), Cambridge University Press, Cambridge, pp. 121–122.
32. Marr T, Love S, Schumacher J and Walson E (1998) *Equine Medicine, Surgery and Reproduction*. WB Saunders, Philadelphia, p. 106.
33. Easley J (1991) Recognition and management of the diseased equine tooth. *Proceedings, American Association of Equine Practitioners*, 37, 129–139.
34. Dixon PM, Tremaine WH, Pickles K *et al.* (1999) Equine dental disease part 1: A long term study of 400 cases: disorders of incisor, canine and first premolar teeth. *Equine Veterinary Journal,* 31(5), 369–377.
35. Percivall W (1852) Hippopathology. In: *Special Report on Diseases of the Horse*, ed. CB Mechener, US Dept of Agriculture, Washington, 1911, pp. 42–43.
36. Holland R (2000) *The Geriatric Horse*. Blood Horse Publications, Lexington, KY.
37. Lowder MQ and Mueller POE (1998) Dental disease in geriatric horses. *Veterinary Clinics of North America: Equine Practice*, 14(2), 365–381.
38. Pugh DG (2000) Care of the geriatric horse. In: *Proceedings, North American Veterinary Conference*, Vol. 14, p. 178.
39. Baker GJ (1979) A study of dental diseases in the horse. PhD Thesis, University of Glasgow, Scotland.
40. Wafa NS (1988) A study of dental disease in the horse. MVM Thesis, National University of Ireland, Dublin, Ireland, pp. 77–174.
41. Hofmeyr CBF (1960) Comparative dental pathology with particular reference to caries and periodontal disease in the horse and dog. *Journal of the South African Veterinary Medical Association*, 31, 471–480.
42. Dixon PM, Tremaine WH, Pickles K *et al.* (2000) Equine dental disease part 3: a long term study of 400 cases: disorders of wear, traumatic damage and idiopathic fractures, tumors and miscellaneous disorders of the cheek teeth. *Equine Veterinary Journal,* 32(1), 9–18.
43. Greene S and Basile T (2002) Recognition and treatment of equine periodontal disease. *Proceedings, American Association of Equine Practitioners*, 48, 463–466.
44. Carmalt JL (2003) Understanding the equine diastema. *Equine Veterinary Education,* 3(1), 43.
45. Brosnahan MM and Pardis MR (2003) Demographic and clinical characteristics of geriatric horses, 467 cases (1989–1999). *Journal of the American Veterinary Medical Association,* 223(1), 93–98.
46. Brosnahan MM and Pardis MR (2003) Assessment of clinical characteristics, management practices and activities of geriatric horses. *Journal of the American Veterinary Medical Association,* 223(1), 99–103.
47. Pugh DG (2002) Feeding the geriatric horse. *Proceedings, American Association of Equine Practitioners,* 48, 21–23.
48. Ralston SL and Breuer LH (1996) Eield evaluation of a feed formulated for geriatric horses. *Journal of Equine Veterinary Science*, 16, 334–338.
49. Mitz C and Allen T (2003) Dentistry in miniature horses. In: *Manual of Equine Dentistry*, ed. T Allen, Mosby, St Louis, pp. 175–192.
50. Vandersplassche M, Simeons P, Bouters R *et al.* (1984) Etiology and pathogenesis of congenital torticollis and head scoliosis in the equine fetus. *Equine Veterinary Journal,* 16, 419.
51. Dixon PM, Tremaine WH, Pickles K *et al.* (2000) Equine dental disease part 4: a long-term study of 400 cases: apical infections of cheek teeth. *Equine Veterinary Journal,* 32(3), 182–194.
52. Michael J (2001) ATR theory and analysis. Academy of Equine Dentistry, Idaho.
53. Scrutchfield WL (1991) Incisors and canines. *Proceedings, American Association of Equine Practitioners*, 37, 117–121.

54. Rucker BA (1995) Modified procedure for incisor reduction. *Proceedings, American Association of Equine Practitioners*, 41, 42–44.
55. Rucker BA (2002) Utilizing cheek teeth angle of occlusion to determine length of incisor shortening. *Proceedings, American Association of Equine Practitioners*, 48, 448–452.
56. Swanstrom OG and Wallford HA (1977) Prosthetic filling of a cement defect in premolar tooth necrosis in a horse. *Veterinary Medicine/Small Animal Clinician*, 1475–1477.
57. Klugh DO (2002) Updating infundibular decay in equine cheek teeth. In: *Conference Proceedings 16th Veterinary Dental Forum*, Savannah, GA, pp. 238–239.
58. Greene S (2001) Equine dental advances. *Veterinary Clinics of North America: Equine Practice*, 17(2), 319–334.
59. Dixon PM (1999) Equine dental disease part 2: a long term study of 400 cases: disorders of development and eruption and variations in position of the check teeth, *Equine Veterinary Journal*, 31(6), 519–528.
60. Becker E (1945) Cited in Becker (1962) Zähne. In: *Handbook of Specialized Pathological Anatomy of the Horse*, Paul Parey, Berlin, p. 146.
61. Mueller POE and Lowder MQ (1998) Dental sepsis. *Veterinary Clinics of North America: Equine Practice*, 14(2), 349–363.
62. Pence P and Basile T (2002) Dental infections. In: *Equine Dentistry: A Practical Guide*, ed. P Pence, Lippincott, Williams and Wilkins, Philadelphia, pp. 215–219.
63. Greene S and Basile T (2002) Recognition and treatment of equine periodontal disease. *Proceedings, American Association of Equine Practitioners*, 48, pp. 463–466.
64. Greene S (2003) Equine dentistry, notes for Advanced Dentistry Laboratory, American Association of Equine Practitioners Annual Meeting, New Orleans, LA.
65. Easley KJ (1996) Equine dental development and anatomy. *Proceedings, American Association of Equine Practitioners*, 42, 1–10.

17 Basic Equine Orthodontics

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Introduction

The prevention and treatment of dental malocclusions is the field of dentistry known as orthodontics. Orthodontics in its most basic form is the controlled movement of teeth through alveolar bone. The purpose of equine orthodontics is to correct or prevent irregularities and malocclusions of the teeth through surgery, dental crown equilibration and/or the application of appliances. Through these measures, the oral function and periodontal health of the horse is preserved.

This chapter is written with the purpose of introducing the equine practitioner to the principles of orthodontics. The subject will be reviewed in its most basic form with clinical examples where orthodontics have been used to improve the dental health of the horse. This area of equine dentistry is truly in its infancy. Hopefully with this chapter, seeds will be planted in the minds of equine practitioners that over the next century, grow into fruit than can be harvested for the benefit of their equine patients.

Orthodontic principles

The general laws of biomechanics apply to all types of tooth movement. The alveolar bone is reabsorbed whenever the root and reserve crown causes compression of the periodontal ligaments for a certain period of time. New alveolar bone is deposited whenever there are stretching forces acting on the bone. However, these laws are subject to numerous variations and exceptions when factors such as magnitude, direction and duration of force are introduced.[1] Dental cementum has the inherent tendency to resorb from pressure but to a lesser degree than bone.

The orthodontic principles of tooth movement are at work on horses that develop abnormal wear patterns on exposed dental crowns. These abnormal wear patterns can place stresses on the dental crown, resulting in movement of the teeth. Such movement affects mastication and oral health. Dental floating and occlusal adjustment are forms of orthodontic correction used or applied to reduce abnormal forces placed on teeth and thus improve occlusion. The application of orthodontic wire, springs, coils, arch bars, bands, brackets and elastics have limited application in correction of common equine malocclusion problems.

The extraction of deciduous teeth in an effort to guide the eruption of the permanent teeth into a favorable occlusion has been referred to as preventative or interceptive orthodontics.[2] It is necessary to have a thorough understanding of the growth and development of the dental and osseous structures to time deciduous tooth removal and avoid complications in attempting to guide teeth into proper occlusion. A lack of understanding and knowledge can create disastrous results, including deterioration of dentition and facial balance. Interceptive orthodontics, when wisely and judiciously applied to well-selected cases, can enhance dental occlusion as well as prevent dental malocclusions that will cause functional problems with mastication and dental wear throughout the horse's life (Fig. 17.1).

Many dental malocclusions involve an abnormal skeletal relationship of the upper and lower jaws. General form and capacity for growth of bone are inherited characteristics. A basic understanding of the growth of the upper and lower portions of the head is important in diagnosis and treatment of many types of malocclusions. The mandible can be divided into a number of anatomical elements: a basic element, two subsidiary elements for the attachment of muscles, and an alveolar process or tooth-bearing element.[3] Growth of the mandible occurs in two ways: appositional growth from endochondral ossification, which occurs in all borders of the mandible, except the cranial border of the ramus, and epiphyseal-like growth of the condyles. The upper jaw is an integral part of the cranium in contact with other bones of the skull and its growth depends mainly on endochondral ossification.

Bone is plastic and its external form and capacity for growth are affected and modified by environmental forces and factors.[4] A branch of orthodontic treatment first referred to as biomechanical orthodontics has developed over the past century. Using the theories of bone plasticity traced back to Fouz and Wolff, several techniques have been employed to correct dentofacial deformities and malocclusions in the horse utilizing the principles of functional orthodontics. A more descriptive term, functional jaw orthopedics, was popularized by Karl Haupl, who refined the concepts and techniques used in this branch of human dentistry today.[5] Pressure, whether functional or artificially created, affects bone growth. Bone cell growth is constantly taking place from an increase in size and change in form in the



Figure 17.1. Crowded lower incisor arch with retained deciduous 701, 801 causing displacement (labiocclusion) of permanent 301, 401. Following the principles of interceptive orthodontics, the deciduous teeth were removed and the exposed mesial crown portion of 702 and 802 were filed, widening the space for 301 and 401 to migrate forward.



Figure 17.2. Removable functional orthodontic device in the mouth of a 5-month-old foal with a parrot mouth deformity. This device is used to improve the dental alignment and encourage lower jaw growth. The device must be worn 16 hours a day and requires intensive foal nutritional management and husbandry for a successful outcome.

young horse to the replacement of dead cells in the adult. Bone metabolism remains constant whether forces acting on the bone are normal or abnormal in direction or amount, but bone grows in the direction of least resistance. Therefore, forces of occlusion, when acting incorrectly, become factors of malocclusion.

The concept of functional orthodontics is to use appropriate appliances, devices or techniques to modify the forces placed on the jaws of young, growing animals. Such modification in youth encourages growth in a way that corrects, or at least limits the extent of, malocclusion in adulthood. Dental filing to reduce hooks and elongated teeth that interfere with normal jaw growth would be the simplest application. Fixed as well as removable appliances have been utilized with mixed results in an attempt to modify jaw growth and dental arch relationships in the foal (Fig. 17.2).

Surgical correction of dental malocclusion and dentofacial deformities in the horse have seen limited application. The most severe types of deformities such as wry nose have been corrected successfully in a limited number of cases by following the principles of orthognathic surgery.[6–8] Ideal occlusion rarely exists in nature and because of the wide range of variation between individuals we must base our diagnosis of abnormal occlusion, or malocclusion, on a highly arbitrary concept of the imaginary ideal. Malocclusion can be categorized in three etiological types: (a) congenital or genetic malocclusion, (b) eruptive malocclusion and (c) traumatic malocclusion. Fundamental to orthodontic diagnosis is understanding the concepts of normal occlusion.

The incisor teeth are poor teeth on which to base an evaluation of jaw relationship. During the growth and development stages of the foal, the incisors are subject to trauma, retained deciduous teeth, systemic disease, overcrowding and developmental variations in size and number. These problems can potentially alter incisor relationships, allowing erroneous conclusions to be drawn about occlusal patterns.

Many factors are evaluated when determining a 'normal' bite. The incisor table surface is in ideal occlusal contact when the horse's head is in the grazing position. The six upper and six lower incisor teeth are in mesiodistal contact at the occlusal surface with the teeth forming an arch at the front of the mouth. As the horse's head is raised, the mandible slightly retracts, moving the occlusal surface of the lower incisors as much as 3–8 mm caudally.[9] The eruption pattern of deciduous incisors is consistent, with a full set of six deciduous incisor teeth coming into occlusion at 6–9 months of age when the incisive portion of the jaw is wide enough to accommodate these teeth. The shedding of deciduous and the eruption of permanent incisors follows a standard sequence and pattern. Continuous crown attrition and tooth eruption maintains a continually functional exposed crown. The exposed crown length and angle of inclination increases slightly with age, but occlusal contact between the upper and lower arcades should remain consistent. The upper corner incisors may not contact the lower corners during all stages of development and eruption. This is why we see the so-called 7- and 11-year hooks forming on the corner upper incisor teeth. The canine teeth erupt in most male horses between 4.5 and 6 years of age. The upper and lower canine teeth do not make occlusal contact. The root of each upper canine should be positioned in the suture between the maxilla and premaxilla. The lower canines erupt further forward making for longer lower diastemata or interdental spaces. The upper first premolar teeth (wolf teeth) normally erupt just rostral to the second premolar teeth. The first premolars are rarely present in the lower jaw. The first upper premolar is not in occlusal contact with other teeth in the molar arcade.

The upper and lower molar arcades should be in full occlusal contact at their rostral and caudal borders. The upper first molarized cheek tooth (premolar 2) is large and contacts the full occlusal surface of lower premolar 2 and

rostral 1/4 of lower premolar 3. There is a precise interdigitation pattern for the remaining molarized cheek teeth. The last upper cheek tooth (molar 3) has the smallest crown diameter and is in full occlusal contact with the caudal 3/4 of lower molar 3. Any change in the pattern of occlusal contact involving the incisor or molarized cheek teeth will lead to abnormal dental wear patterns and subsequent dental pathology. The horizontal occlusal plane from rostral to caudal of the upper and lower molar arcades is level in most horses. Some horses have a curve or bow in the molar arcades that if severe, can predispose to rostral or caudal ramping of the arcades.

The horse is anisognathic with the lower jaw and molar arcade being narrower than the upper. The hypsodont molarized cheek teeth in each arcade are in rostrocaudal contact at the occlusal surface, so that each arcade appears as one large tooth. The attrition or abrasion of the upper and lower molar arcades is dependent on full rostrocaudal occlusal contact of the arcades during normal mastication. The 10°–15° angle of inclination of the molar tables is caused by attrition resulting in more wear of the palatal aspect of the upper and buccal aspect of the lower arcades. The angles of the root and reserve crowns allow the exposed crown to maintain good occlusal contact throughout the life of the horse. This has been referred to as mesial drift in humans when tooth crowns slightly face the rostral or mesial direction. The horse with its wide diastema has the rostral two cheek teeth facing in the caudal direction, thus keeping the cheek tooth arcade packed tightly together. Therefore, a more specific term to use in the equine would be interproximal drift. During function, teeth move individually in their sockets under the heavy stresses associated with mastication. The crowns of hypsodont cheek teeth taper slightly from occlusal surface to apex. Where adjacent teeth are in contact and constantly erupting and as the crowns are being worn, interproximal drift causes shortening of the dental arcade.[10]

The length and angle of the dental arches varies between breeds and head types. The general shape of the equine skull, its length, height and contours are determined by and allow for the shape of the eyes, upper respiratory system and teeth. The shape of the head varies among breeds but should be in proportion with the body. In 1905, JW Axe proposed a general rule of proportions for head and body size, basing his work on measurements by French hippotomists of the nineteenth century in which the head was used as the basis of proportion for all other body parts.[11]

More recent work by Willoughby (1975) has shown that there are breed differences in the proportion of head to body size.[12] The thoroughbred head usually has a straight line from the ears to the muzzle and is the shortest of all breeds measured when taking the length of the head as a percentage of the withers' height. The Arabian head has an undulating profile with a bulging forehead and a dish below the eyes. Arabians and quarterhorses have shorter heads with the quarterhorse having less of a dish in its face and larger jowls than the Arabian. The standardbred typically has a larger head with a slightly arched face or 'Roman nose.' The draft breeds and Shetland ponies (cold-blooded breeds) have heads that are disproportionally large compared with the light horse breeds. Cheek teeth of Arabian horses are said to be only two-thirds as long and broad as those of cold-blooded horses.[13] It would stand to reason that the more massive the teeth, the more powerful, coarse and common-looking the head must be. As we breed horses to 'refine' the head we reduce the space to accommodate teeth. The relationships of the upper and lower jaws and the dental occlusal paths have a dramatic effect on masticatory function. In the horse with its hypsodont dentition, continuous crown abrasion and attrition results in dental occlusion having a dramatic affect on dental crown wear.

Malocclusion in the horse can involve single teeth in an arcade or the entire dental arch relationship (Fig. 17.3A,B). Severe malocclusion is often accompanied by disproportion of the face and jaws. These problems are referred to as dentofacial deformities.[7] Many minor types of malocclusion are not pathologic but simply equine morphologic variation. Most horses with reasonable dentofacial alignment and occlusion with normal jaw function should be considered to

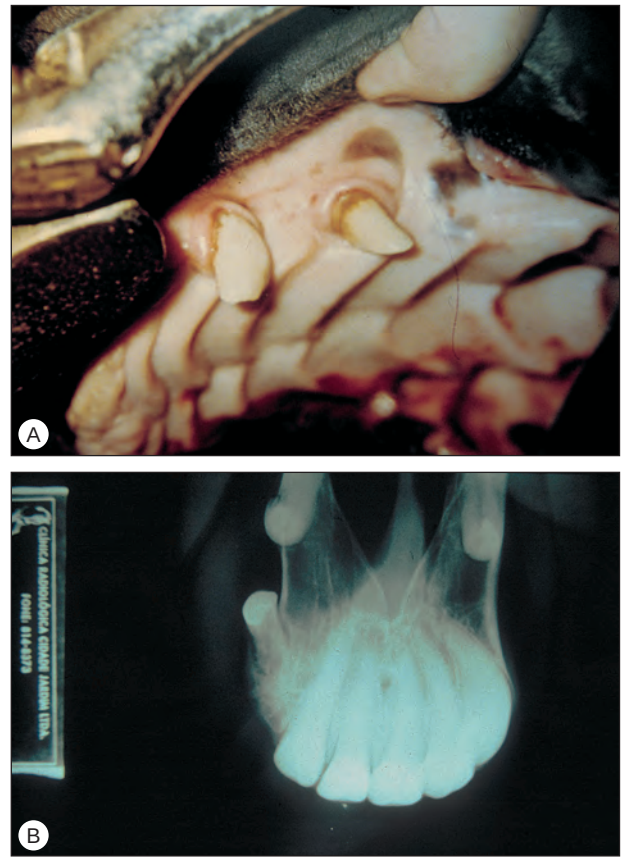


Figure 17.3. (A) and (B) Two caniniform teeth in the maxillary interdental space. A radiograph shows bilateral canine teeth in the normal position. The tooth in the diastema, while having the crown and root shape of a canine tooth, appears to be a displaced corner incisor tooth (C Omura, with permission).

have normal occlusion. While very small abnormalities in occlusal contact can affect dental wear, the impact on function cannot be predicted in all cases from morphology. Equine veterinary literature has given limited space to any type of dental malocclusions with the simple exception of parrot mouth. This condition has been inaccurately stated as the most common malocclusion encountered in equine practice.[14] If the oral examination is limited to the incisor arcades this would appear to be so. Many types of malocclusion occur in the equine species.[15] Colyer (1935) and Joest (1970) described many variations seen in equine teeth that lead to various types of dental malocclusion.[16,17] Hypsodont teeth that are not properly aligned in the dental arcade suffer from severe abnormalities of wear. Abnormal tooth wear has been shown to be the leading cause of dental disease and to adversely affect proper mastication.[18]

To date no research has been done in the equine species to determine what normal craniofacial development and growth entails. Cephalometric studies need to be undertaken to determine normal head and jaw relationships. This chapter presents several types of malocclusions seen in clinical equine practice. Correlations as to the cause and treatment options available are based on information extrapolated from studies carried out in other animal species, including humans. Further academic research is needed to provide better treatment and management of those animals unfortunate enough to be born with or develop various types of dental malocclusions.

Surveys on equine dental patients have shown a high percentage of horses with significant dental malocclusions.[15,19–23] Many of these cases were severe enough to cause clinical problems and a certain percentage were classified as having a handicapping unsoundness.[15] Historically, treatment has been aimed at correcting dental overgrowth.[18,24] More recently, orthodontic techniques have been introduced to equine practitioners to correct some of the more severe problems.[25,26] Poor understanding of biomechanical principles of orthodontics has led to poor clinical results and some severe complications. Dentofacial deformities involve both the dental complex and the facial skeleton. Hopefully, the future will yield a better understanding of the use of combined maxillofacial surgical and orthodontic procedures to correct malocclusions and facial abnormalities.

Factors affecting head shape and dental conformation

It is widely acknowledged that most dental malocclusions in man and animals have a genetic component.[27–29] It is difficult, if not impossible, to quantify how much of a problem is caused by genetics and how much is due to prenatal and postnatal environmental factors. Due to the polygenetic inheritance of craniofacial and dental characteristics, it is extremely difficult at present to identify the cause of most genetic malocclusions. Human studies have concluded that the heritability of skeletal characteristics is high, but that of dental characteristics is low.[30] Studies have shown that cross-breeding dogs of different skull shapes (dolichocephalic, mesocephalic and brachycephalic) reproduces most of the craniofacial and dental malocclusions seen in clinical practice. Tooth malposition without jaw malocclusion has not been documented in these studies.[31] Independence from genetic control of the size of the teeth from both maxilla and mandible was demonstrated. Jaw length and shape and tooth bud position appear to be under genetic control. Tooth form (whether shaped like incisor, canine, premolar or molar) was found to be the most stable characteristic of the canine skull. Additionally, upper and lower jaw conformation and tooth position are independently determined.[31–33]

The genetic nature of some jaw defects seen in horses and cattle has been associated with generalized diarthroidal joint abnormalities.[34–38] Incisor occlusion in various breeds of cattle has been studied and it has been concluded that incisor relationship is heritable but the mode of inheritance cannot be established.[39] Work by Wiener and Gardner concluded that a longer suckling period customary in dairy calf management may reduce the incidence of incisor overjet.[39] Meyer and Becker observed overjet in 31 calves at birth, but found that the anomaly disappeared by 3–6 months of age.[40] Observations in different breeds of sheep have shown that certain skull shapes predispose to certain types of malocclusions. Ovine breeding experiments have alluded to a genetic propensity to malocclusion, but there is some possibility that these conditions can be produced by non-genetic causes such as maternal malnutrition and aging.[41] Goats belonging to the Nubian breeds have strongly convex facial profiles and are predisposed to incisor underjet.[42] The same predisposition has been observed in equine breeds with a dished face.

All domestic horses are of mixed wild origin with widely varying head sizes and types. The size of the teeth and the type and size of the jaws are the legacy of different ancestral types.[13] The genetic determinations of the length of upper and lower jaws as well as tooth size are located on at least three different chromosome sectors (alleles), each of which may be inherited independently of one another.

The genetic and functional environment work closely in determining the resulting growth of all bones.[43,44] The intrauterine environment has a known effect on facial growth and development. Intrauterine molding, when pressure during intrauterine growth distorts the developing face, has been documented in humans and may be the cause of some facial and skeletal limb deformities seen in foals.[45] Postnatal environmental factors include all non-genetic influences brought to bear on the developing individual. These include the environmental effects of muscle function and neuromuscular adaptation.[46,47] In the growing horse, forces placed on the teeth and jaws from abnormal wear patterns on the exposed dental crowns are another factor to consider.

The scientific basis of environmental causes of malocclusion rests primarily on experimental findings with animals.[44] Under certain experimental conditions growth can be modified quite extensively and, in certain circumstances, growth can be stimulated or stunted. The duration rather than the magnitude of soft-tissue pressure has a greater effect on growth. Environmental factors that are recognized as leading to dentofacial abnormalities include: (a) habits of long duration such as sucking, (b) posture of the head, mandible, tongue and lip (posture determines the resting soft-tissue pressures), (c) tooth eruption and crown wear, and (d) trauma (osseous, soft tissue, articular or dental). The current theory for determining craniofacial bone growth states that growth of the face occurs as a response to functional needs and is mediated by the soft tissue in which the jaw is embedded.[48] The soft tissues grow and both the bone and cartilage follow this growth. Function plays an important role in normal jaw growth and is closely related to inherited growth patterns. Jaw growth perturbation may be induced by trauma to the soft tissues.[44] Increased lip pressure results in increased craniofacial disproportion. In response to the disturbance of optimal occlusal relationships, growth of the jaw can be modified to a new functional environment. In humans, it is known that in order to modify inherited jaw growth, the functional disturbance must be of sufficient magnitude and duration (more than 6 hours per day for thumb sucking in children).[5] It has been observed that deviation of the mandible to one side, associated with malocclusion, is common in tethered pigs and is probably due to pulling on their tethers in one direction to reach food and water.[49]

In horses that develop abnormal dental crown wear patterns, functional orthodontics can correct these problems allowing affected horses to realize more fully their genetic potential. Epidemiological studies are needed in order to establish breed or family predisposition to malocclusion. The classification system of malocclusion (modified angle) presently being used in human and small animal dentistry is not well adapted for use in the horse.[2,50,51] While such a classification system does not presently exist for equines, a well-designed system and standardized use by a broad base of well-informed observers is necessary to draw meaningful conclusions. Additionally, cephalometric measurements and studies of jaw interrelation during craniofacial growth in the horse are essential for continued progress in orthodontic therapy.

Sequelae of malocclusion

Horses have hypsodont teeth that are continually erupting and wearing, and malocclusion leads to abnormal wear patterns of the exposed dental crowns. Mechanical forces placed on abnormally wearing teeth can lead to tooth movement in the alveolus. Tipping, rotating or shifting may occur depending upon the angle of force and can lead to diastema (periodontal pocket) formation, the leading cause of periodontal disease.[52,53] Most malocclusions cause teeth to wear in such a fashion as to apply abnormal forces on the teeth and jaws, exacerbating malocclusion (Fig. 17.4A,B). These abnormally worn teeth alter the masticatory pattern in some animals. They can also lead to secondary abnormalities of wear such as altered angle of the molar tables and limited wear of the buccal edges of the upper and lingual edges of the lower arcades. The most severe form of this type of

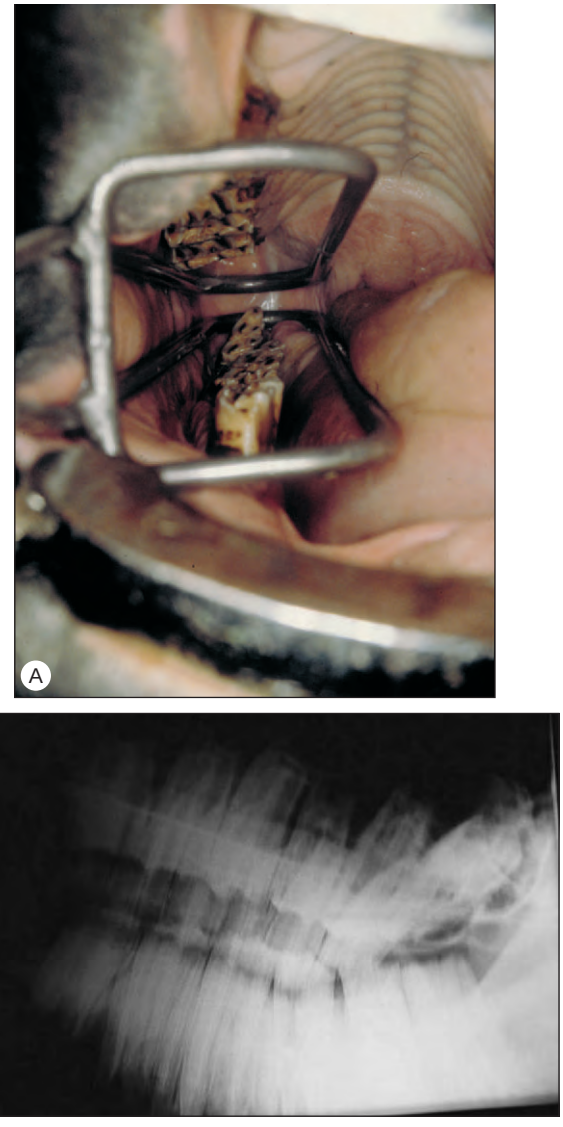


Figure 17.4. (A) and (B) A picture and radiograph of a 4-year-old Appaloosa mare presented with weight loss and quidding hay. Lingual inclination or mesioversion of the lower second molars 310 and 410 has occurred. These teeth are crowded because 311 and 411 have erupted in the curve of the mandible (Curvature of Spee) and are mesially inclined. This has also led to an impaction of the upper 111 and 211. The problem resolved over a 2-year period with frequent crown reductions on lower teeth 10 and 11, keeping them out of occlusion with the uppers. By removing the abnormal masticatory forces from these teeth crowns, they drifted into normal functional occlusion.

altered wear pattern is referred to as shear mouth. Shear mouth is a condition where one or both molar arcades wear at an extremely steep angle, resulting in limited jaw movement.

Examples of altered wear causing tooth movement

Long enamel points or transverse ridges form in the arcade between teeth in the opposing arcade due to malalignment of the upper and lower jaws. These long ridges of enamel act as a wedge between the teeth in the opposite arcade. The enamel wedge forces the teeth apart, creating a diastema into which food will become packed.

Misplaced teeth lead to abnormal crown wear on the occlusal surface, forcing the crown out of alignment with the remaining arcade. The unopposed portion of the crown becomes protuberant and develops an excessive crown angle. Mechanical forces placed on the protuberant crown force the tooth further out of alignment and can cause tipping or increased malalignment of the crown. This leads to periodontal packing of feed around the displaced crown. The tooth in the opposing arcade will not wear normally and may become protuberant or develop excessive enamel points or ridges that mirror the defect in the opposite arcade.

A missing or displaced tooth in one dental arcade will lead to abnormal wear of the opposing teeth. The mesial and distal teeth in the same arcade tend to drift into the space that is unoccupied. This abnormal interproximal drifting can open spaces between adjacent teeth in the same dental arch, leading to diastema and periodontal pockets forming between teeth and inspiring sequential drifting. This is not a consistent feature and at times the entire dental arch will move together and close the gap. This closure shortens the dental arch and predisposes dentition to abnormal wear patterns (hooks) on the ends of the opposite arcade. Some drifted teeth will become angled so that occlusal wear takes place on the side of the crown. The affected angled teeth can have a smooth occlusal surface and excessive crown attrition. This may be associated with a step or wave in the opposing dental arcade.

Rostral 06 or caudal 11 hook formation can place forces on the tooth crown with the protuberance forcing the affected tooth away from the remainder of the arcade. This can cause a diastema, which leads to feed material becoming packed between the teeth. Periodontal disease left uncontrolled may lead to abscess formation, loosening of the tooth in the alveolus and eventually tooth expulsion (Fig. 17.5). Rostral and caudal hook formation can also apply mechanical forces on the jaw that affect growth, mastication, deciduous tooth shedding, head carriage and temporomandibular joint function. As the protuberant crown becomes more prominent at the end of the dental arcade, it can limit



Figure 17.5. Prominent hooks on 106 and 206 due to malocclusion of the upper and lower dental arcades. The rostral pressure placed on this tooth has moved it forward causing a space or diastema between 106 and 107 (arrow). This condition can lead to severe periodontal disease and eventual tooth loss.

rostrocaudal jaw motion and place mechanical forces on the jaws. In the growing foal with a premolar malalignment that predisposes to hook formation, the protuberant tooth crown works mechanically to alter jaw forces and restricts the growth of the shorter jaw. In the adolescent horse with mixed dentition, mechanical forces placed on the jaws and teeth from hooks inhibit growth of the shorter jaw and compress the deciduous tooth crowns, thus limiting the space for shedding deciduous teeth and predisposing erupting permanent teeth to impaction (Fig. 17.6). In the adult horse, hook formation can lead to several pathological processes depending upon the size, shape and position of the hook and the performance demands placed on the horse.

The jaw position changes slightly as the horse moves its head up and down. With the head elevated, the lower jaw retracts caudally in relation to the upper jaw. This can be demonstrated by elevating the head high in the air and



Figure 17.6. Deciduous premolar 606 and 706 caps removed from a 3-year-old quarterhorse with a swelling on the mandible just below 707. The caudal pressure placed on the crown of 706 by the rostral hook on 606 caused crowding and impaction of 707.

noticing the occlusion of the incisor teeth. The cheek tooth arcades also shift with head position. Some speculate that the positional shifting is the reason for a higher incidence of rostral 06 hooks on horses that eat from an elevated hay rack or net as opposed to horses that eat in the normal position off the ground.

As a horse flexes its neck, the lower jaw tends to move forward in relation to the upper jaw.[9] This becomes important when dealing with horses that are asked to perform with the neck bent in collection, such as dressage horses, gaited horses or harness horses worked in an overcheck with their necks forced into flexion. Rostral upper or caudal lower hooks inhibit the forward motion of the lower jaw with the mouth closed. Some horses may tend to open their mouths when collected. Trainers use various nosebands to force the mouth closed, thus preventing relief from the forces placed on the jaws and limiting the amount of flexion the horse can exhibit. Secondary problems such as soreness in the temporomandibular area or in muscles of the neck or back can be associated with the horse's inability to freely move its jaw rostrally and caudally.

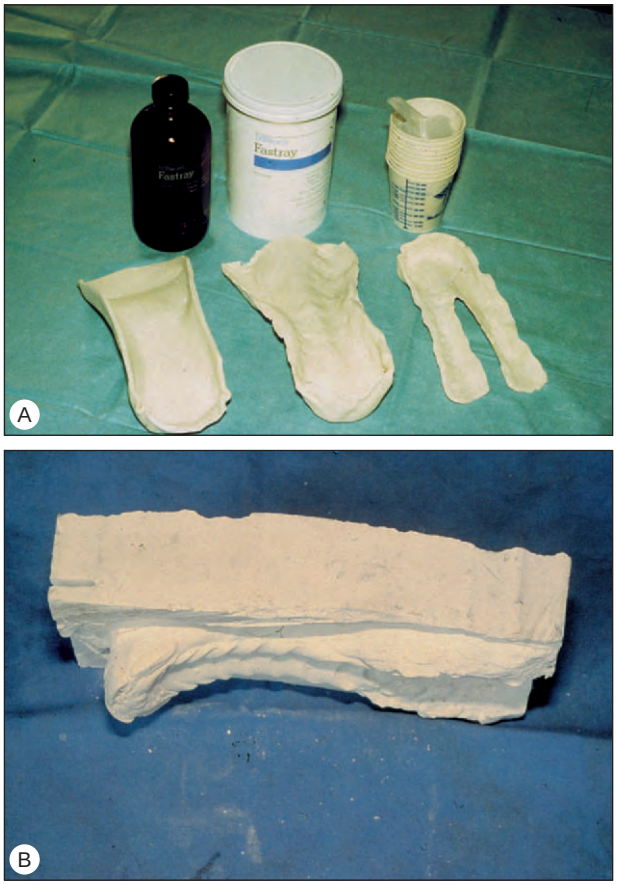


Figure 17.7 (A) and (B) Dental impressions can be taken and stone castings made of all or any portion of the dental arcade. Impression trays are custom made to fit a certain area of the horse's mouth. Plaster of Paris castings are used to document abnormalities and monitor treatment progress.

Documentation of malocclusion and craniofacial deformities

The clinician should document the history and clinical findings of all cases that may require any type of orthodontic treatment. A complete history including the horse's pedigree and an occlusal examination of its parents is helpful in the genetic counseling of the client. Historical information will also allow the clinician to determine whether the condition was noticed at birth or soon after and if it is becoming progressively worse as the horse grows and develops. The proposed use of the horse and breed information are necessary to ethically manage the cases where a hereditary component may be responsible for the deformity.

The clinical assessment should begin with a general physical examination of the patient and a complete, detailed oral examination. Photographs and skull measurements are useful in monitoring clinical progress. Radiographic evaluation of the skull allows for more complete assessment of the problem and is another source of permanent, measurable documentation for monitoring changes over time. Dental impressions and stone castings are helpful in the documentation of deformities as well as in treatment planning (Fig. 17.7A,B). Stone castings can also be used in the fabrication and fitting of removable or fixed appliances. Bite registrations using base plate wax allow for proper alignment of upper and lower stone models as well as for following treatment progress in the live animal.[2]

Parrot mouth

An overjet of the incisor teeth is seen in most animal species, including man. This condition is commonly referred to in the equine species as parrot mouth, brachygnathism, overshot maxilla, buck tooth or overbite. When parrot mouth overjet is slight, the rostral aspect of the lower incisors rests on the lingual aspect of the uppers. When the condition is more severe, the incisors are completely out of occlusion and the premaxilla tends to be bent downward with the lower incisors resting on the hard palate caudal to the upper incisor teeth, causing an overbite. The parrot mouth syndrome in horses can involve the incisor portion of the arcade alone or can occur in combination with varying degrees of malocclusion of the upper and lower cheek teeth arcades. The mismatch in arcade length can be either a brachygnathism of one jaw or a prognathism of another.[16] Mandibular brachygnathism has been reported with other congenital deformity syndromes involving the musculoskeletal system.[35] Without cephalometric norms, it is impossible to categorize these defects as either a short lower jaw or a long upper jaw. Some observations show that the lower jaw is longer (1.5–9.3 per cent) and upper jaw is longer (11.15–18.1 percent) in affected horses.[16] Others conclude that the lower jaw is underdeveloped in some cases of parrot mouth.[17,54] It is not

unusual in adult male horses affected with severe parrot mouth for the upper canines to be positioned rostral to the lower canines.

Cattle inspected for breeding soundness show an incidence of parrot mouth ranging from 2–13 percent. This is in line with the incidence of 2–5 per cent reported in several equine studies.[15,19] The degree to which this condition is expressed at birth and the progression of the problem throughout growth and development of the horse has not been scientifically documented. The parrot mouth condition can be acquired due to avulsion injuries to the incisor teeth or premaxilla, compression fractures of the mandible or illness in a foal immediately prior to or after a growth spurt.[55,56]

Equine parrot mouth is most often the result of breeding two animals with normal dental occlusion but extremely different head types. The classical thoroughbred cross would be the result of a stallion with a sprinter's build and short wide head crossed with a lean-built distance mare with a narrow refined long head. The mixing of different head conformation types produces problems with malocclusions in other breeds. Dish-faced Arabians crossed with narrow, straight-faced American saddlebreds have produced foals with both overjet and undershot jaw conformations. The degree of malocclusion seems to depend upon many factors. Some horses are only affected in the cheek teeth area and others only in the incisor area, while some are affected in both areas. Biomechanically, the horse having hypsodont teeth that depend on normal occlusal contact for wear is more seriously afflicted at all stages in life than most other species.

Few foals are born with the full expression of parrot mouth. Foals born with a slight incisor overjet (upper incisor arcade labial to the lower) will soon develop an overbite (occlusal surface of the upper incisor arcade dropped ventral to the occlusal surface of the lower incisor arcade). As the upper incisors elongate, the palate and incisive bones are pulled downward by gravity. The lower incisors become trapped as they begin to contact the palate behind the uppers. This places caudal pressure on the mandible, inhibiting growth and creating a cascade of events that worsen the deformity. As the lower jaw growth is stunted, the cheek teeth malocclusion worsens, causing rostral hooks to form on the upper first cheek teeth 506 and 606. The unopposed incisor teeth continue to erupt but are not in wear and become elongated. The elongated lower incisors trapped between the wider normal upper incisor arcade, interfere with the normal masticatory cycle and limit free lateral motion of the jaw. This can lead to further abnormal wear of the cheek teeth (Fig. 17.8A,B).

When advising owners about how to manage horses with occlusal abnormalities, keep in mind that it is unclear how malocclusion is inherited. It is a complex conformational trait and is the outcome of multiple genes (polygenetic). Each breeding animal has a different propensity to pass the deformity on to its offspring. It is risky to breed animals that have defects, or to breed animals that have previously produced offspring with defects. Breeders' long-term goals and philosophy should dictate breeding decisions. One should consider the animal's good traits and the seriousness of the defect. Breeding horses with any type of defect will probably increase the incidence of that defect and eventually lead to an intolerable level. An extreme approach would be to neuter animals with defects and remove their sires and dams from the breeding population. While this would prevent animals with a defect from passing it on, it would also prevent them from reproducing their good conformational, performance and behavioral traits. One good strategy would be to not re-mate two animals that have previously produced defective offspring. Another approach would be to re-mate these animals and only retain offspring in the breeding program that do not exhibit the defect. It is a good practice to mate animals with similar virtues and different faults.

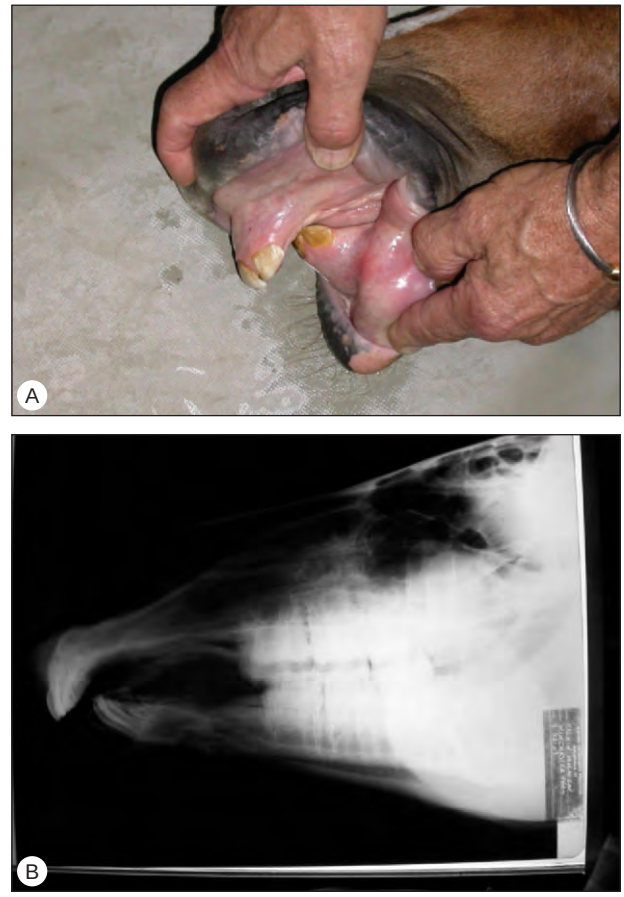


Figure 17.8. (A) Five-month-old parrot-mouthed foal with 1.8 cm overjet and 1.6 cm overbite. (B) Lateral skull radiograph showing a acute vertical curve in the premaxilla. The upper premolar arcade is 0.6 cm rostral to the lower. A protuberance or hook is forming on the rostral edges of 506 and 606.

Any orthodontic management of parrot mouth should follow four basic principles: (1) prevent or reduce abnormal wear of the teeth, (2) prevent or correct downward gravitational drift of the incisive bone and upper incisor teeth, (3) inhibit rostral growth of the maxilla and premaxilla, and (4) stimulate rostral growth of the mandible.

The most important management tool in the correction of parrot mouth is to correct and/or prevent abnormal dental wear. All abnormal dental wear patterns inhibit rostral and lateral movement and growth of the mandible. Rostral hook formation at 106 and 206 and caudal ramping and hook formation on 311 and 411 should be reduced. Excessive transverse ridge formation on the cheek teeth should be reduced and excessive enamel points or vaulted ceiling of occlusion corrected. Excessive incisor length from lack of wear should be reduced to bring the lower incisors out of contact with the soft tissues of the palate. The upper and lower incisors should be reduced to allow free lateral motion of the mandible. Care must be taken to not expose the pulp chamber or damage the tooth during crown reduction.

Foals with minor incisor overjet (less than 5 mm) and with no overbite or cheek teeth malocclusion benefit from occlusal wiring of the upper teeth.[25,26] This technique is used to inhibit rostral growth of the upper jaw from the second cheek tooth forward while allowing the normal growth of the mandible to catch up. This wiring technique is biomechanically unsound for use in animals with incisor overbite or cheek teeth malocclusions.

More severe malocclusion problems have been improved or corrected with the use of functional orthodontic devices early in the horse's life while in the rapid stage of growth and development. Orthognathic surgery has been attempted in a few cases with limited success. Foals born with no contact between the upper and lower incisor teeth have an incisor overjet but no overbite. Within the first 3–6 months of life, gravity and soft-tissue tension on the upper lip cause the premaxilla and incisive bone to tip downward. This downward curve will be evidenced on an oral examination as a bow in the palate midway between the cheek teeth and the incisors. This downward movement of the upper incisors in combination with lack of attrition or wear will lead to an overbite. Removable or fixed functional orthodontic devices combined with retention wires can be used to correct the overbite and allow free movement and rostral growth of the mandible (see Fig. 20.7).

In foals under 6 months of age with sufficient growth left in the lower jaw, tension band wires have been used to inhibit rostral growth of the upper jaw. Stainless steel wire (18–20 gauge, AISI 316L) can be used as a tension band device. Wires placed caudal to the second upper cheek tooth and brought forward around the upper incisors will inhibit growth in this portion of the upper jaw. The lower jaw continues to grow normally and the overjet is corrected.

If no contact is present between any portion of the upper and lower incisor arcade, a combination of tension band wires and a functional orthodontic device is used. Such a device in the most simple form consists of a removable plate attached to a bit, extending rostrally between the incisor arcades. When the mouth is closed, upward pressure is placed on the upper arcade discouraging its ventral drift. A removable orthodontic device can be applied in the standing foal without general anesthesia or invasive surgery. Owner compliance is the most common problem associated with the use of removable appliance, with animal compliance being the second factor limiting their use. Without an educated, enthusiastic and committed owner and/or groom, removable appliances are doomed to failure.

A more sophisticated fixed appliance can be fashioned to fit in the roof of the mouth. This device can be molded from acrylic constructed on a plaster model or fashioned on a live animal in dorsal recumbency under general anesthesia. A metal inclined plane can be incorporated in this device to place rostral force on the lower jaw when the mouth is closed. Application of these devices cannot be 'cook-booked' as each case presents a slightly different set of anatomical and biomechanical situations that requires detailed evaluation and careful planning.

The primary advantage of a fixed dental appliance is that it can be permanently attached in the mouth, making owner and animal compliance less of a factor in case management. Fixed appliances do require special equipment and general anesthesia for their application. With rapid-growing foals and their tendency to put their mouths in and on things that can damage fixed devices, repair and reapplication of the appliance is an all too frequent occurrence. Acrylic fixed appliances that incorporate an aluminum incline plate can be attached to orthodontic retention wires.

The earlier correction is initiated, the better the results. Younger foals in the rapid stages of growth will respond faster and more completely to treatment. It is best to wait until the intermediate upper incisors (503 and 603) are in wear (6–12 weeks of age) to avoid interference with the eruption of these teeth. Prior to orthodontic correction, a full set of skull radiographs and occlusive measurements is indicated. The cheek teeth should be floated to reduce tall transverse ridges and rostral or caudal hooks. The incisor plate will open the bite, thus separating the occlusal contact between the upper and lower cheek teeth arcades. Therefore, 'overfloating' of the occlusal surfaces is discouraged as it prohibits contact during mastication with the incline plate in place. Primiparous mares and dams with small nipples on the udder can present a problem for foals attempting to nurse after surgery. Prior to surgery, foals should be on a diet consisting of a pelleted complete foal ration.

Preoperatively, the foal is medicated with antibiotics and non-steroidal anti-inflammatory drugs. The mouth is rinsed completely with a dilute chlorhexidine solution. General anesthesia is induced with xylazine and ketamine and maintained with a triple drip (a combination of xylazine, ketamine and guaifenesin). The foal is placed in lateral recumbency and a nasal oxygen tube is inserted. Oxygen is delivered at 10 liters/minute during the procedure. The incisor teeth exposed crowns are reduced and leveled with a rasp or power grinder to just above the gum line, taking care not to expose the pulp.

A small area just ventral to the facial crest is clipped and aseptically prepped. With one hand in the mouth, the space between upper PM3 and PM4 on the uppermost side is

identified as an indentation between these teeth in the hard palate. A small skin incision is made at a level just opposite this area below the facial crest, avoiding any branches of the facial nerve, maxillary artery and salivary duct. A 3/32-inch diameter Steinman pin is introduced through the skin incision and directed between the reserve crowns of upper PM3 and PM4 just above the buccal gum line coming out of the mouth 1/8-1/4 inch above the palatal gingiva. Care should be taken to avoid the palatine artery. Intra-operative radiographs and/or fluoroscopy are helpful and necessary at times to properly position the pin between the teeth without damaging dental roots. The pin is removed and a 14 gauge, 1½-inch hypodermic needle is carefully placed in the pin hole to act as a wire guide. A section of 18 gauge stainless steel orthopedic wire is cut to a length at least three times the distance from PM4 to the central incisor teeth. One end of the wire is placed through the hub of the needle exiting in the oral cavity high in the mouth (Fig. 17.9). The needle is removed over the free end of the wire. The free end of the wire is then doubled back and passed through the skin incision and pushed through the buccal mucosa into the oral cavity. Care should be taken not to catch soft tissue or damage a branch of the facial nerve during the process. Forceps are passed through the mouth to grasp the free end of the wire in the buccal space and pull it forward. The palatal end of the wire is also pulled forward, thus forming a loop around the distal aspect of the reserve crowns of upper PM2 and PM3. Kinks in the wires should be avoided as this will lead to premature breaking. The small skin incision is left open to heal by second intention. The foal is then repositioned in lateral recumbency on the opposite side and the procedure for wire placement repeated on the opposite arcade.



Figure 17.9. Parrot-mouthed foal in lateral recumbency for placement of orthodontic wires. A Steinmann pin is used to make a hole between the reserve crowns of 607 and 608 and a 14 gauge needle placed in the hole and used as a guide for the 18 gauge orthopedic retention wire. The needle is pulled over the free end of the wire. A longer free end of the wire is placed back through the same skin incision just below the facial crest, pushed through the mucosa into the buccal recess and brought rostral.

With both wire loops extending out of the oral cavity, the foal is then placed in dorsal recumbency with a pad placed caudal to the poll in order to hyperextend the neck with the roof of the mouth parallel to the ground. The double wire on each side is pulled tight and twisted several times on itself in the interdental space. The wire should be twisted in a manner as to pull downward on the buccal wire and upward on the palatal wire. Keeping the wire high in the mouth is desired to avoid twists contacting the occlusal surface of upper PM2. The twisted double wires from both sides are pulled forward and brought around the labial edge of the upper incisor arcade and twisted together. The wires should lay across the labial surface of the incisors at the gum level. The ends of the wires are cut and tucked between two incisors. A 1/8-inch-thick plate of perforated aluminum is sized to fit over the occlusal surface of the upper incisors and extend back over the hard palate 1/4 inch behind the contact point of the lower incisor arcade. Paraffin rope is placed at the gum level around the upper incisors and pulled under the wires on each side, extending several centimeters back on the roof of the mouth forming a dental dam for acrylic (Fig. 17.10). Hard-setting dental acrylic is mixed and formed into the roof of the mouth, incorporating the wires and buccal surface of the upper incisor arcade. The acrylic should cover the knot in the wire on the labial aspect of the upper incisor arcade, preventing irritation of soft tissue. Splinting of the upper incisor arcade with acrylic is important in order to stabilize the teeth and prevent the force of the orthodontic wires from spreading or twisting the incisors. The acrylic band around the upper incisor teeth and orthodontic wires holds the acrylic firmly in the roof of the mouth. The acrylic is formed with the curved rostral edge of the metal plate resting on the occlusal surface of the upper incisors and the caudal edge of the plate level or slightly higher in the mouth than the rostral aspect. This creates a flat or inclined surface for the



Figure 17.10. Parrot-mouthed foal placed in dorsal recumbency with orthodontic retention wires in place. A paraffin rope dam has been built to retain the acrylic mouth piece. An aluminum plate is cut and sized to fit the occlusal surface of the upper incisors projecting caudal to reach the occlusal surface of the lower incisor arcade.

lower incisors to contact. This inclined plate frees the mandible from caudal force and creates a slight rostral pull as the incisor teeth slide over the plate with chewing. As the foal chews, upward pressure is also applied to the incisors and premaxilla, lifting it into a more normal position (Fig. 17.11A,B).

After the acrylic sets, the foal is allowed to recover from general anesthesia and is placed back with the dam. Most foals quickly learn to nurse with the appliance in place. Foals that do not nurse well need to be supplemented with a complete foal ration and weaned.

Postoperative care consists of keeping the skin wounds clean until healing is complete. While adjusting to the orthodontic appliance, most foals are kept on oral omeprazole for 4–5 days to help prevent gastric ulcers. Postoperatively, most foals eat and nurse well after 1–2 days of adjustment. The plate and wires need to be checked daily at the farm with attention to signs of loose or broken wires or loose acrylic. The foal needs to be examined by a veterinarian on a monthly basis to assure that the appliance is secure and not causing any problems intra-orally. The cheek teeth are inspected and any abnormal wear patterns corrected with careful floating.

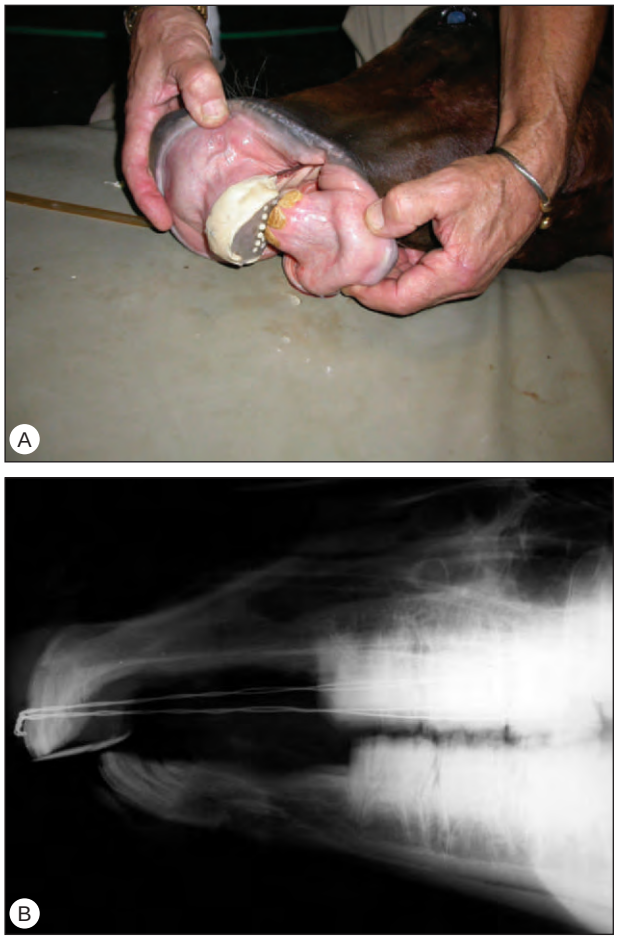


Figure 17.11. (A) Foal with orthodontic wires and acrylic appliance with a metal incline plate in place. (B) Lateral skull radiograph of foal with orthodontic retention wires placed behind the upper third premolar and encircling the upper incisor arcade. The metal incline plate has not been sloped in this first wire application. This would have caused a gap in the premolar arcades. The second appliance will have a slope or mechanical incline to encourage rostral growth of the mandible.

The appliance will loosen and/or wear out over time. Usually after 3–6 months, the appliance and wires need to be removed. If correction is not complete at that time, the surgical process is repeated until desirable results are achieved. Most foals correct about 5 mm every 3–6 months. The most rapid correction is noticed at 2–8 months of age, after which improvement slowly continues to some degree, up to 19 months of age.

Adult horses with the parrot mouth condition that are not diagnosed and corrected at an early age experience long-term adverse effects on dentition and mastication (Fig. 17.12). The molar arcades may develop rostral 06 hooks and/or caudal 11 hooks and/or ramps. Abnormal exaggerated transverse ridges are common and can be quite severe in young horses. The combination of molar abnormalities and elongated incisors tend to limit free lateral excursion of the jaw. This can lead to a steep molar table angle or in some cases, bilateral shear mouth.

Some parrot-mouthed horses function quite well with simply regular dental equilibrations and diet modifications. Dental maintenance entails reduction of rostral and caudal hooks. Abnormal exaggerated transverse ridges and steep molar angles should be corrected. Unopposed upper and lower incisor arcades should be shortened to allow more normal unobstructed jaw motion. Shortening the incisors can be performed in the young horse with deciduous teeth with little risk of damaging the pulp chambers. In older horses with long incisors, reduction is best done in stages.

Older horses with normal incisor occlusion can acquire an incisor overjet due to progressive hooks forming on the upper 06s and lower 11s. As these protuberances form at each end of the molar arcades, they place abnormal caudal forces on the lower jaw. This leads to an increasing malocclusion (overjet) of the incisor arcades. As the condition



Figure 17.12. Adult parrot-mouthed mare with avulsion of four upper right incisor teeth and a portion of the premaxilla. Parrot-mouthed horses are more prone to damaging the long rostrally projected upper incisor teeth. Uneven incisor wear is a sequela of this condition.

becomes more pronounced, the incisors may come out of full occlusion and will likely develop abnormal wear patterns. As the unopposed upper central incisors and lower corner incisors continue to erupt, a ventral curvature or 'smile' wear pattern develops on the incisor arcades. Correction of the cheek teeth wear abnormalities will free up the mandible and allow it to move forward into a more normal physiological position. The abnormally long, unworn areas of the incisor arcade can then come back into occlusion. This will cause the cheek teeth arcades to come out of occlusion, preventing normal mastication of feed. Shortening and leveling the incisors will bring the cheek teeth back into normal occlusal contact.

In addition to routine dental equilibrations on parrot-mouthed horses, diet alterations/modifications should be instituted. Some parrot-mouthed horses have difficulty foraging short-grassed pastures; others have difficulty masticating any type of roughage. Therefore, processed or extruded feeds are beneficial in maintaining these horses in good body condition.



Figure 17.13. (A) and (B) Achondroplasia is a heritable condition in which there is defective development and growth of primordial cartilage. The ancestry of the miniature horse incorporated achondroplastic dwarf mutants into the breed. As in this wry-nosed foal, the genetics are usually partially expressed and only rarely will the head and limb defects as expressed in this case be seen. When shortening of the jaw is marked, the general occlusion is grossly disturbed.

Monkey mouth

The term monkey mouth refers to the condition of the maxilla being shorter than the mandible leading to an incisor underbite. This condition has also been termed sow mouthed, hog mouthed, undershot jaw, underjet, underbite or mandibular prognathism. Retrognathism is a term defined as the mandible being located posterior to its normal position in relation to the maxilla or one or both jaws posterior to normal in their craniofacial relationships. The forward projection of one or both jaws in relation to the craniofacial skeleton has been referred to as prognathism. This condition is seen more commonly in miniature horses and has been associated with achondroplastic dwarfism in cattle.[34,57,58] It can be seen in other breeds of horses, especially those with a dished face such as the Arabian breed. This congenital anomaly has been reported to occur with other deformities of the head and musculoskeletal system. Before therapy is undertaken to correct horses with this type of monkey mouth, owners should receive some genetic counseling (Fig. 17.13A,B).

Principles of therapy in the young growing horse should consist of: (1) encouraging or accelerating growth of the maxilla and premaxilla, (2) supporting the nasal bone and nasal septum, (3) slowing rostral growth of the mandible, (4) preventing interference of the upper and lower incisor arcades, and (5) preventing abnormal wear patterns of the cheek teeth. Adult horses with rostral 06 hooks can develop a slight undershot lower jaw. This abnormality can be corrected with proper molar table floating and periodic incisor reduction.

Wry nose

Deviation of the maxilla, premaxilla and nasal septum, or wry nose (campylorrhinus lateralis), is an infrequently reported condition seen in foals (Fig. 17.14).[59] This condition has been associated with other congenital anomalies such as cleft soft palate, umbilical hernia and contracted tendon syndrome. Affected foals may have difficulty suckling but most seem to thrive until they are several months of age, when dyspnea is often detected. The degree of dyspnea is related to the severity of the nasal septal deviation. Severe cases may require a permanent tracheostomy tube to function.



Figure 17.14. Four-month-old Quarterhorse colt with a wry nose. The premaxilla and upper incisor arcade is deviated to the right at a 60° angle. This foal appeared healthy and growing at a normal rate for his age.





Figure 17.15. (A) and (B) Dorsoventral and lateral radiographs of a 5-month-old foal with wry nose. The lateral deviation of the premaxilla begins at the level of the second premolars. The upper incisor arcade has dropped or curved ventrally. The mandible on this foal has a slight lateral curvature.

As wry-nosed foals are weaned they usually encounter difficulty with prehension and mastication of grass forage. Lateral jaw excursion is limited to the convex side of the premolar arcade in most cases. This limited jaw excursion leads to abnormal wearing of cheek teeth, and shear mouth.

Wry nose is believed to be a heritable condition most often seen in the Arabian and miniature horse breeds.[57–59] However, no scientific evidence exists to support these claims. Affected foals have been successfully treated using principles of orthognathic surgery and have developed into productive athletic and breeding animals. Orthognathic surgical correction requires a two-stage procedure to correct both the dental malocclusion as well as the nasal septal deviation. Candidates for surgery are weaned foals between 5 and 7 months of age.[8,60,61]

Foals should be carefully evaluated to rule out any associated congenital defects and/or aspiration pneumonia. Preoperative oral examination and radiographs can determine the gross nature of the deformity (Fig. 17.15A,B). Computed tomography of the skull has been useful to confirm the position and degree of maxillary deviation and nasal obstruction (Fig. 17.16). Dental impressions of the upper and lower arcades from the premolars rostrally can help the surgeon determine the amount of space that will be created on the concave side of the premaxilla when the deformity is corrected and the upper and lower incisor arcades brought into proper occlusion (Fig. 17.17). Premolar occlusal wear abnormalities should be corrected prior to surgery. This involves re-establishing a normal occlusal angle on the premolar arcades and evening out or leveling the incisors. Preoperatively, the foal should be prepped for a tracheotomy. Occasionally, it will become necessary to pack the nostrils to control hemorrhage during surgery or postoperative swelling may occlude the nostrils. Either of these situations may necessitate respiratory support via tracheostomy.

Prior to surgery and continuing for 5–7 days postoperatively, the foal should be treated with antibiotics, non-steroidal anti-inflammatory drugs and omeprazole. The mouth is rinsed with a dilute solution of chlorhexidine. The foal is anesthetized and positioned in dorsal recumbency. Under sterile conditions an incision is made on each side at the lateral edge of the ventral hard palate from the second premolar rostral to just behind the corner incisor tooth. Both incisions are through the oral mucosa, submucosa and periosteum following the curvature of the deviation (Fig. 17.18). An osteotome is used to cut through the maxilla and nasal processes of the incisive bones, centered over the area of greatest curvature. The premaxilla is then manipulated with slight force into its normal anatomical position. This requires fracturing the palatine process and placing stress on

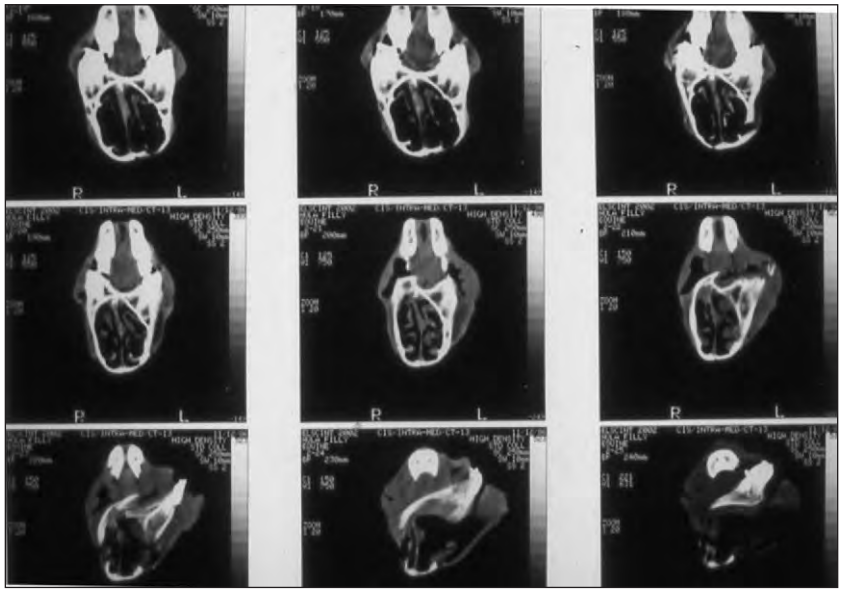


Figure 17.16. Computed tomography images of transverse scans though the head of a 7-month-old thoroughbred filly with wry nose. The filly is positioned in dorsal recumbency and serial sections from the second premolar rostral demonstrate the premaxillary curvature and progressive narrowing of the right nasal passage.

the still cartilaginous nasal bone. The upper and lower incisor arcades should be aligned, which may require some traction and dissection on the unstable upper fragment. The nasal septum can be removed in older foals at this stage of the surgery. However, its presence aids in stabilization of the premaxilla.[62] The upper fragment is stabilized with two unthreaded Steinmann pins inserted between the central and intermediate incisor teeth just above the gum line and seated into the medullary cavities of the maxilla and incisive bones. The pins are cut at the mucosal margins and the tracts left to heal by second intention (Fig. 17.19).

The oral cavity is copiously lavaged with a dilute chlorhexidine solution and the periosteum opposed with absorbable suture. A gap may occur on the concave side which may prevent complete periosteal apposition. The submucosa and



Figure 17.17. Plaster of Paris dental model of the upper arcade from a wry-nosed foal. The model has been cut and straightened to bring the upper and lower incisor arcade into proper occlusion. The white wedge indicates the angle of rostral and lateral displacement required to correct the deformity.

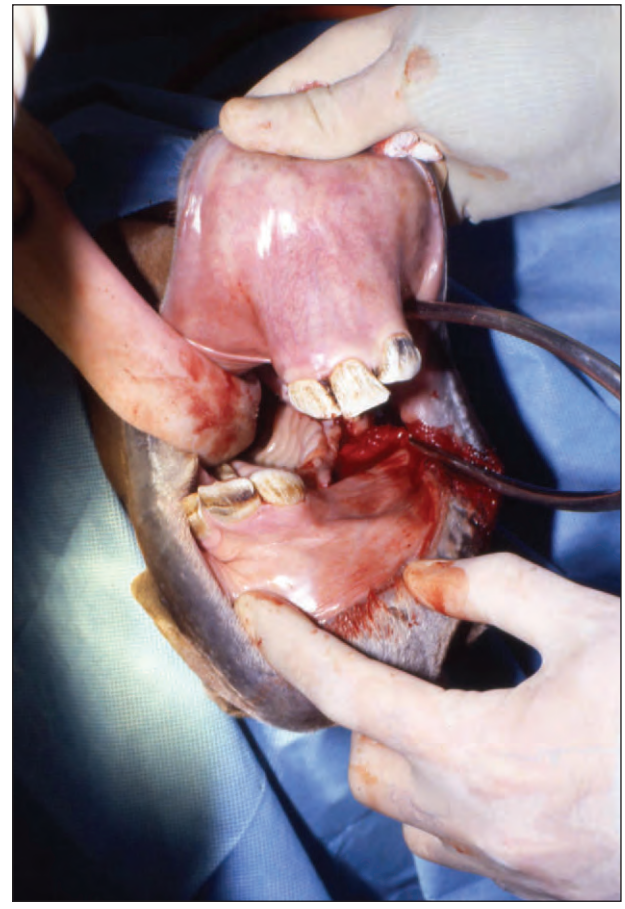


Figure 17.18. Six-month-old wry-nosed foal in dorsal recumbency. An incision has been made on the convex side of the skeletal curvature down through the periosteum. This foal did not have incisor teeth wear abnormalities corrected prior to surgery, making proper alignment of the jaws more difficult.

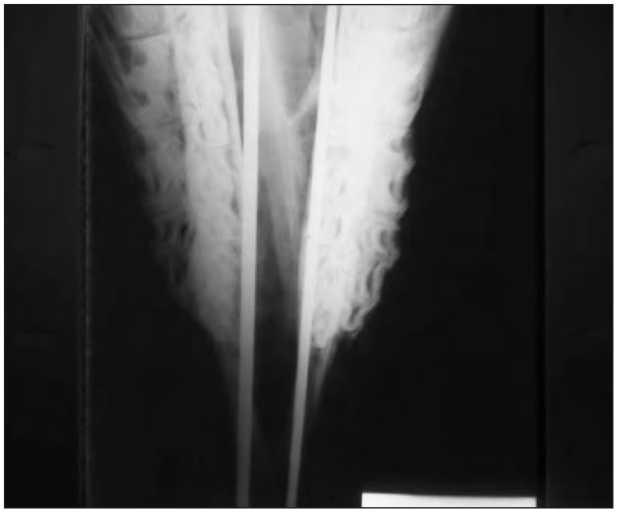


Figure 17.19. Dorsoventral radiograph of an 11-month-old wry-nosed foal 60 days after surgical correction. Two 3/16-inch non-threaded Steinmann pins have been used to stabilize the premaxilla. The osteotomy had healed but the deviated nasal septum needed to be corrected.

oral mucous membranes are closed in layers. From the hard plate and second premolars rostrally to incorporate the upper incisor arcade, a thin acrylic splint is fashioned in the roof of the mouth. This acrylic splint will reinforce the pin stabilization and improve the quality of incisor occlusion.[63]

Some foals benefit from nasogastric feeding for several days postoperatively but this is not necessary in most cases. Foals should be fed a gruel of moistened pellets for 2–3 weeks postoperatively. The foal should have its mouth carefully rinsed daily and inspected for any loosening of the acrylic splint, migration of the pins or dyspnea.

Six to 10 weeks after the first procedure, the foal should be re-evaluated and the acrylic splint and Steinmann pins removed. This can usually be done standing, under sedation. The head should be evaluated radiographically, to confirm healing of the osteotomy site. If the osteotomy site appears to be completely healed and stable, the oral cavity can be examined and any wear abnormalities of the incisors and/or cheek teeth can be corrected.

Most foals require a nasal septal resection to allow proper function without severe dyspnea. The foal is placed under general anesthesia and a tracheotomy performed between two tracheal rings. A soft plastic tracheotomy tube is inserted and sutured in place. The cartilaginous nasal septum is removed opening a 2 × 4 cm hole between the right and left nostrils. Care should be taken not to remove the dorsal and rostral aspect of the nasal septum as this can cause collapse of the nostrils. The nasal passages are packed with sterile gauze to control hemorrhage. The foal is recovered from anesthesia with the tracheotomy tube in place. Two days postoperatively, the nasal packing and tracheotomy tube are removed. A favorable cosmetic and functional result can be expected in cases that survive these corrective procedures. Regular dental care is required to keep the molars and



Figure 17.20. (A) and (B) Twelve-year-old thoroughbred mare with a wry nose. A 'permanent' tracheostomy as a yearling allowed the mare to breathe with no signs of respiratory distress. She had produced six normal foals while being breed to six different stallions. The uncorrected dental and facial deformity caused the mare difficulty with prehension and mastication. She had a severe shear mouth on the concave (right) molar arcade.

incisors in normal wear. Horses not corrected will have life-long problems with eating, breathing and dental wear (Fig. 17.20A,B). Recently, wry nose has been successfully treated using distraction osteogenesis.[64]

Conclusion

All undesirable traits and pathologic conditions present at birth were at one time thought to be entirely genetic in origin. Present-day knowledge has evolved to the point that we now

know that many, if not most, congenital defects are the result of intrauterine events that result from extrauterine influences. Congenital defects caused by viruses and toxins are well documented. Certain conditions that were previously thought to be genetic are now suspected, with good evidence, to be created by viral or toxic insults. Congenital defects do not indicate inheritance but simply that the defect was present at birth.

There are characteristics in horses that are genetically influenced. Horses have been selectively bred for centuries to promote or discourage these characteristics. The selection for or against inherited tendencies is the basis for our current breed registries. Size, power, color, speed, conformation and many other characteristics that are genetically influenced, are selected for or against by certain breed registries. Variations from ideal may be undesirable but they are not genetic defects.

The American Veterinary Medical Association (AVMA) recently restated a policy that surgical correction of 'genetic defects' for the purposes of concealing the defect is unethical.[65] The AVMA statement refers specifically to correction of genetic defects. By definition a genetic defect is a pathologic condition of proven genetic origin. Only three genetic defects have been definitely identified in the equine species: hyperkalemic periodic paralysis in the quarterhorse, lethal white syndrome in the paint horse and combined immunodeficiency in Arabian horses.

While equine practitioners should support the intent of the AVMA statement, it should be applied to genetic defects and not misapplied to congenital defects or inherited tendencies. Surgical correction of debilitating conditions should be considered by the equine practitioner. There is no doubt that correction of debilitating dental malocclusions and facial deformities is in the best long-term interest of the horse's oral and dental health. Some equine breed registries require horses possessing certain undesirable traits and/or conditions commonly considered to be a 'genetic defect,' to have the condition indicated on their registration certificate. This requirement should be brought to the attention of an owner or breeder when a severe malocclusion is diagnosed. It is considered unethical to attempt to correct a known genetic defect in order to allow an owner to misrepresent the animal in the show ring or breeding shed.

In human research, scientists are using elegant image processing as well as microscopic, physiologic, biophysical, biochemical and genetic engineering research procedures to study dentofacial deformities. Clinical observation and detailed documentation will further promote understanding of why and how malocclusions and interjaw malrelations appear and how they can be prevented or treated. The equine practitioner can benefit greatly from the new human biomedical discoveries. Genetic studies to detect the chromosomal factors that play a role in head shape will have a bearing on genetic consultation and mating engineering. Such studies and/or research may help reduce the incidence of equine dental malocclusions in the future.

Equine orthodontic principles are at work in the mouth starting in utero and continuing well into old age. Changes occur as the deciduous teeth erupt, the jaws grow and develop, deciduous teeth are shed, permanent dentition erupts and wear of the hypsodont teeth occurs.

The equine practitioner who is familiar with the principles of diagnosis and documentation of malocclusion is better able to use the controlled movement of teeth and adjustments of jaw growth for treatment. Knowledge obtained through observation, diagnosis, documentation and appropriate adjustments will provide the equine dental patient with the best possible occlusion and will help maintain proper oral health.

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REFERENCES

1. Roberts EW (1994) Bone physiology, metabolism, and biomechanics in orthodontic practice. In: *Orthodontic Practice, Current Principles, and Techniques* (2nd edn), eds. TM Graber and RL Vanarsdall, Mosby, St Louis, pp. 193–234.
2. Wiggs RB and Lobprise HB (1997) *Veterinary Dentistry Principles and Practice*. Lippincott-Raven, Philadelphia, pp. 435–481.
3. Osborn JW (1981) *Dental Anatomy and Embryology*. Blackwell Scientific Publishing, Oxford, pp. 255–260.
4. Scott EJ (1938) An experimental study in growth of the mandible. *American Journal of Orthodontics*, 24, 925–934.
5. Graber TM (1994) Functional appliances in orthodontics. In: *Orthodontic Practice, Current Principles, and Techniques* (2nd edn), eds. TM Graber and RL Vanarsdall, Mosby, St Louis, pp. 383–436.
6. Musich DR (1994) Orthodontics and orthognathic surgery. In: *Orthodontic Practice, Current Principles, and Techniques* (2nd edn), eds. TM Graber and RL Vanarsdall, Mosby, St Louis, pp. 835–907.
7. Tucker MF (1993) Correction of dentofacial deformities. In: *Contemporary Oral and Maxillofacial Surgery* (2nd edn), Mosby, St Louis, pp. 613–656.
8. Valdez H, McMullen W, Hobson H *et al.* (1978) Surgical correction of a deviated nasal septum and premaxilla in a colt. *Journal of the American Veterinary Medical Association*, 173, 1001–1004.
9. Carmalt JL, Townsend HGG and Allen LA (2003) Effect of dental floating on the rostrocaudal mobility of the mandible of horses. *Journal of the American Veterinary Medical Association*, 23, 666–669.
10. Gidley JW (1901) Tooth characters and revision of the North American species of the genus equus. *Bulletin of American Museum of Natural History*, 14, 91–141.
11. Axe JW (1907) *The Horse: Its Treatment in Health and Disease*, Vol. 1. Gresham Publishing, London, pp. 36–51.
12. Willoughly DP (1975) *Growth and Nutrition in the Horse*. AS Barnes, New York.

13. Shafer M (1980) *An Eye for a Horse*. JA Allen, London, pp. 103–107.
14. Orsini PG (1992) Oral cavity. In: *Equine Surgery*, ed. JA Auer, WB Saunders, Philadelphia, pp. 299–300.
15. Uhlinger C (1987) Survey of selected dental abnormalities in 233 horses. *Proceedings of the 33rd Annual Meeting of the American Association of Equine Practitioners*, pp. 577–583.
16. Miles AEW and Grigson C (1990) *Colyer's Variations and Diseases of the Teeth of Animals* (rev. edn), Cambridge University Press, Cambridge.
17. Joest E (1970) *Handbuch der Speciellen Pathogischem Anatomie der Haustere,* Vol. V/I, Paul Parey, Berlin and Hamburg.
18. Dixon PM (2000) Removal of equine dental overgrowths. *Equine Veterinary Education,* 12(2), 68–81.
19. Duke A (1989) Equine bit analysis. Handout notes from Annual Conference of American Veterinary Dental Society, New Orleans.
20. Dixon PM, Tremaine WH, McGorum BC *et al.* (1999) Equine dental disease — a long term study of 400 cases, part 1: Introduction and disorders of incisor, canine and first premolar teeth. *Equine Veterinary Journal*, 31, 369–377.
21. Dixon PM, Tremaine WH, McCorum BC *et al.* (1999) Equine dental disease — a long term study of 400 cases, part 2: Disorders of development, eruption and variations in position of cheek teeth. *Equine Veterinary Journal*, 31, 519–528.
22. Dixon PM (2002) The gross, histological and ultrastructural anatomy of equine teeth and their relationship to disease. In: *Proceedings of 48th Annual Convention of the American Association of Equine Practitioners*, pp. 421–437.
23. Kilic S, Dixon PM and Kempon SA (1997) A light and ultrastructural examination of calcified dental tissues of the horse: 1. The occlusal surface and enamel thickness. *Equine Veterinary Journal*, 29, 190–197.
24. Baker GJ (1985) Oral diseases of the horse. In: *Veterinary Dentistry*, ed. CE Harvey, WB Saunders, Philadelphia, pp. 220–221.
25. McIlwraith CW (1984) Equine digestive system. In: *The Practice of Large Animal Surgery*, ed. PB Jennings, WB Saunders, Philadelphia, pp. 558–560.
26. DeBowes RM (1990) Brachygnathia. In: *Current Practice of Equine Surgery*, eds. AN White and JN Moore, JB Lippincott, Philadelphia, pp. 469–472.
27. Emily P (1990) The genetics of occlusion. *Veterinary Forum*, June, 22–23.
28. Goldstein G (1990) The diagnosis and treatment of orthodontic problems. In: *Problems in Veterinary Medicine (Dentistry)*, 2(1), 195–213.
29. Kertesz P (1993) Orthodontics. In: *A Color Atlas of Veterinary Dentistry and Oral Surgery*, ed. P. Kertesz, Wolfe Publishing, London, pp. 63–72.
30. Harris EF and Johnson M (1992) Heritability of craniometric and occlusal variables: a longitudinal sibling analysis. *American Journal of Dentofacias Orthopedics*, 99, 258.
31. Stockard CR (1941) The genetic and endocrine basis of differences in form and behavior. *The American Anatomical Memoirs*, No. 19, Wister Institute Anatomy and Biology, Philadelphia.
32. McKeown M (1975) Craniofacial variability and its relationship to disharmony of the jaws and teeth. *Journal of Anatomy*, 119(3), 579–588.
33. McKeown M (1975) The allometric growth of the skull. *American Journal of Orthodontics*, 67(4), 412–422.
34. Jayo M, Leipold HW, Dennis SM and Eldridge FE (1987) Brachygnathia superior and degenerative joint disease, a new lethal syndrome in Angus calves. *Veterinary Pathology*, 24, 148–155.
35. Lear TL, Cox JH and Kennedy GA (1997) Autosomal trisomy in a thoroughbred colt: 65, XY, +31. *Equine Veterinary Journal*, 31(1), 85–88.
36. McLaughlin GB and Doige LE (1981) Congenital musculoskeletal lesions and hyperplastic goiter in foals. *Canadian Veterinary Journal*, 22, 130.
37. Myers VS and Gordon GW (1975) Ruptured common digital extensor tendons associated with contracted flexor tendons in foals. In: *Proceedings of the 21st Annual Convention of the American Association of Equine Practitioners,* p. 67.
38. Buton TE (1985) Spontaneous craniofacial malformations and central nervous system defects in an aborted equine fetus. *Journal of Comparative Pathology*, 95, 131–135.
39. Wiener G and Gardner WJF (1970) Dental occlusion in young bulls of different breeds. *Animal Production*, 12, 7–12.
40. Meyer VH and Becker H (1967) Eine Erbliche Keiferanomalie Bein Rind. *Deutsche Tierarztliche Wochenschrift,* 74, 309–310.
41. Purser AF, Wiener G and West DM (1982) Causes of variation in dental characters of Scottish blackface sheep in a hill flock and relations to ewe performance. *Journal of Agricultural Science*, 99, 287–294.
42. Epstein H (1971) *The Origin of the Domestic Animals of Africa*, Vol. 2. Africana Publishing, London, p. 296.
43. Petrovik AG (1984) Experimental and cybernetic approaches to the mechanism of action of functional appliances of mandibular growth. In: *Malocclusion and the Periodontium, Cranial Growth Series*, ed. JA McNamara (monograph), p. 15.
44. Burdach J and Mooney MP (1984) The relationship between lip pressure following lip repair and craniofacial growth: an experimental study in beagles. *Plastic and Reconstruction Surgery*, 73(4), 544–555.
45. Crowe MW and Swerczek TW (1985) Equine congenital defects. *American Journal of Veterinary Research*, 46, 353–358.
46. McNamara JA (1973) Neuromuscular and skeletal adaptations to altered function in the orofacial region. *American Journal of Orthodontics*, 64, 136, 189, 578.
47. McNamara JA (1980) Functional determinants of craniofacial and shape. *European Journal of Orthodontics*, 2, 131.
48. Hennet PR and Harvey CE (1992) Craniofacial development and growth in the dog. *Journal of Veterinary Dentistry*, 9(2), 11–18.
49. Harvey CE and Penny RHC (1985) Oral and dental diseases in pigs. In: *Veterinary Dentistry,* ed. CE Harvey, WB Saunders, Philadelphia, pp. 481–551.
50. Angle EH (1899) *The Angle System of Regulation and Retention of the Teeth and Treatment of Fractures of the Maxillae* (5th edn). SS White Manufacturing, Philadelphia.
51. Hennet PR, Harvey CE and Emily PP (1992) The angle classification of malocclusion: is it appropriate for use in veterinary dentistry? *Journal of Veterinary Dentistry*, 9(3), 10–12.
52. Carmalt JL (2003) Understanding the equine diastema. *Equine Veterinary Education,* 5(1), 43–44.
53. Greene S and Basile JP (2002) Recognition and treatment of equine periodontal disease. In: *Proceedings of the 48th Annual Convention of the American Association of Equine Practitioners,* pp. 463–466.
54. Becker E (1970) Zähne. In: *Handbuch der Speziellen Pathologischen Anatomie der Haustiere*, (3rd edn), Vol. 5, Alimentary System, Paul Parey, Berlin, pp. 83–313.
55. Pence P and Wilewski K (2002) Newborn, weanling and adolescent horse dentistry. In: *Equine Dentistry: A Practical Guide,* ed. P Pence, Lippincott, Williams and Wilkins, Baltimore, pp. 115–139.
56. Boero M (2003) Correction of brachygnathism in foals. In: *Manual of Equine Dentistry,* ed. T Allen, Mosby, St Louis, p. 173.
57. Mitz C and Allen T (2003) Dentistry in miniature horses. In: *Manual of Equine Dentistry,* ed. T Allen, Mosby, St Louis, pp. 175–192.
58. Pence P and Mitz C (2002) Miniature horse dentistry. In: *Equine Dentistry: A Practical Guide,* ed. P Pence, Lippincott, Williams, and Wilkins, Baltimore, pp. 191–199.
59. Baker GJ (1998) Problems involving the mouth. In: *Equine Internal Medicine,* eds. S Reed and W Bayly, WB Saunders, Philadelphia, p. 605.

60. Easter JL and Watkins JP (1999) Diseases of the head and neck. In: *Equine Medicine and Surgery,* ed. PT Calahan, AM Merritt, JN Moore and JG Mayhew, Mosby, St Louis, p. 1669.
61. Stashak TS (1991) *Equine Wound Management.* Lea and Febiger, Philadelphia, pp. 139–144.
62. Yarbrough TB, Carr EA, Snyder JR and Hornof JW (1997) Nasal septoplasty for correction of septal deviation in a foal. *Veterinary Surgery,* 26, 340–345.
63. Peavey CL, Edwards RB, Escarcega AJ *et al.* (2003) Fixation technique influences the monotonic properties of equine mandibular fracture constructs. *Veterinary Surgery,* 32, 350–358.
64. Puchol JL, Herran R, Durall L, Lopez J and Diaz-Bertrana C (2004) Use of distraction osteogenesis for the correction of deviated nasal septum and premaxilla in a horse. *Journal of the American Veterinary Medical Association,* 224(7), 1147–1150.
65. American Veterinary Medical Association Membership Directory (2003) Principles of veterinary medical ethics of the AVMA (1999 revision) IV. *Therapies, Genetic Defects,* 36.

18 Exodontia

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PART 1: Equine tooth removal

Introduction

Equine tooth removal has been described in the early veterinary literature[1] and remains the most frequently performed oral surgery in the horse. Wolf tooth removal was described for alleviation of behavioral problems associated with the bit in the eighteenth century[2,3] and is still performed for this reason. Tooth removal is also performed commonly for treatment of advanced dental disease, which is non-responsive to more conservative treatments. Although it is possible to perform endodontic treatments in selected cases with severely diseased teeth, in order to preserve the tooth, the current technical limitations mean that extraction of severely diseased teeth often remains the only viable option.

Tooth removal should be considered after other more conservative treatments have failed or offer a poor prognosis. The technique selected will depend on the tooth to be extracted, the nature of the disease affecting that tooth and the preference of the veterinary surgeon performing the procedure. Dental removal has historically been thought of as a straightforward or unsophisticated procedure. However, the high complication rate reported in the literature indicates that equine dental removal is a technically challenging procedure demanding careful preparation, specialized equipment and meticulous technique to avoid complications. Dental removal ranges from relatively straightforward to extremely complicated and frustrating for both patient, owner and veterinarian, and the potential costs and complications should be addressed at the outset. While many dental extractions can be performed in the sedated standing horse, the necessity for general anesthesia in difficult patients, or for complicated procedures, must always be considered. Intra-operative radiography or fluoroscopy is an essential aid for a successful outcome.

The most common reasons for dental removal include:

- retained deciduous incisors or premolars
- teeth affected by severe periodontal disease
- loose teeth
- fractured teeth
- displaced or malaligned teeth
- supernumerary teeth
- dental impaction
- teeth with apical abscesses
- teeth devitalized as a consequence of mandibular or maxillary fracture
- dental overgrowths resulting in severe soft-tissue trauma
- orthodontic surgery.

Incisor removal

Deciduous incisors are often associated with avulsion fractures of the mandible or incisive bone (premaxilla) in foals and young horses. Where possible, and where soft-tissue attachments remain, the teeth should be salvaged and incorporated into any repair. Many incisors that may initially appear to be devitalized are salvageable if sufficient soft-tissue attachment and immobilization can be achieved. Deciduous incisors that are devitalized may be removed with minimal effect on the subsequent eruption of the permanent secondary dentition (see Chapter 21).

Retained deciduous incisors (Fig. 18.1), not displaced by the permanent incisors, which normally erupt on the palatal or lingual aspect of the temporary teeth, may result in deviation of those teeth as they erupt. These retained teeth rarely cause difficulty with food prehension and are of cosmetic consequence only. Deciduous incisor removal is easily performed in the standing sedated horse, and local gingival infiltration of mepivicaine or lidocaine can be used if necessary. Deciduous incisors have short reserve crowns, and the periodontal attachments are easily loosened with a small periodontal elevator (Fig. 18.2). Once loosened, the tooth can be extracted successfully using small wolf tooth extracting forceps (Fig. 18.3). No treatment of the vacant

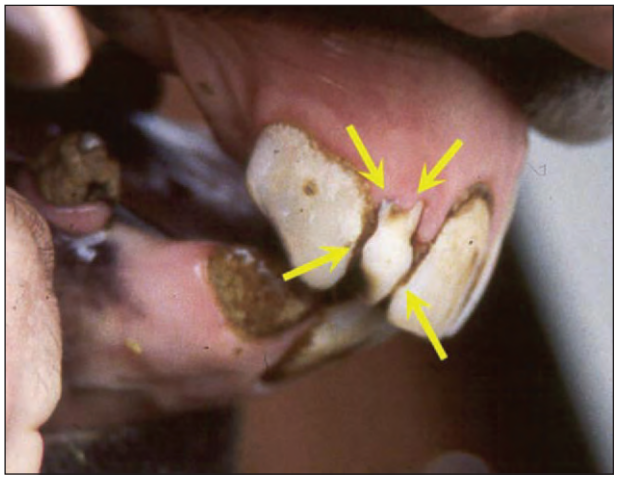


Figure 18.1. Retained deciduous incisors (arrows) rarely prevent eruption of the permanent incisors and are usually of cosmetic significance only.



Figure 18.2. Small periodontal elevators such as these are suitable for elevating incisors and 'wolf' teeth.



Figure 18.3. Small dental extractors that are suitable for removing retained deciduous incisors.

alveolus is necessary, although tetanus prophylaxis should be ensured.

Supernumerary incisors

Supernumerary permanent incisors occur sporadically, and more than one may be present. The occlusal surfaces of the teeth appear similar to normal incisors and they have reserve crowns, which are often equal in length and shape to normal incisors. Radiographs are useful to detect unerupted permanent incisors and to discriminate between retained primary teeth and supernumerary incisors. However, despite careful examination and radiographs it can be difficult to distinguish the supernumerary teeth from the normal teeth. Supernumerary permanent incisors usually cause minimal signs of disease other than cosmetic asymmetry, and horses with them rarely suffer any difficulty with food prehension. The extensive apices render the extraction of supernumerary incisors a major surgical undertaking (Fig. 18.4) and it is usually unnecessary and of little benefit to the horse. The removal of all six mandibular incisors in a vicious stallion has been reported, although this procedure is seldom



Figure 18.4. Supernumerary incisors may have long apices. In addition to their close association with adjacent incisors, this makes their extraction difficult.

indicated. Extraction of permanent incisors may necessitate general anesthesia, because horses appear to be sensitive to incisor concussion. In compliant horses the procedure may be performed under sedation with an alpha-2 agonist and opiate analgesic. Regional desensitization of the lower incisors can also be facilitated by perineural anesthesia of the mandibular nerve as it emerges from the mental foramen (Fig. 18.4) on the lateral aspect of the mandible in the interdental space. Supernumerary incisors can be removed by freeing the periodontal attachments around the whole circumference of the tooth gradually until it is sufficiently loose to extract. Teeth which cannot be sufficiently loosened can be sometimes extracted after making a gingival incision and removing part of the labial alveolar plate, using a narrow (1 cm) osteotome. The incisor can then be loosened with a periodontal elevator until sufficiently loose to extract with a pair of small incisor extraction forceps. Incisors which have become totally separated from their gingival attachments as a result of trauma or avulsion of the corner of the incisive bone or rostral mandible should be removed. However, incisors that retain some gingival attachments may remain viable and can often be salvaged after reduction and immobilization of the fracture using stainless steel wire.

Removal of canine teeth

There are few indications for removal of canine teeth. The exceptions are severe periostitis, possibly associated with bit injuries or pulpitis resulting from fractures of the canine or of the surrounding mandible or incisive bone. Canine teeth are frequently the site of tartar accumulation, which may result in gingival recession and periodontal disease (Fig. 18.5), although the clinical significance is undetermined. Removal of the sharp clinical crowns to reduce canine-induced injury to other horses and during oral examinations is as effective and less invasive than removal of the entire tooth. Loose canines can be removed by further periodontal elevation and subsequent extraction of the tooth using incisor or small animal dental forceps. The canine tooth has a sharp curvature and is widest below the level of the alveolar crest, which renders those with intact peridontia difficult to remove. Endodontic treatments may be applicable for apically infected teeth before resorting to dental removal. Removal of the alveolar bone on the lateral aspect of the canine tooth is necessary before extracting long teeth (Figs 18.6 and 18.7), although care should be taken to avoid trauma to the mental nerve emerging from the mental foramen on the lateral aspect of the mandible in the interdental space.

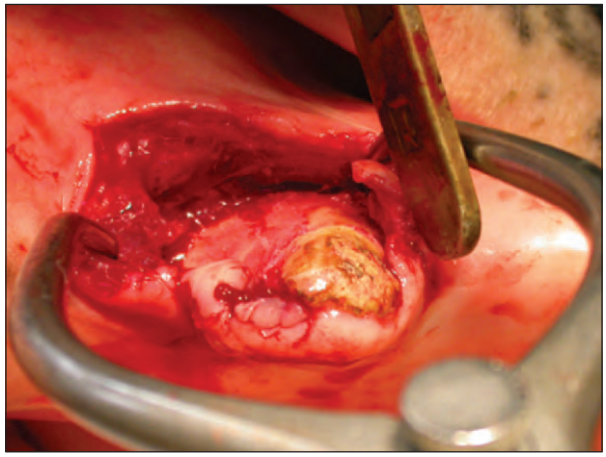


Figure 18.6. The extensive apical of the canine tooth portion necessitated a lateral alveolar osteotomy to expose the tooth to enable its extraction.

Wolf tooth extraction

The first upper premolar (Triadan 105, 205) is commonly referred to as the 'wolf tooth,' and is highly variable in position, shape and size. The clinical crown may emerge on the lateral aspect of the mandible or maxilla, or may be subgingival, and is a poor indicator of the size of the hidden clinical crown. Upper wolf teeth are present in 40–80 per cent of horses, although they may be unilateral.[4] Eruption times



Figure 18.5. This horse presented with resentment to palpation of this canine tooth, which showed radiographic periodontal changes.

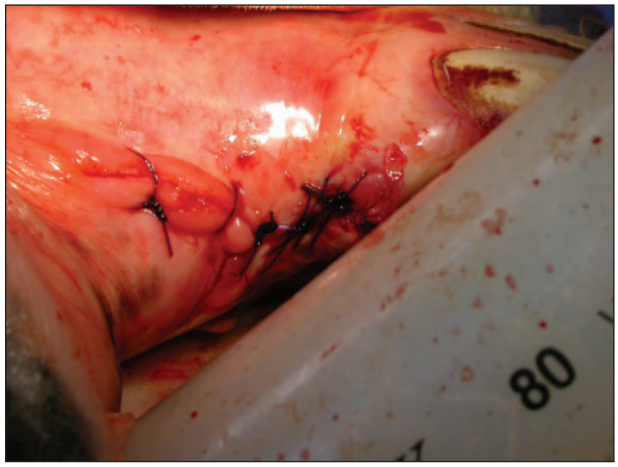


Figure 18.7. The gingiva was repaired with absorbable monofilament suture, following extraction of the canine tooth.

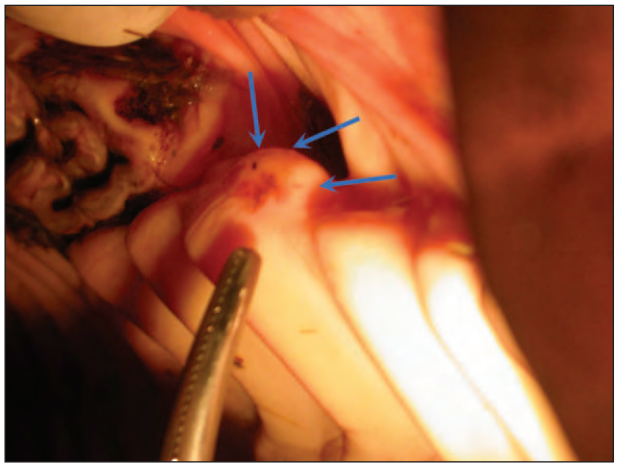


Figure 18.8. Unerupted wolf teeth (arrows) are usually palpable subgingivally in the interdental space.

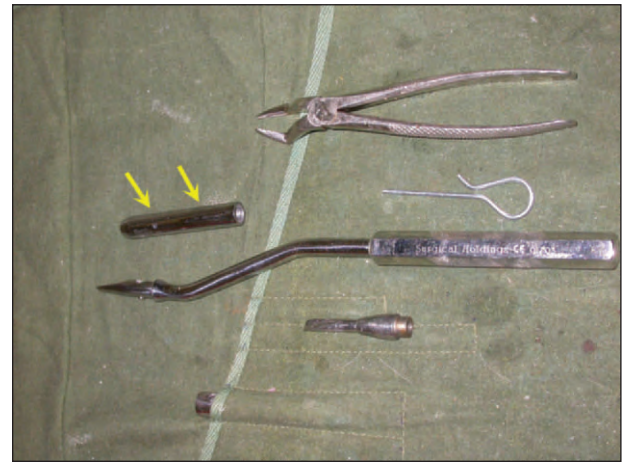


Figure 18.9. The Burgess elevator enables the peridontium around the 'wolf' tooth to be elevated around all 360°, thus facilitating its subsequent extraction.

are also variable but usually occur between 6 and 18 months. The most common site is immediately rostral to the second upper premolar (Triadan 106, 206); however, they are not infrequently displaced on the buccal aspect or palatal aspect. Unerupted wolf teeth are usually detectable by palpating hard subgingival nodules in the interdental space (Fig. 18.8) and these are occasionally associated with gingival ulceration. Radiographs may be useful to demonstrate the size and direction of the apical portion before attempting extraction of subgingival wolf teeth.

Wolf teeth are traditionally extracted, although evidence for the clinical benefit of this procedure is controversial. In some individual horses, especially those with extremely large, sharp or aberrantly placed or mobile wolf teeth, pain is associated with the wolf teeth, resulting in non-acceptance of the bit, or performance-related disorders. Entrapment of the buccal mucosa between the bit and the wolf tooth or rostrolateral aspect of the second premolar can result in discomfort and consequently a lack of responsiveness on the bridle. In addition, rasping material from the rostral and buccal aspect of the second premolar (first cheek tooth, Triadan 106, 206, 306, 406), can be hindered by the presence of the wolf teeth. Consequently, wolf teeth are usually prophylactically removed, especially prior to creating so-called 'bit-seats.'

Wolf teeth can be usually extracted *in toto* under standing chemical restraint often used in combination with subgingival local anesthesia, or perineural analgesia of the mental nerve. Wolf teeth with a normal configuration can be extracted easily by elevation of the periodontal attachments using a Burgess type extractor (Fig. 18.9) or circumferentially elevating the tooth using a small curved Coupland periodontal elevator (Fig. 18.10). Aberrantly placed teeth or very large wolf teeth should be radiographed to assess the size and placement of the apical portion before attempts at extraction. Once periodontal elevation is completed, the tooth can be extracted using a small pair of incisor or specialized wolf tooth extractors. Subgingival wolf teeth can be exposed by a small (Fig. 18.11) incision in the overlying gingival mucosa. Periodontal attachments can be carefully loosened using an osteotome placed pointing caudally between the rostrally angled tooth and the hard palate (Figs 18.12 and 18.13). The alveolus can be packed with gel foam or gauze to prevent food impaction, although this is rarely necessary. Failure to loosen the periodontium sufficiently can result in fracture of the tooth. Remaining sharp fragments should be elevated and removed to enable healing of the alveolus. Subgingival apical fragments rarely cause clinical signs, and loose fragments may be removed after several days during which they usually migrate to a more superficial position. Mandibular lower first premolars (Triadan 305, 405) are rarer and if present can usually be palpated on the ventral mandible rostral to the first cheek tooth. They are commonly small, but vary in size and position and are more likely to be associated with discomfort with the bit, and are a noteworthy observation during



Figure 18.10. Wolf tooth extraction set containing Coupland and Burgess type elevation and extraction forceps.

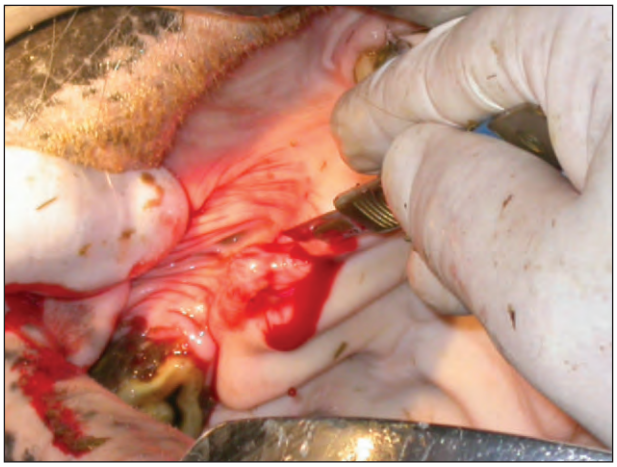


Figure 18.11. A small gingival incision exposes subgingival wolf tooth prior to extraction.

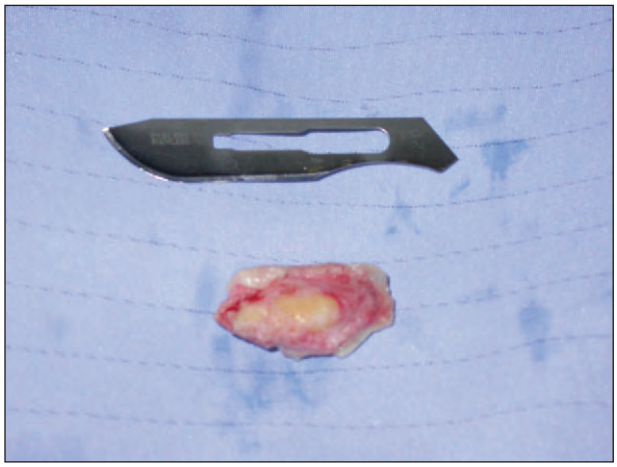


Figure 18.13. A subgingival wolf tooth post-extraction.

prepurchase examinations. The technique for their removal is as described previously.

Deciduous premolar extraction

The permanent premolars erupt between approximately 2.5 and 4 years of age, displacing the remnants of their primary counterparts. The nutrition to the primary dentition is eventually compromised by pressure on its roots from the erupting secondary tooth. The worn crown of the primary tooth or 'cap' eventually loses its gingival attachments and normally becomes loose and is shed spontaneously during mastication. The eruption sequence of the secondary dentition sometimes results in delayed shedding of the deciduous molars. This can result in entrapment of the cap between the adjacent erupted secondary teeth. The cap overlying the premolars 307, 308, 407 or 408 is most frequently involved. Accumulation of food under the cap after recession of the gingival attachments or the presence of dental fragments remaining subgingivally if shedding is incomplete can result in gingivitis and oral pain. This can present as non-acceptance of the bit or poor performance, especially in racing thoroughbreds. Impacted primary dentition has been implicated as a cause of displacement of the erupting secondary dentition.[5] Facial swellings on the rostral maxilla or ventral mandible are sometimes present, usually with only cosmetic consquences.[6]

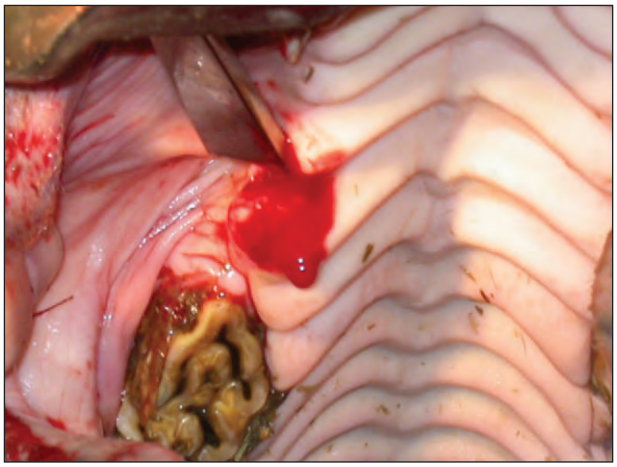


Figure 18.12. Large sublingual wolf teeth may require additional loosening using a narrow osteotome placed in the periodontal pocket and directed caudally.

The indications for removal of caps include: oral pain or gingivitis associated with retained primary molars, caps which are digitally loose to palpation, and those in which there is no gingival attachment to the deciduous tooth. The latter can be identified by the presence of a palpable fissure between the retained cap and the underlying secondary tooth, which has no overlying gingival. Radiographs can be helpful to confirm the presence of retained primary molars. Various cap extraction forceps are available for removal of retained primary molars, such as Reynolds cap extractors. The technique for their removal is straightforward. For more rostral teeth the cap can, on some occasions, be elevated by inserting a flat-bladed elevator into the fissure between the deciduous and underlying permanent premolar on the buccal aspect, and levering it in a coronal direction. The cap is extracted by grasping it firmly with suitable forceps, and withdrawing it lingually in order to loosen and displace the cap while attempting to avoid fragmentation of the buccal roots. Care is taken to avoid tearing of gingiva, especially on the buccal aspect of maxillary caps. Any slivers of tooth resulting from root fracture can be removed with a small dental pick. The underlying permanent tooth will normally be in wear in 3–4 months. The removal of deciduous premolars that are not loose and not associated with clinical signs or oral discomfort is not advised purely on the basis of the horse's age. Premature exposure of the secondary dentition may occur, and potentially predispose the permanent to caries.

Permanent cheek tooth extraction

Currently in many cases, conservative treatments of severe dental diseases are unsuccessful and dental removal remains

the main course of treatment.[7] Dental extraction has been practiced in the conscious horse since the nineteenth century with little modification in the available techniques.[8] The technical difficulties and development of improved sedatives and anesthetics in the latter half of the twentieth century contributed to the search for alternative, more invasive techniques and consequently dental repulsion became widely practiced. The technical limitations of this technique and the high incidence of complications[7,9] necessitated the exploration for further techniques. Removal of the rostral three mandibular cheek teeth or the rostral three maxillary cheek teeth via a buccotomy and lateral alveolar osteotomy is an alternative technique to repulsion and is associated with a lower incidence of complications.[10,11] However, this technique is technically more complicated, always necessitates general anesthesia, and iatrogenic damage to branches of the dorsal buccal nerve or parotid salivary duct can occur.[12]

Techniques for exodontia *per os* were originally described by Merillat[13] and have been recently reviewed and found to be successful in both conscious sedated and anesthetized horses. Since 1995, extraction *per os* has been the author's (WHT) technique of choice for dental removal, and has been associated with a considerably reduced incidence of complications than those associated with repulsion.[7,14] The outcome of oral extraction is considerably hindered by previous unsuccessful attempts at dental removal or where there is a fracture involving the clinical crown.

Endodontic treatment of advanced apical abscesses requires careful case selection, specialized equipment and a precise technique so that its application for dental conservation is currently limited. More detailed understanding of the etiopathogenesis of apical infections combined with more sensitive diagnostic techniques such as scintigraphy and computed tomography may enable improvements in endodontic techniques leading to an improved success in the future.[15–17]

Indications for oral extraction

Apical abscesses, severe diastemata, or dental displacements with food impaction and gingivitis are progressive conditions and conservative treatments are usually disappointing, possibly because many are chronic by nature before veterinary attention is sought. Mandibular apical abscesses and rostral (Triadan 106–108, 206–208) maxillary cheek teeth infections, typically present with a firm, non-painful swelling over the affected cheek tooth apex, possibly associated with a discharging tract. Apical infections involving the caudal mandibular cheek teeth (310, 311, 410, and 411) may also present with possibly painful swellings involving the masseteric muscles or pharyngeal and submandibular areas which may be painful especially if there is pressure deep to the masseter muscles. Apical abscesses of the more caudal (108–111, 208–211) maxillary cheek teeth commonly discharge into the rostral or caudal maxillary sinuses and cause a secondary suppurative empyema.

A careful oral examination with a full mouth speculum (gag) will usually identify the presence of food pocketing due to diastemata associated with a displaced or supernumerary tooth, as this leads to periodontitis, and a digital examination, or exploration with a dental pick or molar forceps, can reveal loose or fractured cheek teeth, or the presence of putrefying impacted food between the cheek teeth. Careful inspection using a dental mirror or rigid endoscope facilitates the identification of obscure diastema. The occlusal surface of a cheek tooth with an apical abscess commonly shows no gross defects, although in some cases close inspection of the occlusal surfaces will reveal pits or carious defects of the infundibula, which can communicate with the dental pulp chambers. Apical abscesses occur most commonly in horses with a median age of 6 years for mandibular and rostral maxillary cheek teeth, and median age of 8 years for caudal maxillary cheek teeth, with the second and third mandibular (307, 308, 407, 408) and the second and fourth maxillary cheek teeth (107, 109, 207, 209) most commonly being infected.[7] Primary periodontal disease is rare, but in horses with dental displacements and diastema, the overwhelming majority show periodontal disease (median age 7 years).[7]

The purpose of all extraction procedures is to separate the tooth from the jaw, and to achieve this it is necessary to break down the structure that attaches the tooth to the alveolus, i.e. the periodontal ligament. In horses with periodontitis, the dense collagen of the periodontal ligament between the tooth and the lamina dura denta of the alveolus where the periodontal attachments are weakened, and in aged horses, in which the reserve crowns are shorter, dental extraction *per os* can be easily performed. O'Connor[8] reported 'to extract a molar tooth from a sound alveolus in a young horse is almost an impossible task.' However, with the advent of sedative-analgesic techniques and appropriate instrumentation this is no longer true. Extraction of cheek teeth *per os* is greatly more difficult in younger horses, especially in those with apical abscesses in which there is no gross periodontal destruction, but using a patient, logical and carefully planned approach it can be achieved, minimizing the potential for iatrogenic damage.

Ancillary diagnostic techniques

A comprehensive series of radiographs should be made for those horses with mandibular or maxillary swellings or paranasal sinusitis, possibly associated with dental apical infection. Straight lateral, lateral–30°-dorsolateral, and dorsoventral projections constitute the standard projections for radiography of the maxillary dental arcades, with straight lateral, lateral–45°-ventrolateral and dorsoventral projections for the mandibular teeth.[14] In addition, open-mouthed oblique views are extremely useful for inspection of the coronal aspects of the cheek teeth[18] in horses with displaced diastematous, fractured or supernumerary teeth. The use of blunt metal probes introduced into discharging tracts is helpful to provide contrast to identify the tooth involved in a periapical abscess. The limitations of radiographs in equine cheek teeth disorders have been described and identification

of the presence and position of a diseased tooth may be assisted by other ancillary modalities such as gamma scintigraphy,[19] MRI or computed tomography, although the latter are currently available in a very limited number of centers. Gamma scintigraphy appears to be more sensitive than radiography for detecting early dental apical and alveolar inflammation.[18] Despite the use of these ancillary techniques, the identification of a diseased cheek tooth in horses with sinusitis can be frustrating. Inspection of the suspect dental apex via an exploratory trephine or bone flap osteotomy is ultimately necessary in some cases. Dental removal should never be undertaken where doubt remains as to whether the tooth is diseased.[20]

Restraint

Extraction *per os* is considerably less traumatic than repulsion or extraction by buccotomy, and most horses tolerate the procedure under standing chemical restraint, but general anesthesia is necessary in a small proportion of nervous or fractious horses.

The costs and risks associated with equine general anesthesia mean that the ability to extract cheek teeth in conscious sedated horses offers considerable advantages. Even when attempted extraction proves unsuccessful in sedated horses, extraction *per os* performed under general anesthesia has advantages over other techniques in view of the reduced level of postoperative care and lower incidence of complications. In those horses where the initial attempt at extraction *per os* is unsuccessful, subsequent repulsion, or dental removal by buccotomy, is greatly facilitated by the weakening of some of the periodontal attachments, and thus the trauma associated with tooth repulsion is reduced.

Case selection

Extraction *per os* is currently the technique of choice for the majority of horses. Extraction of the caudal teeth (Triadan 110, 111, 210, 211, 310, 311, 410, 411) is technically more difficult because the limited range of opening of the equine temporomandibular joint frequently impairs accurate instrument placement, and the opposing row of teeth can hinder withdrawal of teeth with long reserve crowns. Oral extraction of fractured cheek teeth is sometimes possible if there is a sufficiently large parent fragment, but many fractured teeth do not need to be extracted, especially where the pulp cavities are not exposed. Typically these teeth, which are more commonly in the maxillary arcades,[7] have a parasagittal lateral slab fragment, which does not involve the pulp, and which can be removed without difficulty, and the larger non-displaced stable fragment does not require removal. Mandibular cheek teeth, although less commonly fractured, typically fracture along a line connecting two pulp cavities, and require removal. However, oral extraction of teeth with severe caries usually results in fragmentation of the tooth, which may necessitate subsequent removal of dental sequestra at a later stage. In horses with maxillary apical abscesses with secondary sinusitis, dental extraction *per os* is possible, but in addition, surgical irrigation and drainage of the contaminated sinus cavities is often necessary. In some chronic cases removal by repulsion followed by sinus curettage and lavage is indicated, particularly when inspissated pus is present. Oral extraction should not be attempted in conscious horses of unsuitable temperament, where the safety of the horse or personnel may be at risk. Dental extraction is contraindicated wherever doubt remains over the etiology of the sinusitis or where identification of the diseased apex is unclear.[19]

Extraction *per os* in the standing horse is best achieved with the horse restrained in stocks, with the head supported on a rigid head collar or head-stand (Fig. 18.14). Cheek tooth extraction constitutes a major surgical procedure, especially in younger horses and effective sedation and good analgesia are prerequisites. An alpha-2 agonist, such as romifidine, detomidine or xylazine in combination with opioid analgesics such as butorphanol or morphine provides an effective combination of restraint and analgesia, and the choice of drug protocol depends on the environment, temperament of the animal, and individual preference. Desensitization of the mandibular alveolar nerve at its entry into the caudal mandibular foramen (Fig. 18.15) may additionally improve analgesia of the mandibular teeth.[21]

Extraction *per os* under general anesthesia

Extraction *per os* necessitates good access to the oral cavity involving the use of bulky instrumentation in a limited space. When extraction is to be performed under general anesthesia,



Figure 18.14. Good restraint and oral exposure to perform oral dental extraction in the conscious patient is achieved with the horse restrained in stocks with the head supported in a dental head collar.



Figure 18.15. Desensitization of the mandibular nerve at the level of the mandibular alveolar foramen provides additional analgesia for mandibular dental extractions.

a narrow-diameter, cuffed endotracheal or nasotracheal tube should be used to prevent inadvertent inhalation of exudates, while still enabling sufficient remaining space to perform the extraction. The horse is placed in lateral recumbency with the affected tooth uppermost.

Technique for oral extraction

The techniques described by O'Connor[8] and Guard[22] have been modified. Antibiotics are routinely administered preoperatively. A bright head-light is necessary for accurate instrument placement. The gag (speculum) is inserted and opened sufficiently wide to allow digital palpation to clearly identify the tooth to be removed. After visual and digital confirmation of the tooth to be extracted, the gingiva on the buccal and palatal or lingual aspects of the affected tooth are then elevated from the tooth using a flat-bladed dental pick or small periodontal elevator (Fig. 18.16). Molar separators are placed rostral and then caudal to the affected tooth to loosen the rostral and caudal periodontal attachments (Figs 18.17 and 18.18). For extraction of a second cheek tooth, the molar separators should not be forced between the first and second cheek teeth because of the high risk of inadvertent loosening of the healthy first cheek tooth (Triadan 106, 206, 306, 406), nor between the caudal two teeth for similar reasons. Aggressive use of the molar separators is unnecessary and also increases the risk of irreversible damage to a healthy tooth by loosening or fracture.

Once the tooth has been separated from its gingival attachments the molar extractors are placed on the tooth (Fig. 18.19). Molar extractors are available in different sizes and no single instrument is perfect for every tooth (Fig. 18.20). Mandibular cheek teeth are narrower than their maxillary counterparts and therefore will require an instrument with

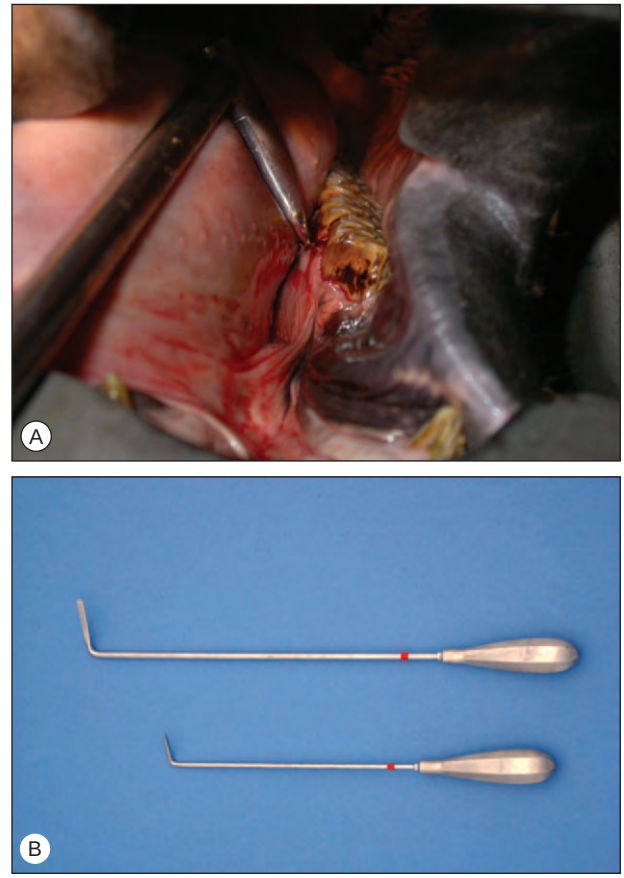


Figure 18.16. Flat-bladed periodontal elevators are used gently to elevate the gingival and periodontal attachments from the buccal and lingual aspects of the tooth.

a narrower space between the jaws when the handles are closed (Fig. 18.21). Maxillary teeth will usually require a wider jawed instrument. Good instrument–tooth contact is essential, and instruments with toothed or knurled jaws are preferable. A visual inspection, to ensure that the correct tooth is grasped, and that the instrument jaws do not extend



Figure 18.17. Molar separators with varied head designs achieve different degrees of separation.



Figure 18.18. The molar separators (Fig. 18.17) are placed in the interdentium rostral and caudal to the affected tooth and cautiously closed to stretch the mesial and distal periodontal attachments (Fig. 18.18).

onto the adjacent tooth, is essential. Provided that no repositioning is necessary the extractor handles are then fixed using the locking mechanism, a rubber bandage or adhesive tape.

The extractors are oscillated with slow, low-amplitude movements in the horizontal plane only, around an axis through the center of the sagittal axis of the cheek tooth (Fig. 18.22). A visual inspection during the first few oscillations should be performed to ensure that the extractor has



Figure 18.19. The molar extractors are placed on the tooth to be extracted attempting to gain maximum contact between forceps and tooth.



Figure 18.20. Many molar extractors are available with different jaws and locking mechanisms.

maintained the grip on the tooth and that the tooth is moving slightly. Incorrect or loose placement of the molar extractors can result in abrasion of the clinical crown, which becomes rounded and is then impossible to grip at subsequent attempts. Twisting movement along the axis of the extractor handles can result in fracturing of the clinical crown and should be avoided. The amplitude of the oscillations is increased *slightly* as the tooth becomes loose, but excessive force or attempts to perform large oscillations should also be avoided, because they can result in shearing of the crown before periodontal attachments are loosened. When the periodontal attachments are sufficiently disrupted, a distinctive 'squelching' sound can be heard, and the resistance to oscillation of the extractor decreases. This is frequently accompanied by fresh foamy hemorrhage around the gingival margins (Fig. 18.23). In addition to disrupting the periodontal membrane, it has been suggested that the loosening oscillations contribute to expansion of the alveolus, also facilitating extraction (J Easley, personal communication). Several hundred oscillations, possibly taking more than 2 hours, may be necessary before the tooth is



Figure 18.21. Different jaw widths are necessary to enable a good contact with both mandibular and maxillary cheek teeth.



Figure 18.22. The handles of the molar extractors, once locked in position, are oscillated in small movements in a horizontal plane at 90° to the long axis of the tooth.

sufficiently loose to withdraw. Some authors recommend postponing the extraction until the following day when alveolar hemorrhage may have contributed to further loosening of the tooth.

A dental fulcrum (Fig. 18.24) appropriate to the extractors is advanced to lie between the forceps and the cheek tooth rostral to the affected tooth (Fig. 18.25). The mechanical advantage is maximized by advancing the fulcrum as far as possible along the row of cheek teeth. Firm pressure is applied to the handles, while keeping the molar extractors

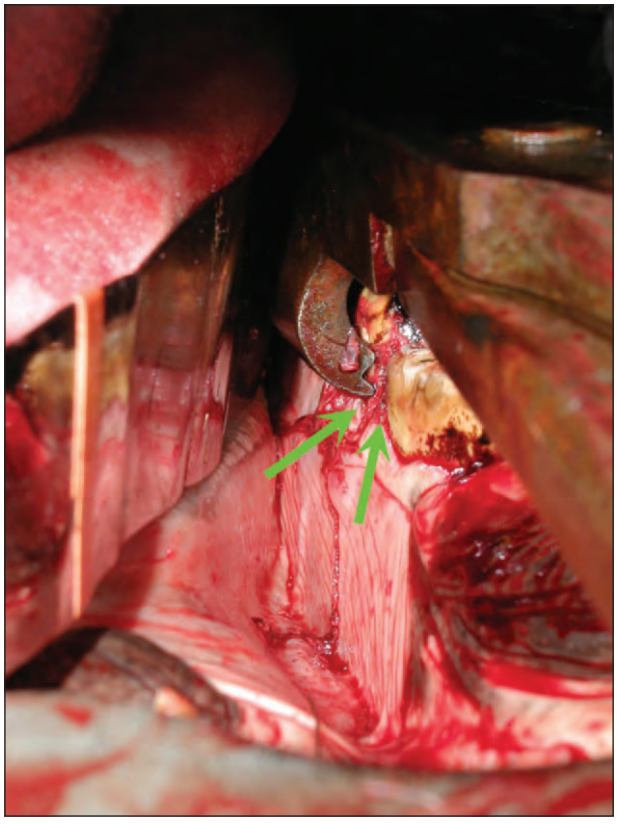


Figure 18.23. Loosening of the periodontal attachments is accompanied by fresh hemorrhage at the gingival margins.



Figure 18.24. A fulcrum is placed between the molar extraction forceps and the cheek tooth rostral to the one to be extracted.

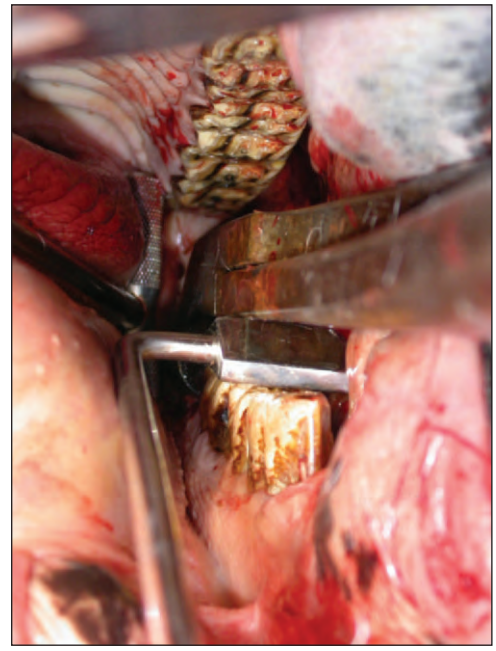


Figure 18.25. Once the fulcrum is positioned carefully, extraction can be performed by exerting a firm force on the handles of the extractors to elevate the tooth from the alveolus.

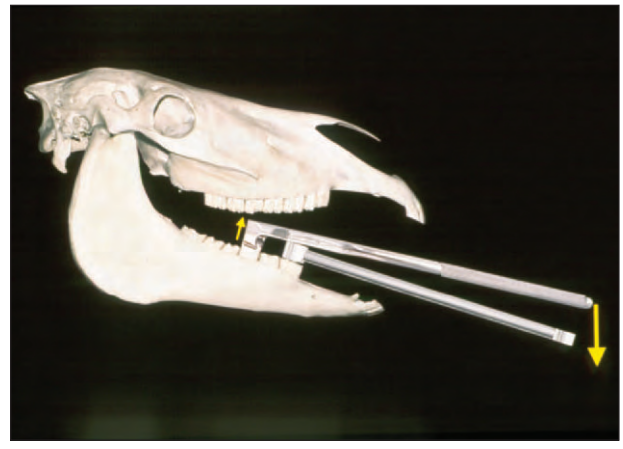


Figure 18.26. A force is exerted on the extractors in a vertical direction to attempt to move the tooth orad from the alveolus.

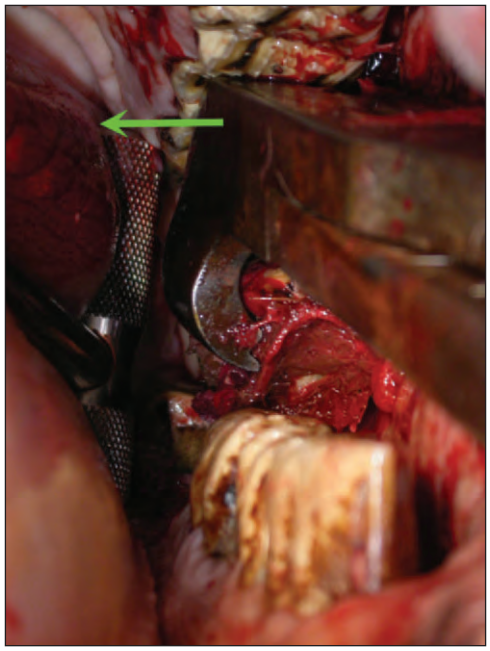


Figure 18.27. When extracting cheek teeth in young animals with extensive apical portions, the extracted clinical crown may need directing palatally (arrow) or lingually to clear the opposing row of teeth.

firmly gripped on the affected tooth, to lever the tooth over the fulcrum and extract it in a straight line parallel with its long axis (Fig. 18.26). Once the tooth has been partially extracted, it may be necessary to release the extractors and replace in a more apical position to release the remaining part of the tooth. Axial twisting of the extraction forceps, along their length, should be avoided until the tooth has been totally freed from periodontal attachments. However, once the tooth is partially displaced, it may be necessary to direct the clinical crown medially to allow extraction of the apical portion without obstruction from the opposing row of cheek teeth (Fig. 18.27). Loosening of the caudal (fifth and sixth) cheek teeth in young (<7 years) horses can be particularly frustrating, possibly due to their obliquely, caudally



Figure 18.28. Looped curettes are used for curetting dental and alveolar fragments from the alveolus prior to insertion of the impression compound.



Figure 18.29. Dental impression compound can be used to prevent food contamination of the alveolus until granulation occurs. This will normally be ejected spontaneously during mastication.

angled reserve crowns. It may be necessary to section them to allow their total extraction from the deep alveoli, although this must be done with care to prevent the apical portion from returning deep into the alveolus after it is sectioned, whence it may be difficult to retrieve.

Once extracted from the alveolus, the tooth is withdrawn from the mouth and inspected for integrity, paying special attention to the apical area. The alveolus should then be carefully palpated digitally for the presence of remaining dental or alveolar bone fragments. If remnants remain, the alveolus is carefully curetted (Fig. 18.28) until the alveolus is smooth to digital palpation, and no dental fragments can be detected. Individual roots may fracture during an extraction, but these do not appear to require repulsion in all horses, unless infected dental or alveolar remnants remain. Post-extraction radiographs should be taken if the tooth fractured during extraction and there is suspicion of remaining dental fragments.

In mandibular or rostral maxillary cheek tooth extractions (with no associated external sinus tracts) the alveolus may be temporarily loosely protected with dental wax, impression compound (Fig. 18.29) or a gauze swab soaked in an antibacterial solution (or metronidazole paste). The packing will be gradually extruded as the alveolus fills with organizing granulation tissue. Care must be taken to avoid sealing infected or necrotic fragments into the alveolus.

In horses with dental disease associated with an external sinus tract or secondary paranasal sinusitis, the alveolus may require sealing from the maxillary sinus to prevent development of an oroantral fistula. In such cases treatment of the sinusitis by surgical debridement or irrigation via a lavage catheter may be necessary.

The dental alveolus can be protected using orthopedic polymethyl methacrylate bone cement (Palacos-R Glaxo-Smith-Kline) (Fig. 18.30) or dental impression compound (Kerr UK Ltd, Peterborough).[23] The alveolus should be cleaned and dried with gauze swabs before prosthesis placement.

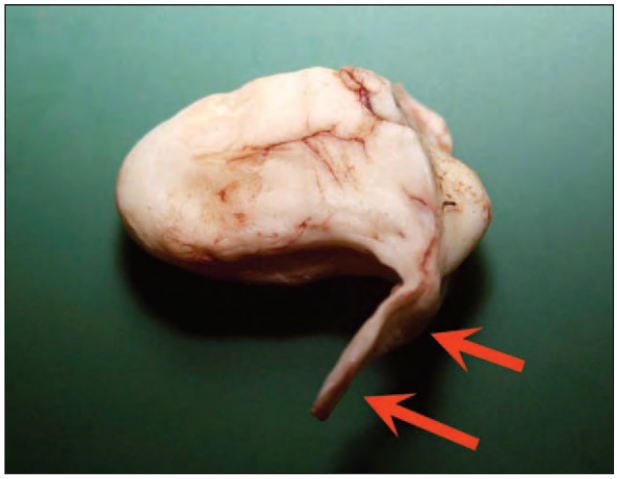


Figure 18.30. Bone cement can be used for a more permanent alveolar implant. Care must be taken when molding it (arrows) to avoid excessive flares, which may cause discomfort during mastication and can also facilitate premature removal.

The methyl methacrylate prosthesis is molded to approximately one-third of the length of the alveolar cavity and placed while still malleable and pressed into the alveolus to lie flush with the gingival margins. Methacrylate protruding into the gingival margins can result in a flange, which facilitates removal of the implant by the horse's tongue. Excessively long prostheses appear to result in delayed alveolar healing.

Aftercare

Minimal aftercare is necessary after oral extraction. However, non-steroidal anti-inflammatory drugs are suitable to provide analgesia for 24–72 hours. A soft or soaked diet can be fed for a few days postoperatively. Sinus lavage for several days may be necessary in those cases with an associated sinusitis. A digital inspection of the alveolus 12–14 days post extraction for any remaining dental fragments is beneficial in those cases where the tooth was extracted in more than one fragment.

Complications

Although complications are rarer after dental extraction *per os* than after repulsion[7] (only two of the first 49 cases of oral extraction performed at the University of Edinburgh[14] showed complications), serious sequelae can arise. Dental extraction can be difficult and fracture of the affected tooth, with retention of the apical portion, can occur, necessitating repulsion of its apical portion (Fig. 18.31). Dental fracture (Fig. 18.32) frequently results in pulpal exposure and contamination from the oral cavity, which can lead in turn to apical infection. Incorrect instrument placement may result in iatrogenic damage to healthy adjacent teeth.[7] Excessive use of force, while loosening the tooth, or when removing it from the alveolus, can occasionally result in mandibular fracture, especially when extracting caudal mandibular teeth in young horses. Ill-fitting bone cement prostheses can be displaced prematurely, resulting in food impaction in the cavity, and the formation of an oroantral fistula following extraction of the caudal maxillary cheek teeth (Triadan 108–111, 208–211). In some situations the dental alveolar plugs may become loose, allowing food into the alveolus, and are associated with discomfort. Removal and replacement of the implant is indicated, and this can be facilitated by leaving a slight flare on the gingival margin, when placing the plug. After dental removal, the opposing tooth will grow faster with reduced attrition from mastication, and frequent (e.g. biannual) dental rasping will be necessary to avoid a 'step-mouth' occurring. Following dental removal, some dental drift will occur in the affected arcade to close the gap resulting from the extraction, which can in the long-term result in diastema between other teeth in the same row. Continuing nasal discharges, in cases with secondary sinusitis, indicate the presence of remaining dental sequestra, or inspissated pus or ingesta in the maxillary sinuses, requiring further investigation and treatment.

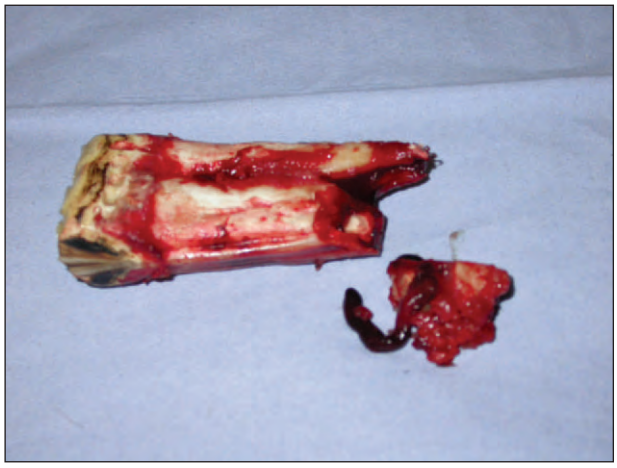


Figure 18.31. Excessive force during oral extraction may lead to fracture of an individual root and subsequently a second procedure may be necessary to repulse any remaining fragments.

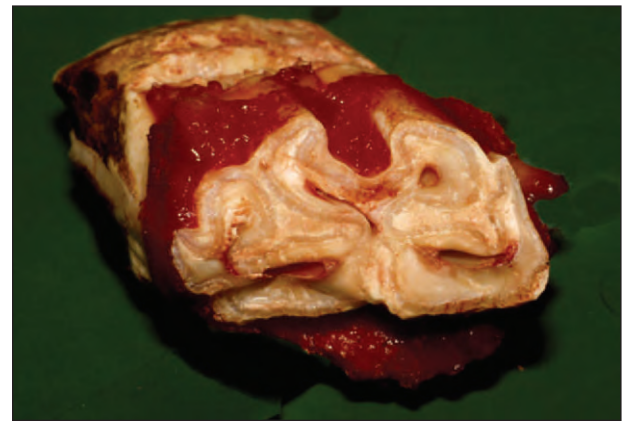


Figure 18.32. Mandibular cheek tooth, which fractured transversely during loosening due to excessively large movements with the extraction forceps.

Conclusion

Extraction of cheek teeth *per os* is a viable alternative to other techniques for dental removal in selected cases. The ability to perform extraction *per os* in standing sedated horses, in combination with the relatively low incidence of complications, offers considerable advantages over other techniques. Cases with lesions affecting the rostral three cheek teeth in horses more than 8 years old are most easily extracted *per os.* However, extraction *per os* is possible with caudal cheek teeth and of cheek teeth in young horses and each case should be individually evaluated. Dental apical infections associated with secondary paranasal sinusitis have an increased risk of complications after treatment and dental removal under general anesthesia may be indicated in such cases, if debridement of the sinuses via a bone flap is required, or if the animal's temperament renders a standing procedure unsafe. Complications associated with dental removal can occur even when performed by experienced clinicians with a full range of instruments, and veterinarians without the appropriate facilities are ill-advised to attempt it except in older horses or where periodontal disease is severe. Clients should be advised of the risk of complications and possibly large bills before commencing any extractions, although the technique compares favorably with repulsion, and extraction via buccotomy.

PART 2: Surgical extraction of equine molars and premolars (cheek teeth)

Introduction

It has already been affirmed that the extraction of an equine molar or premolar tooth is a formidable undertaking with many potential complications in the intra-operative and post-operative periods. In addition there are long-term sequelae for the horse's oral conformation. Nevertheless, the technique of extraction by repulsion has been practiced more or less unchanged since general anesthetics were first administered to horses 150 years ago. The technique necessitates an apical approach to the diseased tooth, which is then driven out into the mouth with a hammer and punch. Buccotomy extraction comprises a horizontal surgical approach into the mouth through the cheek followed by the removal of the lateral bone plate supporting the alveolus. The tooth is sectioned longitudinally so that it can be withdrawn laterally piecemeal. Vertical alveolar osteotomy is similar but uses a vertical incision and is particularly applicable to the fourth and fifth mandibular cheek teeth (CTs 4 and 5 – 309, 310, 409 and 410).

In order for extraction procedures to be successful it is essential accurately to identify which tooth, if any, is diseased and then to remove the *right tooth,* the *whole tooth,* and to damage *nothing but the tooth*.[20] Evidence that a tooth requires extraction is obtained from the external signs, detailed oral examination and imaging modalities such as radiography, MRI and computed tomography. In view of the repercussions for the horse whenever a tooth is lost, the evidence must be convincing that extraction is necessary before proceeding to perform such major surgery. Incomplete extraction with dental sequestration will inhibit alveolar healing and provoke on-going suppuration. Inadvertent damage to the important structures adjacent to the clinical crowns and roots of the cheek teeth should be avoided.

There has been a recent resurgence of enthusiasm for the oral extraction of equine teeth by forceps, and this technique has been described above. However, there will inevitably be situations when this method cannot be applied, for example in the face of an impacted tooth where the dental crown is obscured, when the tooth to be extracted has been damaged leaving insufficient crown to secure mechanical purchase, or when the reserve crown is too wide to be drawn between the neighboring teeth. Of course, it is always correct to attempt forceps extraction before resorting to the surgical removal of an equine tooth but in inexpert hands the use of 'spreaders' and forceps can inflict major injuries to the jaws, to the tooth being extracted and to the adjacent teeth. The frequency of maxillary, mandibular and dental fractures has increased in parallel with the renewed popularity of forceps extraction.

The indications for surgical extraction of equine mandibular cheek teeth include: idiopathic apical abscessation;[24] pathological and iatrogenic fracture;[25] impaction;[26] maleruption with secondary periodontitis;[24] and malformations including supernumerary teeth[27] and developmental tumors.[28] For the maxillary arcade the indications are similar but with the addition of periapical abscessation secondary to infundibular caries.[25,27,29]

Most authors in the modern era advise that forceps extraction be attempted before considering surgical extraction of an equine cheek tooth.[14,20,25,27,29–31]

Principles of surgical dental extraction

In human dental practice the days of mass extraction of teeth have declined with the advent of fluoridation of drinking water and of toothpaste. Nevertheless the extraction of teeth (exodontia) remains the most common minor surgical technique in general dental practice.[32] The objective of extraction in any species must be to separate the offending tooth from the head with as little damage as possible to adjacent structures. Shira[33] proposed three basic principles for human dental extraction: (1) obtain adequate access, (2) create an unimpeded pathway for removal of the tooth, and (3) use controlled force. The periodontium is the structure that holds the tooth to the head, and therefore disruption of this tissue is a prerequisite of successful extraction.[32] Hooley and Golden[34] define surgical extractions as procedures

that include a variable combination of tooth sectioning, mucoperiosteal flap reflection and bone removal before withdrawal of the tooth with forceps or elevators. Transalveolar extractions use resection of alveolar bone and the surrounding soft tissues to open a pathway for the tooth to be withdrawn, and the basic steps for such surgery comprise the raising of a flap, removal of bone, tooth division, removal of the tooth intact or in fragments, wound lavage and primary closure.[32]

Extraction by repulsion

Dental repulsion represents the traditional method for the surgical extraction of cheek teeth in horses.[20,27–31,35–37] It comprises an osteotomy directly over the apex of the diseased tooth before driving it into the mouth with a punch. The osteotomy may be made using a trephine providing limited exposure of the tooth apices[20,27,29,30,35–37] or by the creation of a maxillary bone flap to widen the approach to the roots of the upper molar teeth.[27,28,31,38] Post-extraction radiographs are recommended to ensure complete tooth removal.[30,31,39] The oral opening into the vacated alveolus is protected to prevent contamination by ingesta and saliva. The materials recommended for this purpose include: dental wax;[39] medicated gauze rolls secured with umbilical tape;[29,36] gauze rolls replaced once healing is under way by vinyl silicone, dental wax or gutta percha;[9] and dental-base plate wax or Zip paste.[30] Postoperative nursing aims to maintain a healthy alveolus as it fills with granulation tissue so that the plugging eventually falls away and the mucosal defect epithelializes. The repulsion of mandibular CT6 (311 and 411) requires specific consideration because the roots are remote from the ventral border of the mandible and must be approached obliquely either by a vertical incision parallel with the fibers of the masseter muscle[40] or after an 'up-flap' of the masseter muscle.[30]

Buccotomy extraction

The purpose of buccotomy is to make a surgical approach into the mouth using an incision through the cheek.[10] This is followed by removal of the buccal crest of the alveolar bone before dividing of the tooth and withdrawing it laterally. Although the principles advocated by Dimitroulis[31] are broadly followed, a mucosal flap is not normally used to close the defect between the oral cavity and the alveolus. It is apparent that most subsequent authors have abided by the original author's recommendations to use a dorsally based flap sited over the tooth to be extracted[27,30] and they commend the technique for maxillary CTs 3–5 (108, 109, 110, 208, 209 and 210). There have been no published details of the results achieved by buccotomy extractions performed in this manner, although Easley[30] comments that he could see no advantage in the technique when it was applied to the premolar teeth. Kertesz[41] describes horizontal buccotomy to improve access to the oral cavity of the aardvark, bovidae, camelids and rabbits and Lane[42] and Kertesz[41] provided an illustrated account of the horizontal buccotomy approach to premolar extraction from the mandible or maxilla of horses.

Vertical alveolar osteotomy

This technique was described by Lane[43] and the concept is to turn the horizontal buccotomy approach through 90° so that it will lie parallel to the parotid duct and linguofacial artery and vein. However, it may be made rostral or caudal to these structures depending on whether mandibular CT4 or CT5 (309, 310, 409 or 410) is to be extracted. The technique complies with the guidelines of Dimitroulis[32] other than that no attempt is made to close the oral alveolar defect with a mucosal flap.

Surgical techniques for cheek tooth extraction

Preparation for surgery

All surgical extractions are performed under general anesthesia and there are no descriptions of attempts to extract a cheek tooth by repulsion or buccotomy with the patient standing. Suffice it to say that surgical extraction procedures can be prolonged and thought should be given to the prevention of recumbency myopathies by aggressive fluid therapy and the maintenance of blood volume. Also the option to use the nasal route of intubation for anesthetic maintenance should be considered as this will help to clear the oral cavity of at least one obstruction. Once the identity of a diseased tooth has been established and the decision for extraction has been confirmed with the owner, the skin over the surgical site should be cleansed, and the surgeon and one assistant prepare themselves as if for aseptic surgery. However, an unscrubbed assistant is invaluable to provide intra-oral guidance in the positioning of incisions and in the alignment of instrumentation. Facilities for intra-operative radiography are essential, together with the necessary arrangements to maintain surgical cleanliness by the radiographers and their equipment.

Technique of extraction by repulsion

Repulsion is suggested as the surgical option of choice for the maxillary molar teeth – CTs 4–6 (109, 110, 111, 209, 210



Figure 18.33. Mandibular skull specimen showing the traditional sites for osteotomy prior to repulsion. Today repulsion is recommended only for the extraction of 311 and 411.



Figure 18.34. Maxillary skull specimen showing the traditional sites for osteotomy prior to repulsion. Today repulsion is recommended only for the extraction of 109, 209, 110, 210, 111 and 211.

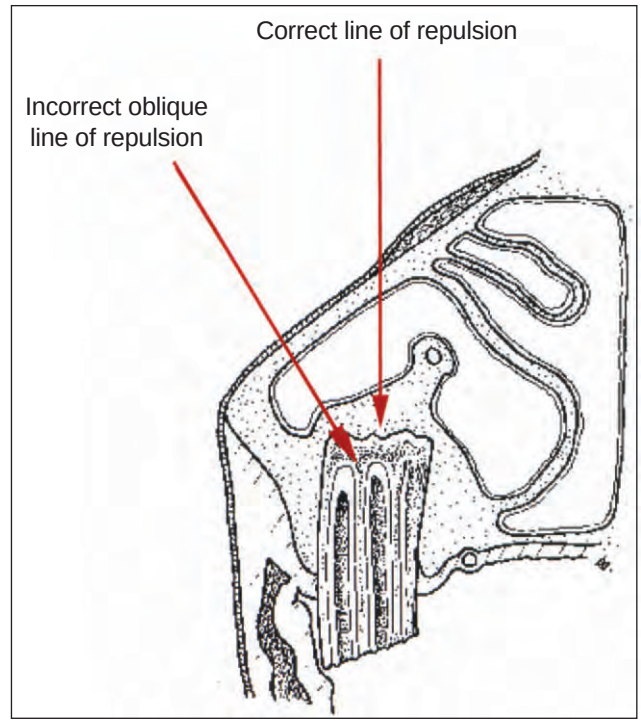


Figure 18.35. Diagram to illustrate the correct positioning of the trephine hole directly over the apex of the tooth to be extracted.

and 211) – and a modified repulsion technique may be used for CT6 in the lower jaw (311 and 411) (Figs 18.33 and 18.34). Although there are protagonists for the use of a lateral maxillary bone flap approach to expose the roots of these teeth[9,27,30,38] the published results are limited and suggest that the results are less satisfactory than for traditional trephination. The position of the trephine hole should be located as directly over the apices as possible (Fig. 18.35) and is estimated on the basis of the tooth involved and the age of the horse. The chosen site must take account of the significant underlying superficial structures (Fig. 18.44B,C), the position of the nasolachrymal duct (Fig. 18.34) and the optimal mechanical line of repulsion (Fig. 18.35). A sterile metallic marker should be placed through the skin and into the bone surface as a means to check by radiography the alignment of the surgery before starting. A rounded skin incision is made, leaving sufficient margin for the introduction of a 19 mm skull trephine (Holborn Surgical Instrument Co.) (Fig. 18.36). The subcutaneous soft tissues are divided and structures such as the angularis occuli artery and vein are retracted to avoid disruption. The periosteum is also divided before a circular osteotomy is made with the trephine. Again, radiographs should be routinely made with a metallic marker directed onto the roots of the diseased tooth to ensure that the line of repulsion is correct (Fig. 18.37) and before the punch is driven onto the roots and irreparable damage is inadvertently inflicted upon a healthy tooth (Fig. 18.46). The unscrubbed assistant palpates the crown of the diseased tooth *per os* to confirm positive vibrations through the tooth once the repulsion process is under way. Movement of the crown is reported together with any indications of maldirection of the punch. The displaced tooth is delivered into the mouth either intact or piecemeal (Figs 18.38, 18.41F). When the more caudal teeth in either arcade are repelled the space afforded between the top and bottom jaws in the fully opened mouth is limited such that medial displacement or fragmentation of the tooth is necessary. The repelled tooth is checked macroscopically for any major missing pieces and, after curettage of the vacated alveolus, radiographs should be taken of the surgical field to look for residual dental fragments, particularly in the root regions. On completion of the extraction the oral defect of the alveolus should be closed and JGL's preference is to use a plug of dental impression compound (Kemco Precision: Associated Dental Products Ltd) (Fig. 18.39A,B). The material becomes malleable when immersed in warm water and the plug can be fashioned to overlie the gingival margins at the buccal and palatal surfaces and yet to lie below the levels of the occlusal surfaces of the teeth on either side. The plug should be shaped to occupy no more than one-third of the alveolus. Fortunately, horses do not eat toffees, and the forces of mastication tend to maintain the plug in position until it is extruded naturally.

Although the description of the repulsion technique above is applicable to CT4 (109 and 209) – the tooth most frequently requiring extraction – specific adjustments are suggested for the remaining teeth. A triple trephination technique (Fig. 18.40) is recommended for CT5 (110 and 210) using three sites: (1) dorsomedial to the medial canthus for the repulsion of the tooth itself, (2) ventrorostral to the medial canthus for guiding the trephine onto the tooth roots and for postoperative nursing including alveolar inspection, and (3) a catheter inlet port into the caudal maxillary sinus at the angle between the bony orbit and the facial crest. The skin incision over the repulsion site was fashioned as a flap for closure with sutures on completion of



Figure 18.36. Drapes removed to show repulsion of 109 via a trephine hole.

the extraction. A similar method is advised for maxillary CT6 (111 and 211) other than that the location for the trephine hole is into the frontal sinus and the punch is passed onto the roots through the frontomaxillary foramen.

The position of the roots of mandibular CT6 are such that an oblique lateral approach is inevitable (Fig. 18.41A–H). However, this necessitates the creation of an extensive upward flap of the masseter muscle from the ventral margin of the angular process of the mandible and away from its insertion into the underlying masseteric fossa. The osteotomy through the lateral cortex for the repulsion punch is inevitably directed onto the tooth roots slightly obliquely. The incision should be partly closed to allow temporary packing with medicated gauze on completion of the surgery and this is withdrawn over a 3-day period, leaving the incision to heal by second intention.

A maxillary facial flap may be used to widen access to the roots of the maxillary molar teeth (Fig. 18.42) or to assist in the removal of a supernumerary CT7 (312 or 412). The incisions should be shaped to allow space for the placement of an irrigation catheter into the caudal maxillary sinus as described above. Otherwise the bone is preserved and replaced; it is held in place by overlying continuous sutures in the periosteum and subcutis (3.5 metric polyglactin 910, Vicryl: Ethicon Ltd) and simple interrupted sutures of monofilament nylon (3.5 metric, Ethilon: Ethicon Ltd) in the skin.

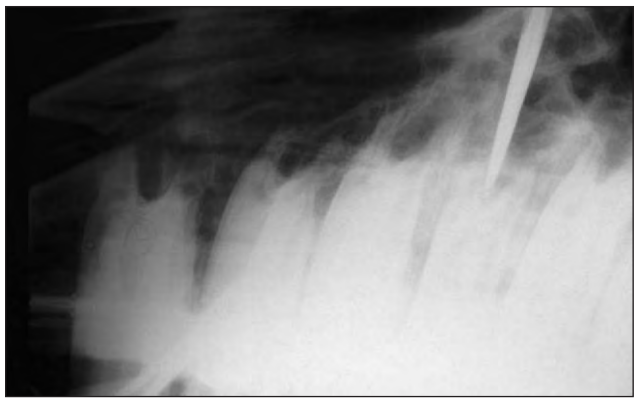


Figure 18.37. Oblique intra-operative radiograph showing a metallic marker used to check the line of repulsion for the extraction of 209.

The strategy to be employed for aftercare requires careful consideration. In those instances where there is evidence of secondary sinus empyema in the form of a putrid nasal discharge, the intercompartmental septum between the rostral (RMS) and caudal maxillary sinuses (CMS) is bluntly broken down via the trephine hole to establish continuity between the two compartments. An irrigation catheter is routinely placed into the CMS for twice-daily lavage of the sinus compartments using very dilute povidone iodine solution (Betadine: Seton Healthcare Group plc) or saline (Figs 18.40, 18.43). The facial trephine hole overlying the alveolus serves as an ideal route through which to monitor the healing of the alveolus and should be prevented from healing by plugging with gauze tampons. These are removed and replaced regularly to inspect the underlying defect with a pen torch. A smooth tube of pink granulation tissue is deemed to be a sign of normal healing. The presence of a malodorous discharge and/or rough projections from the walls of the alveolus are indications for curettage under sedation. The presence of ingesta in the alveolus or the escape of fluid into the mouth during irrigation should be taken as signs that the oral plug has been lost or is loose. The plug can be replaced *per os* with the mouth gagged open but this may require sedation. When these measures fail to provide an improvement further diagnostic radiographs, more careful inspection of the lining of the alveolus and more aggressive curettage may all be needed but it may be necessary to resort to a short repeat general anesthetic. The size of the gauze tampon maintaining the patency of the trephine hole can be progressively reduced to nil but only when the discharges at the nostril have ceased and the lining of the alveolus itself has become healthy.

Owners of all horses where cheek tooth extractions have been performed are advised of the necessity for routine rasping of the occluding tooth in order to prevent malocclusion through dental overgrowth.

Extraction by buccotomy (Fig. 18.44A–L)

The teeth that lend themselves to removal by buccotomy are the premolars in all arcades (Triadan 106, 107, 108, 206, 207, 208, 306, 307, 308, 406, 407 and 408), and the advantage of this method over repulsion lies in the avoidance of iatrogenic fracture of the lateral plate of the maxilla and the lateral and medial cortices of the mandible. On the basis that the clinical crown of the diseased tooth is exposed before it is traumatized in the extraction process, it should not be possible to remove the incorrect tooth or damage an adjacent crown.

A horizontal skin incision centered over the diseased tooth (Fig. 18.44A) is made parallel with the occlusal surfaces of the cheek teeth, but at the level of the dorsal or ventral buccal cleft for maxillary or mandibular extractions respectively. Care is taken to preserve the dorsal and ventral buccal

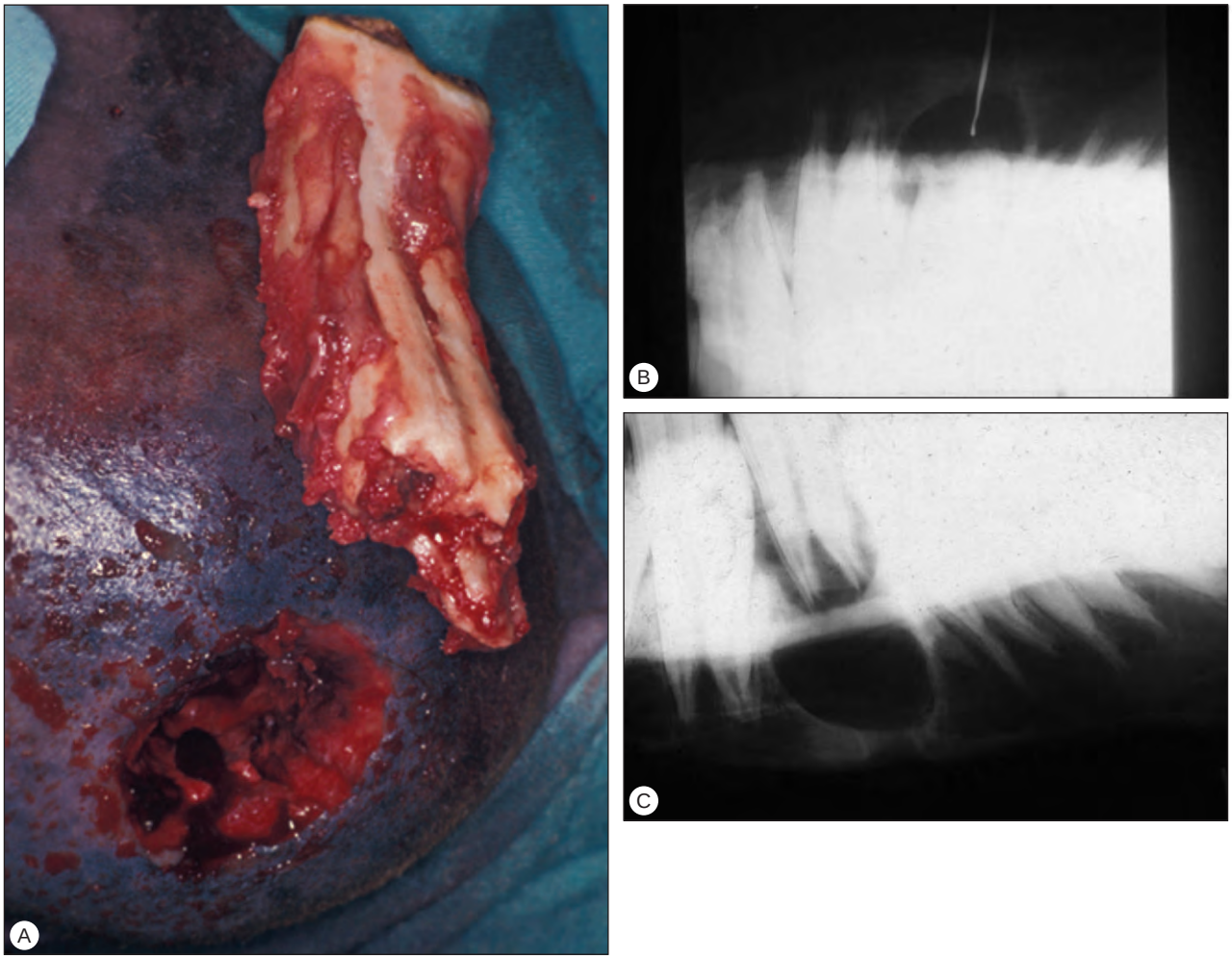


Figure 18.38. (A) Preoperative radiograph of pony with periapical suppuration of 308. – a metallic marker has been placed in a discharging tract at the ventral aspect of the mandible. (B) Extracted 308 – the specimen is checked for missing fragments. (C) Postoperative radiograph confirming complete extraction of 308.

branches of the facial nerve and the parotid duct (Fig. 18.44B) which enters the mouth at the level of the rostral margin of CT3 (108/208). Thus, maxillary buccotomies should be made dorsal to the parotid papilla and the mandibular incisions ventral to it. The buccal nerves can be identified and drawn away from the line of dissection whenever necessary. The deeper dissection negotiates the buccal venous plexus – the labialis communis, labialis maxillaris and labialis mandibularis veins – and the ventral buccal glands before entering the mouth (Fig. 18.44C,D) through the tough oral mucous membrane. The tooth to be removed is identified (Fig. 18.44E) by reference to radiographs and also by counting back from CT1. A gingival flap is raised to expose the lateral cortex of the maxilla or mandible. The plate of bone lying lateral to the diseased tooth is resected either with an oscillating saw, a surgical fissure burr or a chisel to expose the reserve crown and roots of the tooth. In some cases space for extraction can be created by a longitudinal cut in the tooth, again using a burr, otherwise the diseased tooth is split longitudinally, i.e. parallel with the dental arcade, and it is then removed piecemeal with the assistance of curved elevators (Fig. 18.44E,F). Postoperative radiographs are only necessary when there is doubt whether all of the dental tissue has been removed (Fig. 18.38B,C). After removal of the tooth the vacant alveolus (Fig. 18.44G) is packed with ribbon gauze impregnated with bismuth subnitrate and iodoform paste (Lennon Pharmaceuticals) or soaked with betadyne solution, and this is led out through a separate stab incision to the side of the face (Fig. 18.44H) to be withdrawn a few inches at a time over the following 14 days. The oral defect should be protected with a cap of dental impression compound sitting over the gauze packing (Fig. 18.44I), but on occasion the gingival flap may be closed to the palatal mucosa at the medial margin of the alveolus (Fig. 18.44J). The buccotomy incision is closed in three layers of 3.5 metric polyglactin 910 (Vicryl: Ethicon Ltd) starting with the gingival mucosa, and simple interrupted or a continuous interlocking suture of monofilament nylon (3.5 metric, Ethilon: Ethicon Ltd) (Fig. 18.44K,L) for the skin.

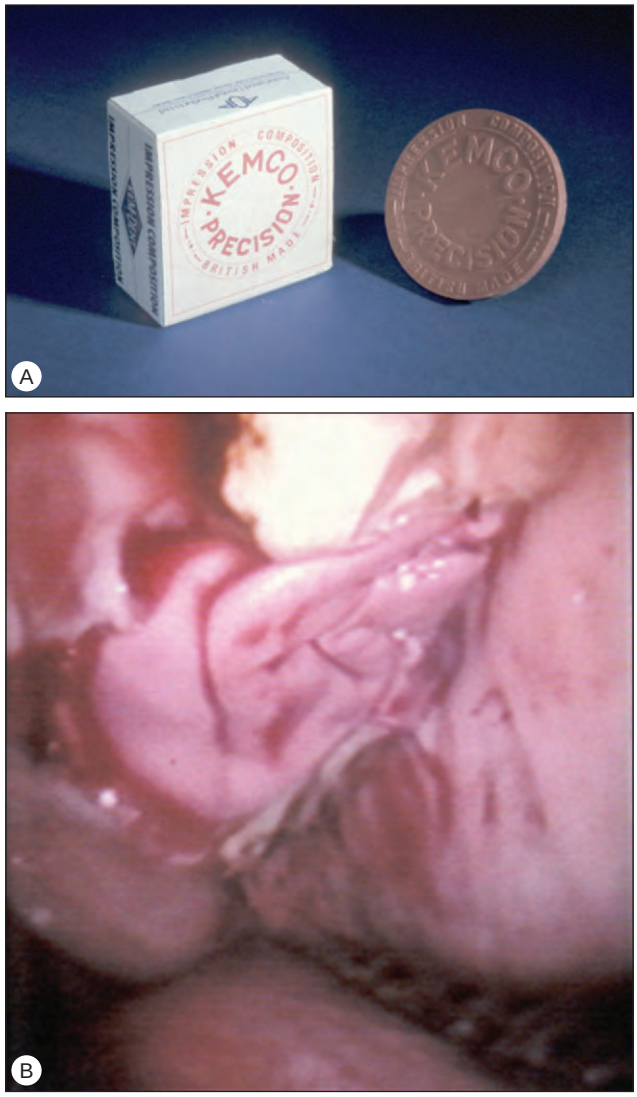


Figure 18.39. (A) Dental impression compound. (B) Dental impression *in situ*, lying below the level of the occlusal plane.



Figure 18.40. Horse following the extraction of 210 by repulsion using the triple trephine technique: (1) sutured incision over trephine hole used for the passage of the repulsion punch, (2) plugged trephine hole used for guidance intra-operatively and monitoring postoperatively, and (3) port for the irrigation catheter.

Extraction by vertical alveolar osteotomy

(Fig. 18.45A–G)

This modification of the buccotomy technique takes account of the position of the linguofacial artery and vein as well as the parotid salivary duct and offers a predictably satisfactory surgical method to extract CTs 4 and 5 from the mandible (309, 310, 409 and 410). The usual indication for the removal of these teeth is malalignment of the clinical crowns with painful periodontitis. Bilateral extractions are often required to curtail the severe oral phase dysphagia which accompanies this disorder. A vertical skin incision is made parallel to the linguofacial artery and vein and the parotid duct (Fig. 18.45A,B). For mandibular CT4 (309 and 409) extractions the incision lies rostral to these structures and for CT5 (310 and 410) it lies caudal to them, but rostral to the masseter muscle. The ventral buccal branch of the facial nerve is more likely to intrude into the dorsal segment of the dissection in CT4 extractions but it can be easily recognized and preserved. The remainder of the dissection onto the lateral cortex of the mandible is uncomplicated by major structures but does require vigorous retraction of the masseter muscle in particular. An unscrubbed assistant is, again, invaluable for confirmation of the line of the incision and of the correct point of entry through the buccal mucosa (Fig. 18.45C). The lateral alveolar wall should not be resected until the diseased tooth has been positively identified. In this manner the vertical osteotomy incisions are made accurately in line with the interproximal spaces at either margin of the tooth. At least two-thirds of the lateral alveolar wall is removed using a chisel or osteotome before attempting removal of the tooth (Fig. 18.45D). Again, curved elevators are ideal to remove residual root tissues. Postoperative radiographs are generally not necessary because there is little doubt when all of the dental tissue has been removed (Fig. 18.45E). The alveolus is packed with medicated ribbon gauze as above which is again led out through a separate stab incision to the side of the face (Fig. 18.45F) to be withdrawn a few inches at a time over the following 14 days.

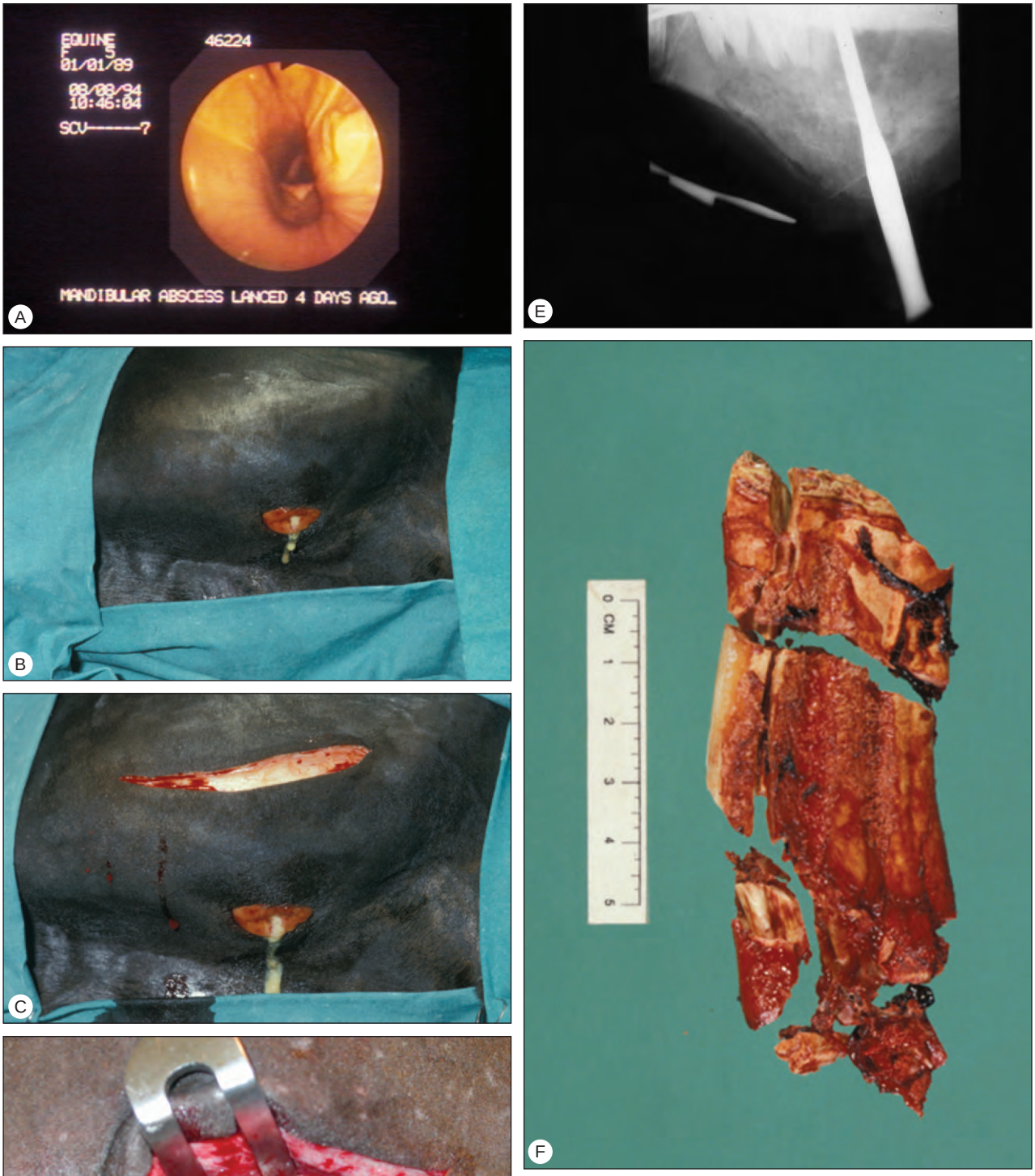


Figure 18.41. Case study of extraction of 311 in a 5-year-old Thoroughbred. (A) Endoscopic view of pharynx distorted by abscessation in parapharyngeal tissues following iatrogenic fracture of 311 during hook removal by an EDT. (B) Drainage of the abscess at the ventral aspect of the horizontal ramus. (C) and (D) Raising the masseteric muscle flap to access the lateral aspect of the mandible over the apices of 311. (E) Oblique radiograph for intra-operative alignment of repulsion punch. (F) Specimen after piecemeal repulsion of 311. Note the absent fragment at the caudal aspect of the crown – removal of this precipitated the abscessation.



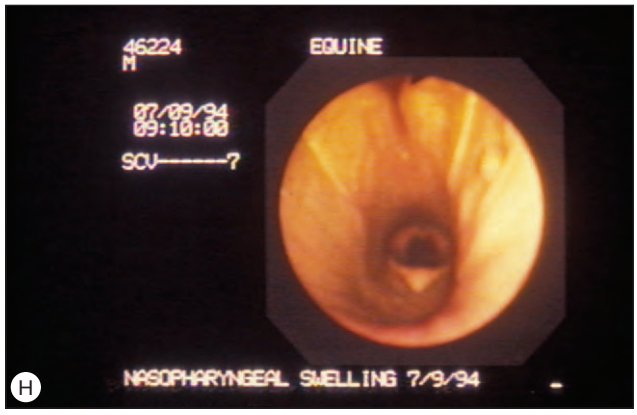


Figure 18.41, cont'd, (G) Healing of the incision by second intention. (H) Postoperative appearance of nasopharynx.

The oral defect is once again protected with a cap of dental impression compound and the mucosal defect is closed with an interrupted layer of 3.5 metric polyglactin 910 (Vicryl: Ethicon Ltd). The soft tissues require closure in two continuous layers of polyglactin and the skin with a continuous interlocking suture of monofilament nylon (3.5 metric, Ethilon: Ethicon Ltd) (Fig. 18.45G).

Complications and prognosis

This author's views concur with the observations reported previously that the extraction of an equine cheek tooth by repulsion is a technique that necessitates prolonged healing,



Figure 18.42. Drapes removed to show repulsion extraction via a maxillary facial flap. Reported evidence suggests that the results of this technique are no better than when a trephine hole is used, possibly worse.

carries a high risk of complication, and not infrequently results in persistence of localized suppuration.[27,30,37,39,43,44] A wide range of complications of repulsion surgery have been reported.[39,43] These include iatrogenic damage to neighboring teeth (Fig. 18.46); traumatic fracture of the mandible; inadvertent disruption of adjacent structures such as the nasolachrymal duct and greater palatine artery; dental sequestration (incomplete extraction) and alveolar sequestration (Fig. 18.47); and oroantral fistula formation. However, many of these can be avoided with careful preparation and an appropriate knowledge of the anatomy of the region. Much has been made in the literature of alveolar sequestration and post-extraction cementoma formation, yet it is hardly surprising that fragments of alveolar lining are shed in the postoperative period. The teeth are subjected to severe mechanical forces during repulsion, which are transferred through the periodontium to the lamina dura of the alveolus. There is also the possibility of direct trauma of the alveolar bone by a maldirected repulsion punch. Monitoring of the healing alveolus should take account of the possibility of sequestrum formation and provides a sound justification for in-patient nursing until it becomes clear that the alveolus is forming a tube of smooth granulation tissue. For a technique which has been in use for almost 150 years, it is surprising that there are few detailed retrospective studies of results from substantial case series of horses subjected to dental repulsion. In one study[43] resolution of signs was ultimately achieved in 92 per cent of maxillary repulsions, but 42 per cent experienced sufficiently major complications to justify at least one further general anesthetic. The common causes of these temporary setbacks include alveolar and dental sequestration and contamination of the healing alveoli by ingesta. The patients continue to show a nasal discharge or persistence of suppuration to the surface of the face until the cause is eliminated. In the 8 per cent that were never resolved the cause of failure was invariably oroantral or orofacial fistula formation.



Figure 18.43. (A) Placement of an irrigation catheter into the caudal maxillary sinus after the removal of 209 by repulsion. The septum between the rostral and caudal sinuses must be removed for irrigation to be effective. (B) Placement of an irrigation catheter after a limited flap has been used to extract 109.

Many previous authors have intimated that oroantral fistula formation is a particularly challenging complication of equine cheek tooth extraction[9,20,27,37,39,44] and also constitutes a serious complication following molar extractions in man.[34] However, the superior access available for surgery within the human oral cavity permits the closure of defects in the floor of the antrum using a mucoperiosteal flap.[34] Equine oral surgery will inevitably be constrained by the poor access afforded when the range of opening by the temporomandibular joint is so restricted and the teeth lie deep within the oral cavity. Thus, the intra-oral mucosal flap techniques which are commonly used in man and small animals to improve access during extraction and for closure of the oral defects which result from surgical extraction are not yet applicable to horses. On the basis that chronic oroantral fistulae after extraction of upper CTs 4–6 arise through recurring contamination from the mouth, one group has suggested translocation of part of the levator nasolabialis muscle to close the defect, albeit still using an acrylic plug on the oral surface.[45] Dixon[37] has concentrated on the materials used to protect the oral aspect of the alveolus and reports very favorable results in the prevention of fistula formation by placing bone cement across the defect.

Osteoplastic facial flaps have been advocated by some authors[9,27,30,38] to aid the extraction of maxillary CTs 4–6 and they unquestionably provide superior exposure of the tooth roots at the time of extraction and should increase the accuracy of the surgery. However, once the osteoplastic flap is closed at the end of surgery, access to monitor the progress of the healing alveolus and to perform minor curettage, for example to remove sequestra, is no longer available unless the flap is reopened.

Those who argue that traditional trephination over the tooth roots still has a place for dental repulsion must accept the need for great accuracy in both diagnosis and surgical alignment unless irreparable damage is to be inflicted on healthy teeth. The time dedicated to pre-, intra- and postoperative radiography is never wasted time when weighed against the possibility of a double dental extraction from the same jaw (Fig. 18.44).

The mechanical requirements of repulsion are such that access must allow the application of the punch not only

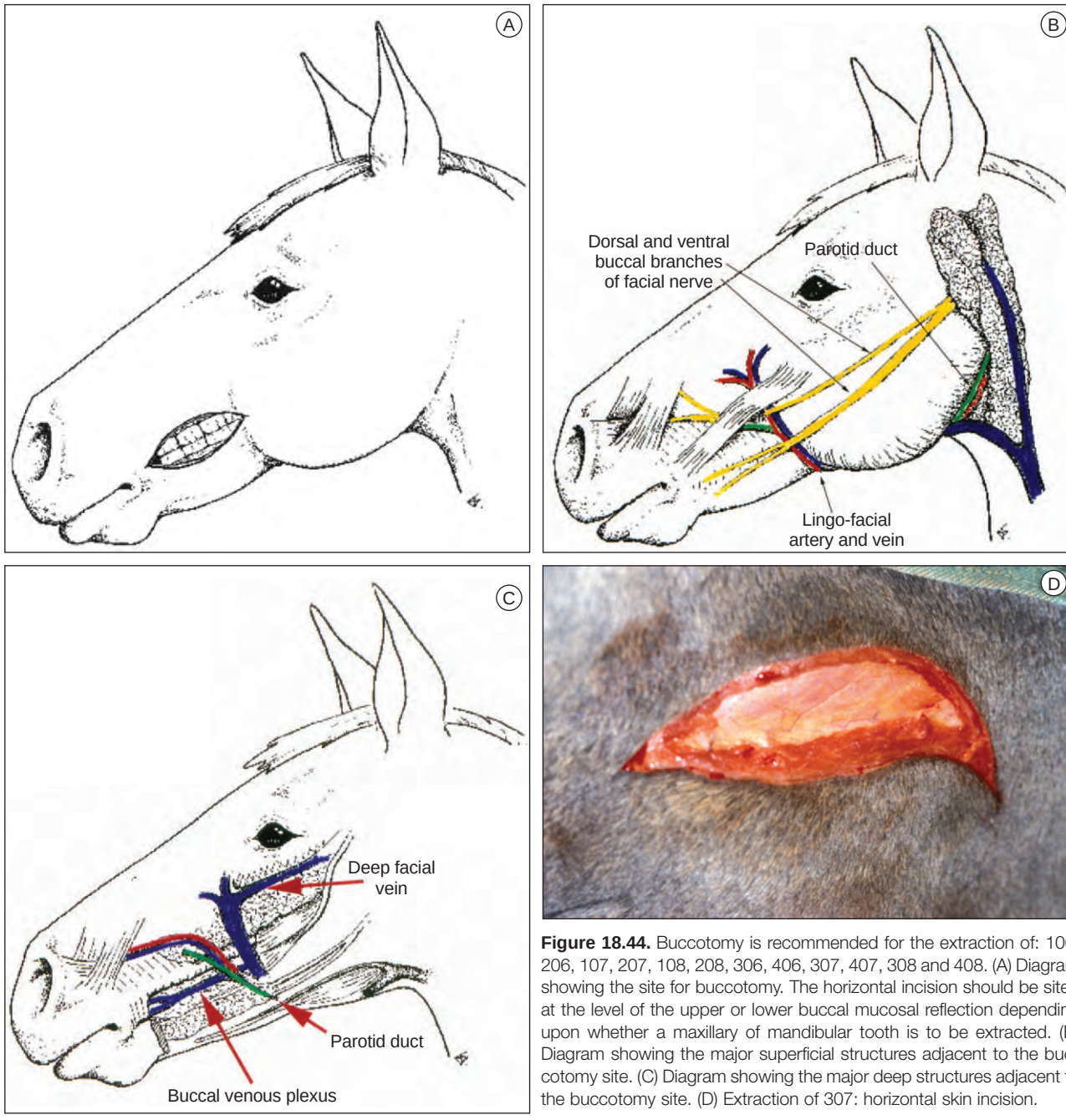


Figure 18.44. Buccotomy is recommended for the extraction of: 106, 206, 107, 207, 108, 208, 306, 406, 307, 407, 308 and 408. (A) Diagram showing the site for buccotomy. The horizontal incision should be sited at the level of the upper or lower buccal mucosal reflection depending upon whether a maxillary of mandibular tooth is to be extracted. (B) Diagram showing the major superficial structures adjacent to the buccotomy site. (C) Diagram showing the major deep structures adjacent to the buccotomy site. (D) Extraction of 307: horizontal skin incision.

directly onto the tooth root but also in line with its long axis (Fig. 18.38). Inadvertent damage to adjacent structures is far more likely to arise when the punch is applied obliquely. In younger horses whose reserve dental crowns are long, the roots of CTs 3 and 4 lie adjacent to the line of the nasolachrymal duct and it is, perhaps, predictable that one horse which sustained disruption of this structure was 3 years old and undergoing repulsion of a maxillary CT3.[43]

The surgical complications associated with buccotomy and its variant, vertical alveolar osteotomy, are much less by comparison. Partial wound dehiscence is occasionally encountered but this can be expected to heal by second intention. However, it is surprising that these incisions, which are contaminated by ingesta from the oral aspect, do not show a greater incidence of delayed healing. Occasional temporary facial nerve deficits are to some extent predictable when the position of the dorsal and ventral buccal branches of facial nerves makes them particularly vulnerable to stretching by retractors during buccotomy. However, the horizontal incisions can be sited accurately between them and physical transection should never occur. Early reports[10,27,30] advocated buccotomy for the extraction of maxillary CT4 (109 and 209) using curved incisions over the diseased tooth, but the dangers which this poses to adjacent structures have

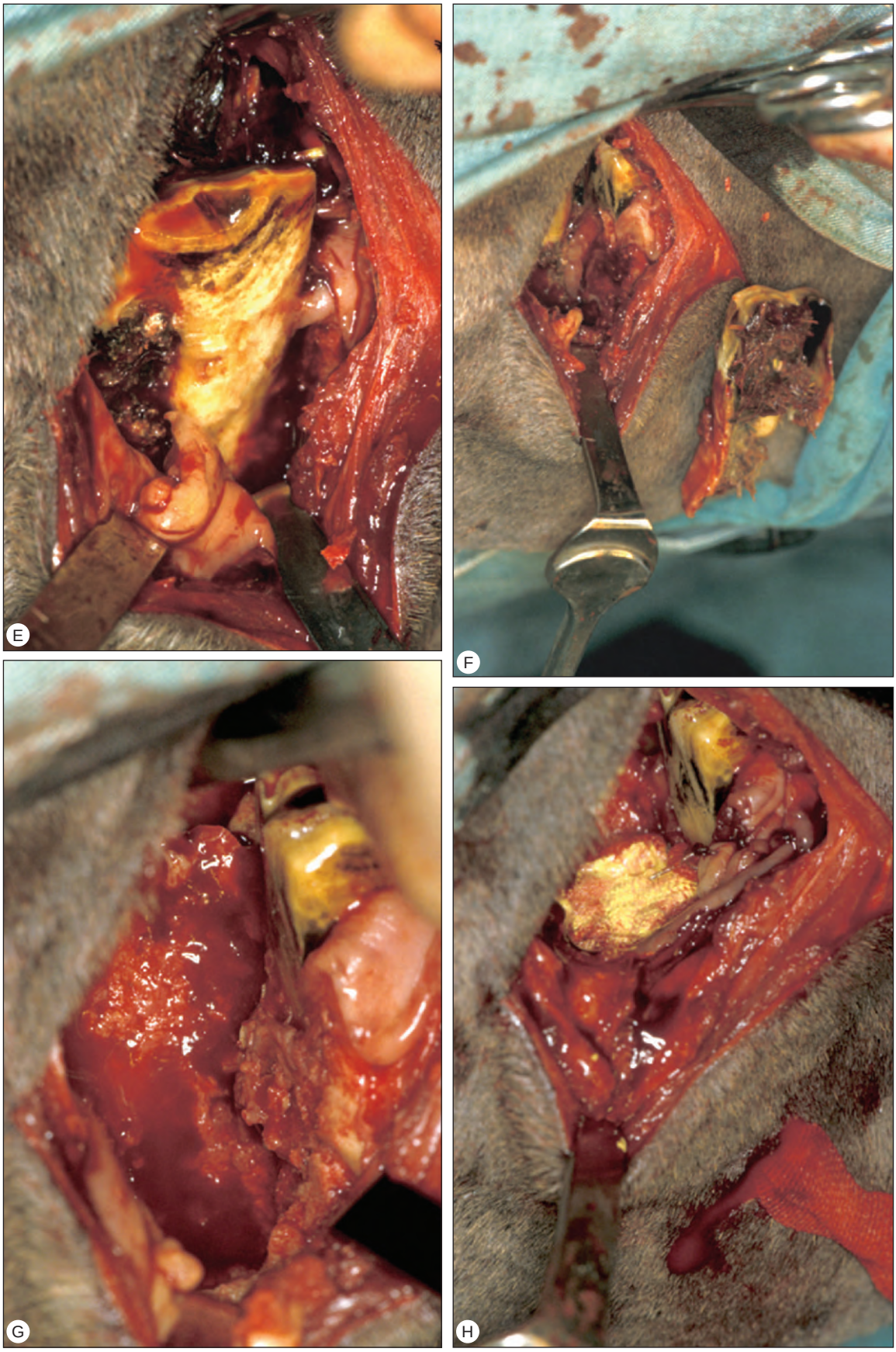
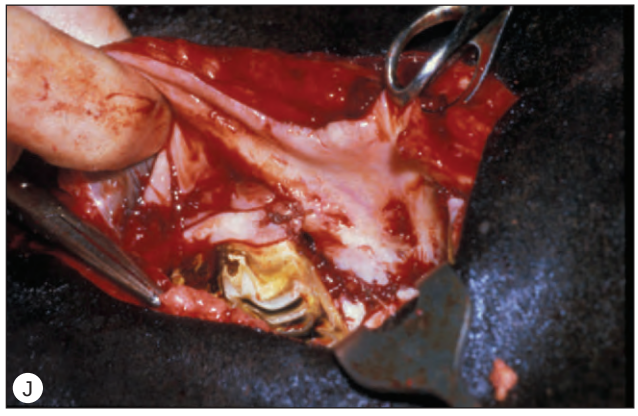


Figure 18.44, cont'd, (E) Exposure of the fragmented 307 (F) Removal of 307 piecemeal. (G) Empty alveolus. (H) Alveolus packed with medicated gauze.





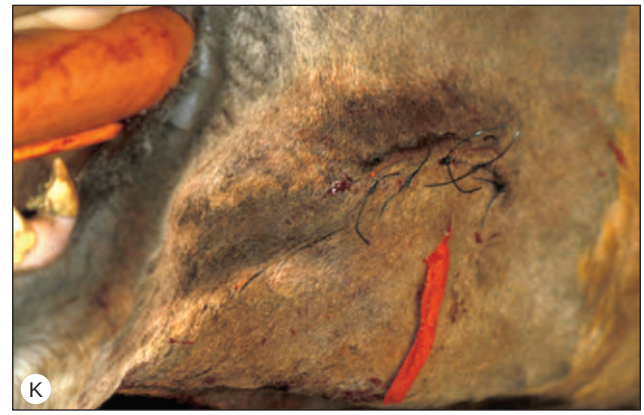




Figure 18.44, cont'd, (I) Impression compound cap placed to protect underlying alveolus. (J) Buccal mucosal flap used to protect alveolus after buccotomy extraction. (K) Incision after closure. Note medicated gauze exited through stab incision. (l) Appearance at 12 days after buccotomy – the medicated gauze has been totally withdrawn but the healing stab incision can still be seen.

probably been the major reason why the principle has not been exploited more extensively. The dissection through the soft tissues over the cheek at this site is hazardous as it necessitates negotiating the insertion of the masseter muscle and the deep facial vessels. The straight horizontal incisions used by Kertesz[41] for captive wild animals proved to be helpful in steering a path between the branches of the facial nerve for the extraction of equine premolar teeth, but bring limitations caudally in the forms of the linguofacial artery and vein, the parotid duct and the masseter muscle.[42]

In the light of the low incidence of complication,[43] buccotomy is commended as the surgical extraction technique of choice for the more rostral teeth of both jaws in the horse and one of the major advantages of the technique is that there is little chance that an incorrect tooth will be extracted or that an adjacent tooth will be damaged. The aftercare is simple and healing by first intention is the norm.

Similarly vertical alveolar osteotomy provides predictably favorable results for the extraction of the rather more challenging teeth – CTs 4 and 5 in the lower jaw (309, 310,

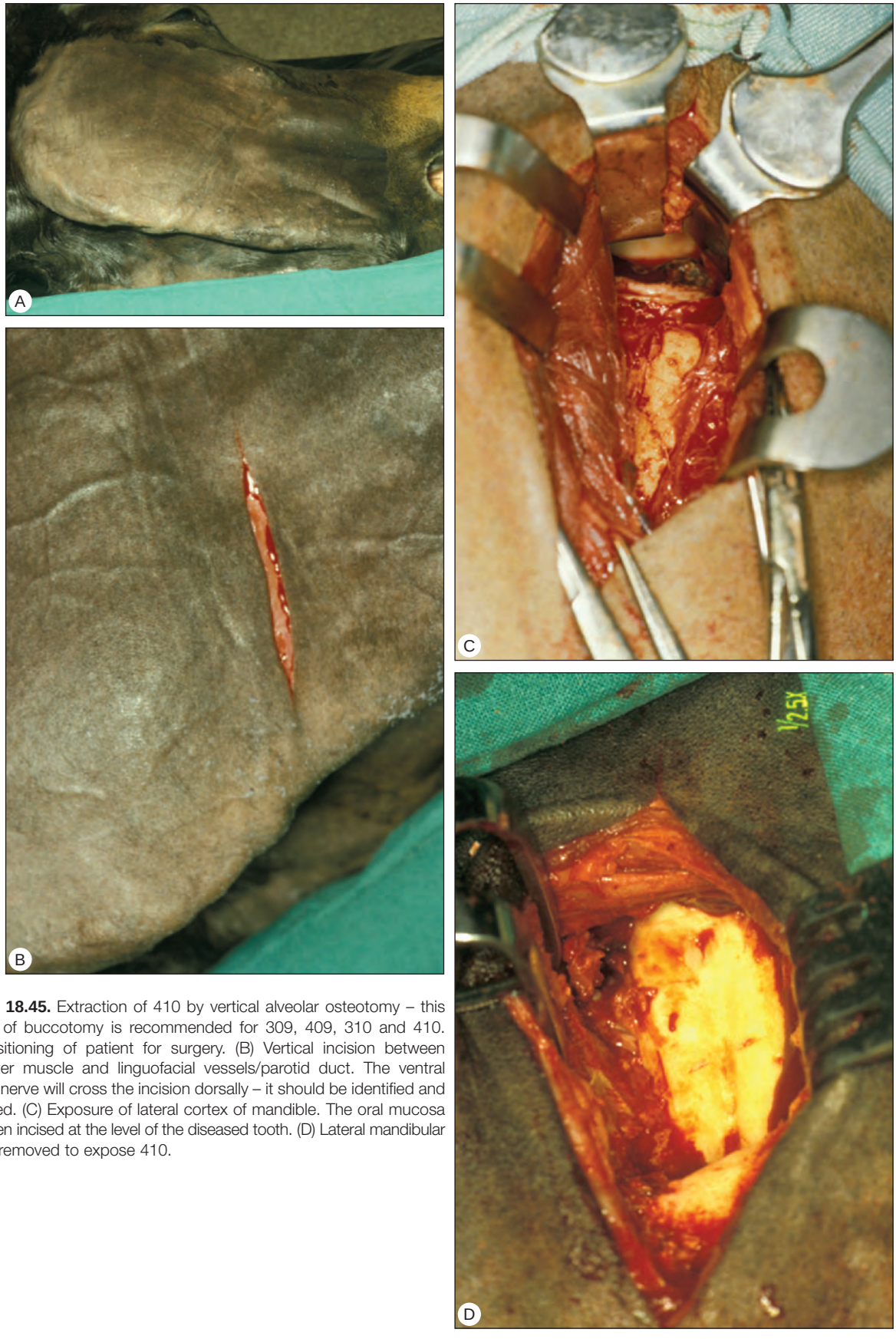
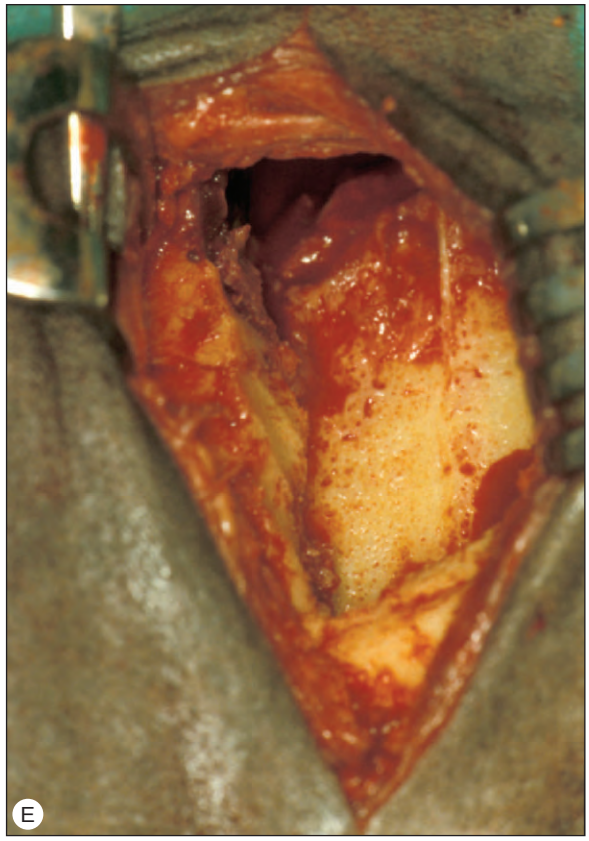


Figure 18.45. Extraction of 410 by vertical alveolar osteotomy – this variant of buccotomy is recommended for 309, 409, 310 and 410. (A) Positioning of patient for surgery. (B) Vertical incision between masseter muscle and linguofacial vessels/parotid duct. The ventral buccal nerve will cross the incision dorsally – it should be identified and retracted. (C) Exposure of lateral cortex of mandible. The oral mucosa has been incised at the level of the diseased tooth. (D) Lateral mandibular cortex removed to expose 410.





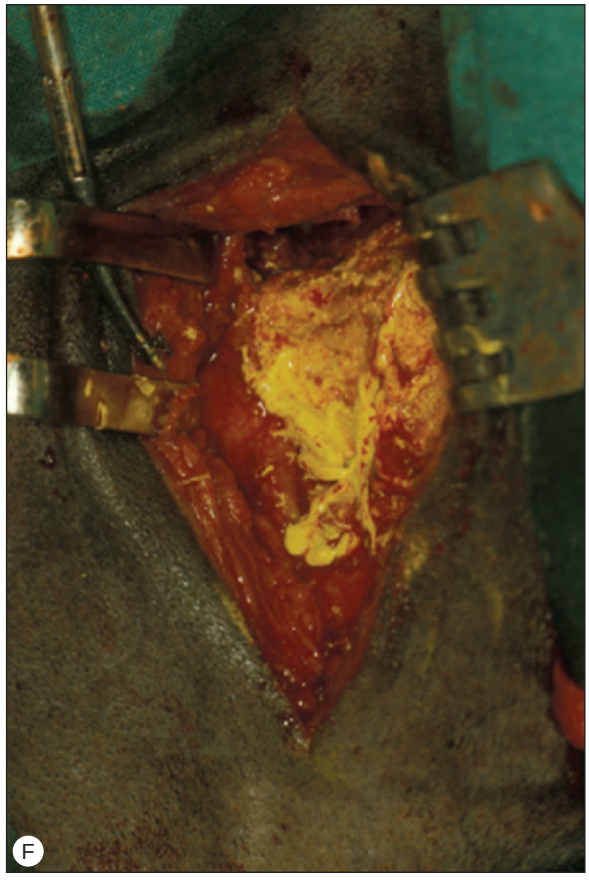


Figure 18.45, cont'd, (E) Vacant alveolus after removal of tooth. (F) Alveolus packed with medicated gauze. (G) Vertical incision after closure. Note the medicated gauze exited via a separate stab incision ventrally.



Figure 18.46. Radiograph showing iatrogenic fragmentation of the caudal root of 208 during the repulsion extraction of 209. The patient continued to show a putrid nasal discharge which only resolved after the removal of 208.

409 and 410) – and should be used in preference to repulsion when oral extraction has not been possible.

The permanent fourth maxillary cheek tooth (109 and 209) is consistently the tooth most frequently in need of extraction. Fortunately, it is the first to erupt and, therefore, has the shortest reserve crowns and roots, rendering it particularly suitable for oral extraction. However, there will be occasions when the crown is damaged and where there is insufficient dental tissue to be grasped with forceps: a surgical extraction would then be required. Surgical access to the lateral aspect of CT4 is limited by the rostral maxillary sinus, the facial crest and the structures ventral to it – the masseter muscle and the transverse and deep facial veins. Rostral extension of a surgical incision at this site is inhibited by the facial artery and vein and the parotid duct. Thus, through lack of a suitable alternative approach it is likely that repulsion will remain the accepted technique for the routine extraction of CT4. The roots of CT5 and CT6 lie progressively further medial with respect to the lateral surface of the maxilla and for similar reasons alternative alveoloplastic techniques are impracticable.

Similar anatomical constraints to those which exist for maxillary CT4, in the forms of the masseter muscle mass and the deep facial blood vessels, apply to mandibular CT6 (311 and 411) rendering it unsuitable for any form of buccotomy or alveoloplastic extraction. Repulsion following the creation of an upward muscle flap remains the likely option for an extraction which is rarely indicated. Easley (personal communication) advocates a two-stage technique, the first to create the muscle flap and to expose the lateral aspect of the mandible, and the second to expose the tooth roots for repulsion. The exposure afforded by the upward flap is more generous than that which can be achieved by a vertical split of the masseter muscle.[40]

Whenever a diagnosis of alveolar periostitis or periodontitis is made and recommendations for treatment are being discussed, owners must be cautioned that dental extraction in the horse is not as straightforward as in man, that the recovery period may be prolonged and that the costs of treatment can be formidable.

Conclusion

Exodontia remains the most appropriate procedure for the treatment of advanced dental disease, which is not amenable to any restorative treatments. Careful planning and selection of the appropriate technique should be made on an individual basis. Specialized instrumentation, and detailed knowledge of the technique and any potential complications are required for all of the techniques described, and the provision of intra-operative radiographic or fluoroscopic imaging is a prerequisite for success. Despite the complications described previously the overall prognosis for complete clinical remission is very good following exodontia,[7,14,43] although more frequent reduction of the opposing teeth, which may supererupt when not in occlusion, will be necessary throughout the horse's life.

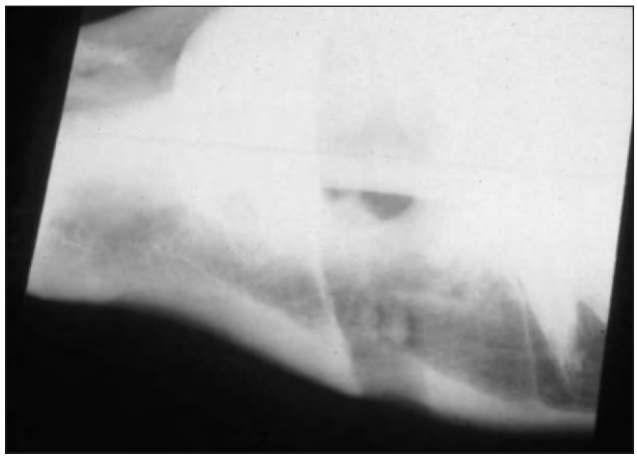


Figure 18.47. Radiograph showing alveolar sequestrum in the space left after the extraction of 307. This pony showed a persistent discharging tract at the ventral aspect of the mandible after repulsion of 307.

REFERENCES

1. Blundeville T (1566) *The Fower Chiefyst Offices Belonging to Horsemanshippe*. London.
2. Markham G (1723) *Markham's Masterpiece*. G Conyers, London.
3. Bartlett J (1782) *The Gentleman's Farriery*. London.
4. Nickel R, Schummer A and Seifarle E (1979) *The Anatomy of Domestic Animals* (2nd edn). Paul Parey, Berlin and Hamburg.
5. Lane JG (1998). In: Equine Medicine, Surgery and Reproduction eds. Mair T, Lore S, Schumacher J and Watsa E. WB Saunders Philadelphia. p. 106.
6. Easley J (1999) Equine tooth removal. In: *Equine Dentistry*, eds. G Baker and J Easley, WB Saunders, Philadelphia, pp. 228–229.
7. Dixon PM, Tremaine WH, McCann J et al. (2000) Equine dental disease: a long term study of 400 cases. Part 4; Apical infections of cheek teeth. *Equine Veterinary Journal*, 32, 182–194.
8. O'Connor JJ (1942) Operations. In: *Dollar's Veterinary Surgery* (3rd edn), Baillière, Tindall and Cox, London, pp. 250–261.
9. Prichard MA, Hackett RP and Erb HN (1992) Long term outcome of tooth repulsion in horses; a retrospective study of 61 cases. *Veterinary Surgery*, 21, 145–149.

10. Evans LH, Tate LP and LaDow CS (1981) Extraction of the equine 4th upper premolar and 1st and 2nd upper molars through a lateral buccotomy. *Proceedings American Association of Equine Practitioners*, 27, 299–302.
11. Lane JG (1987) Equine dental extraction – repulsion vs. lateral buccotomy, techniques and results. *Proceedings World Veterinary Dental Congress*, Birmingham, pp. 135–138.
12. Easley J (1999) Equine tooth removal. In: *Equine Dentistry*, eds. GJ Baker and J Easley, WB Saunders, Philadelphia, pp. 220–249.
13. Merillat MA (1906) Tooth removal. In: *Equine Dentistry and Diseases of the Mouth*. Veterinary Surgery, Vol. 1, Alex Eber, Chicago.
14. Tremaine WH (1987) Oral extraction of equine cheek teeth: A Victorian Technique revisited. *Proceedings World Veterinary Dental Congress*, Birmingham, pp. 139–143.
15. Butson RJ, Robinson J, Schramme MC and May SA (1997) Endodontic therapy for periapical infection in 10 equine cheek teeth: Preliminary results. *Proceedings World Veterinary Dental Congress*, Birmingham, pp. 143–145.
16. Boswell JC, Schramme MC, Livesey LC and Butson RJ (1999) Use of scintigraphy in the diagnosis of dental disease in 4 horses. *Equine Veterinary Education*, 11, 294–298.
17. Boswell JC, Schramme MC, Livesey LC et al. (2001) Endodontic therapy for periapical infection of the cheek teeth a study of 19 cases. *Proceedings 10th Annual Conference ECVS*, Velbert, pp. 216–218.
18. Barakzai S and Dixon PM (2003) Open-mouthed oblique projection for evaluating the clinical crowns of erupted equine cheek teeth. *Equine Veterinary Education*, 15, 143–150.
19. Weller R, Livesey L, Maierl J et al. (2001) Comparison of radiography and scintigraphy in the diagnosis of dental disorders in the horse. *Equine Veterinary Journal*, 1, 49–58.
20. Lane JG (1994) A review of dental disorders of the horse, their treatment and possible fresh approaches to management. *Equine Veterinary Education*, 6, 13–21.
21. Scrutchfield L, Schumacher J and Martin T (1996) Correction of abnormalities of the cheek teeth. *Proceedings American Association of Equine Practitioners*, 42, 11–21.
22. Guard WF (1951) Equine operations. In: *Surgical principles and Techniques*, WF Guard, Columbus, Ohio, pp. 78–89.
23. Dixon PM (1997) Dental extraction and endodontic techniques in horses. *Compendium of Continuing Education for the Practicing Veterinarian*, 19, 628–637.
24. Dixon PM (1997) How do periapical infections develop in the horse? *Proceedings of Fifth World Veterinary Dental Congress*, Birmingham, pp. 130–134.
25. Baker GJ (1982) Dental disorders in the horse. *Compendium of Continuing Education for the Practicing Veterinarian*, 4, S507–515.
26. Edwards GB (1993) Retention of permanent cheek teeth in 3 horses. *Equine Veterinary Education*, 5, 299–302.
27. Orsini PG (1992) The oral cavity. In: *Equine Surgery*, ed. JA Auer. WB Saunders Philadelphia, pp. 296–305.
28. Pirie RS and Dixon PM (1993) Mandibular tumours in the horse: a review of the literature and 7 case reports. *Equine Veterinary Education*, 5, 287–294.
29. Mueller POE (1991) Equine dental disorders, cause, diagnosis and treatment. *Compendium of Continuing Education for the Practicing Veterinarian*, 13, 1451–1461.
30. Easley J (1991) Recognition and management of the diseased equine tooth. *Proceedings American Association of Equine Practitioners*, 37, 129–139.
31. Schumacher J and Honnas CM (1993) Dental surgery. In: *Veterinary Clinics of North America (Equine Practice) – The equine head*, eds. CM Honnas and AL Bertone. WB Saunders, Philadelphia, pp. 133–152.
32. Dimitroulis G (1997) Simple and surgical exodontia. In: *A Synopsis of Minor Oral Surgery*. Butterworth-Heinemann, Oxford, pp. 31–47.
33. Shira RB (1968) Principles of exodontia. In: *Textbook of Oral Surgery*, ed. WC Guralnick. Little, Brown and Co, Boston, p. 79.
34. Hooley JR and Golden DP (1994) Surgical extractions. In: *Dental Clinics of North America – Basic procedures in oral surgery*, ed. KR Koerner. WB Saunders, Philadelphia, pp. 217–236.
35. Baker GJ (1985) Oral surgical techniques In: *Veterinary Dentistry*, ed. CE Harvey. WB Saunders, Philadelphia, pp. 229–234.
36. Turner AS and McIlwraith CW (1989) Repulsion of cheek teeth. In: *Techniques in Large Animal Surgery* (2nd edn). Lea and Febiger, Philadelphia, pp. 235–239.
37. Dixon PM (1997) Post-extraction oro-maxillary (antral) fistula: is a bone cement prosthesis the solution? *Proceedings of Fifth World Veterinary Dental Congress*, Birmingham, pp. 147–150.
38. Wheat JD (1974) Sinus drainage and tooth repulsion. *Journal of the American Veterinary Medical Association*, 164, 363–364.
39. Baker GJ (1985) Oral examination and diagnosis: management of oral diseases. In: *Veterinary Dentistry*, ed. CE Harvey. WB Saunders, Philadelphia, pp. 217–228.
40. McIlwraith CW and Turner AS (1987) Caudal molar extraction. In: *Equine Surgery: Advanced Techniques*. Lea and Febiger, Philadelphia, pp. 260–263.
41. Kertesz P (1993) Dental diseases and their treatment in captive wild animals. In: *A Colour Atlas of Veterinary Dentistry and Oral Surgery*, ed. P Kertesz. Wolf, London, pp. 215–281.
42. Lane JG and Kertesz P (1993) Equine dental surgery. In: A Colour Atlas of Veterinary Dentisry and Oral Surgery. ed P Kertesz Wolf London, pp 199–214.
43. Lane JG (1998) A study to evaluate the surgical methods used to extract the cheek teeth of the horse. DESTS Thesis, Royal College of Veterinary Surgeons, London.
44. Pascoe JR (1991) Complications of dental surgery. *Proceedings American Association of Equine Practitioners*, 37, 141–150.
45. Orsini PG, Ross MW and Hamir NM (1992) Levator nasolabialis muscle transposition to prevent an orosinus fistula after tooth extraction in horses. *Veterinary Surgery*, 21, 150–156.

19 Endodontic Therapy

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Introduction

Endodontics is that branch of dentistry that covers the diagnosis, management and prophylaxis of diseases of the dental pulp and periapical tissues.[1] As early as 1884, it was commented that 'A few moments consideration of the original cause of trouble at the apex of roots will enable us to realize what is required to be accomplished in the way of successful treatment. If the original cause is admitted to be irritation from decomposing pulp, its removal will, in most cases, affect a cure.'[2]

In man, at least, dental caries and dental trauma are the most common causes of acute pulpitis. The etiopathogenesis of pulpitis in the horse is less well documented, but it is seen that trauma, impaction, periodontal disease and caries of cementum may all be involved.[3] Prior to 1900, many surgical techniques were developed that did, in many cases, successfully drain and resolve periapical abscesses and preserve the teeth.[4]

Many of the principles of endodontic therapy used in man can be applied to the horse and in recent years, clinical experience and case results have shown success in preserving teeth.[5,6] As in man, the reimplantation of teeth – particularly incisors – is frequently successful in that the teeth and associated permanent tooth germs have survived. It is not uncommon, however, for maleruptions to follow such events. In 1959 it was suggested that some cases of 'alveolar periostitis' in young horses could be managed by apicoectomy (root end resection) and drainage of root abscesses.[7] More recently, mixed results have been reported from pulp ablation and root canal therapy in horses. The indications are that the results are better in mandibular teeth than maxillary teeth.[5] In an experimental study of a coronal access (through erupted crown) to the pulp chamber of normal incisor teeth in the horse, it was shown that when 44 incisor teeth of eight horses aged from 6–11 years of age were treated, there were no surgical complications and the teeth continued to erupt and to wear over an 18-month period.[8]

In this chapter, the principles of endodontic therapy will be described, together with important guidelines for the choice of care and the management of complications and sequelae.

Management of acute pulp exposure

Accidental fractures of the teeth may expose the pulp (see Chapter 8). In some instances this may occur during dental work when incisor bites (e.g. parrot-mouthed horses) or major arcade irregularities and overgrown teeth are cut. Under such circumstances the clinicians should be prepared to offer first aid that is directed at minimizing the degree of pulp necrosis and complications. It has frequently been claimed that pulp exposure under such conditions does not require treatment as the natural defense mechanisms of reparative dentin are adequate and no complications result. In fact, in some species it is routine, as a management technique, to cut particular teeth, for example the canines of piglets and fighting teeth (canines) of new world camelids (llamas). This apparent conflict in complication detection is possibly due largely to lack of observation. If cases in which the pulp has been exposed are followed up, it will be found that at least 25 per cent of them develop apical swellings that are painful to pressure and which may form root abscessations. In young animals, when deciduous teeth are involved, these swellings are often missed and many complications resolve when the permanent teeth erupt.

When the crown is fractured and the pulp exposed, it should be either capped or the pulp removed by a pulpotomy procedure. Many materials have been used as pulp-capping agents. Most of these supplement the most widely used material, calcium hydroxide. The supplementation consists of the use of enzymes, antiseptics, antibiotics and anti-inflammatory agents.[9,10] Pulp capping is the covering of exposed pulp with a medicated dressing in an attempt to preserve the pulp's vitality. Pulpotomy is the removal of the coronal portion of the pulp and covering of the remaining pulp to inhibit periapical disease and preserve the vitality of the radicular (tooth root) pulp tissues.

Calcium hydroxide has been the preferred material since the 1930s.[11] Prior to that time, pulp therapy had consisted of the application of arsenic and other fixative agents that devitalized the exposed pulp. The advantages of calcium hydroxide are its initial devitalization of contact pulp and contiguous tissues, followed by the formation of a dentin

bridge (reparative dentin) at the junction of the necrotic and vital tissues. The exact mechanism of this process is not clearly understood; however, when other compounds of similar alkalinity (pH 11) are used, an extremely unhealthy liquefactive necrosis takes place. Consequently calcium hydroxide (e.g. Pulpdent, Henry Schein Inc., 5 Harbor Park Drive, Port Washington, New York 11050) has remained the material of choice. It is interesting to note that in the history of drugs used in pulp capping and pulpotomy in man that in 1883 Hunter reported to the Missouri Dental Association on the successful use (98 per cent) of sparrow droppings mixed with sorghum molasses for pulp capping.[10]

Technique for tooth capping

The extent and duration of pulp exposure is evaluated and documented. If necessary, crown fragments from the damaged tooth are removed and prepared. Depending on the site of the tooth this may be carried out under sedation and peripheral nerve blocks. Some cases will require general anesthesia to facilitate the procedure. It is important that damaged pulp tissue be removed. The use of an operative loupe (for magnification) and spoon excavator allows the surgeon to cut down to the vital pulp. Any hemorrhage must be controlled before the pulp cap material (calcium hydroxide) is applied. This may be achieved by epinephrine mixed with saline washes and by pressure packing. The use of vacuum and air drying achieves the ideal operative site. Calcium hydroxide is mixed according to the manufacturer's instructions and used to cover the exposed pulp.

Care is then taken, once the material has set up, to modify the occlusal contact made by the treated tooth so that occlusion is prevented for 3 months or more. In this manner the reparative dental bridge is completely formed before attrition from dental wear begins. In the horse, provided occlusal contact is avoided, then additional restorative covers are not needed over the calcium hydroxide (Fig. 19.1A–D).

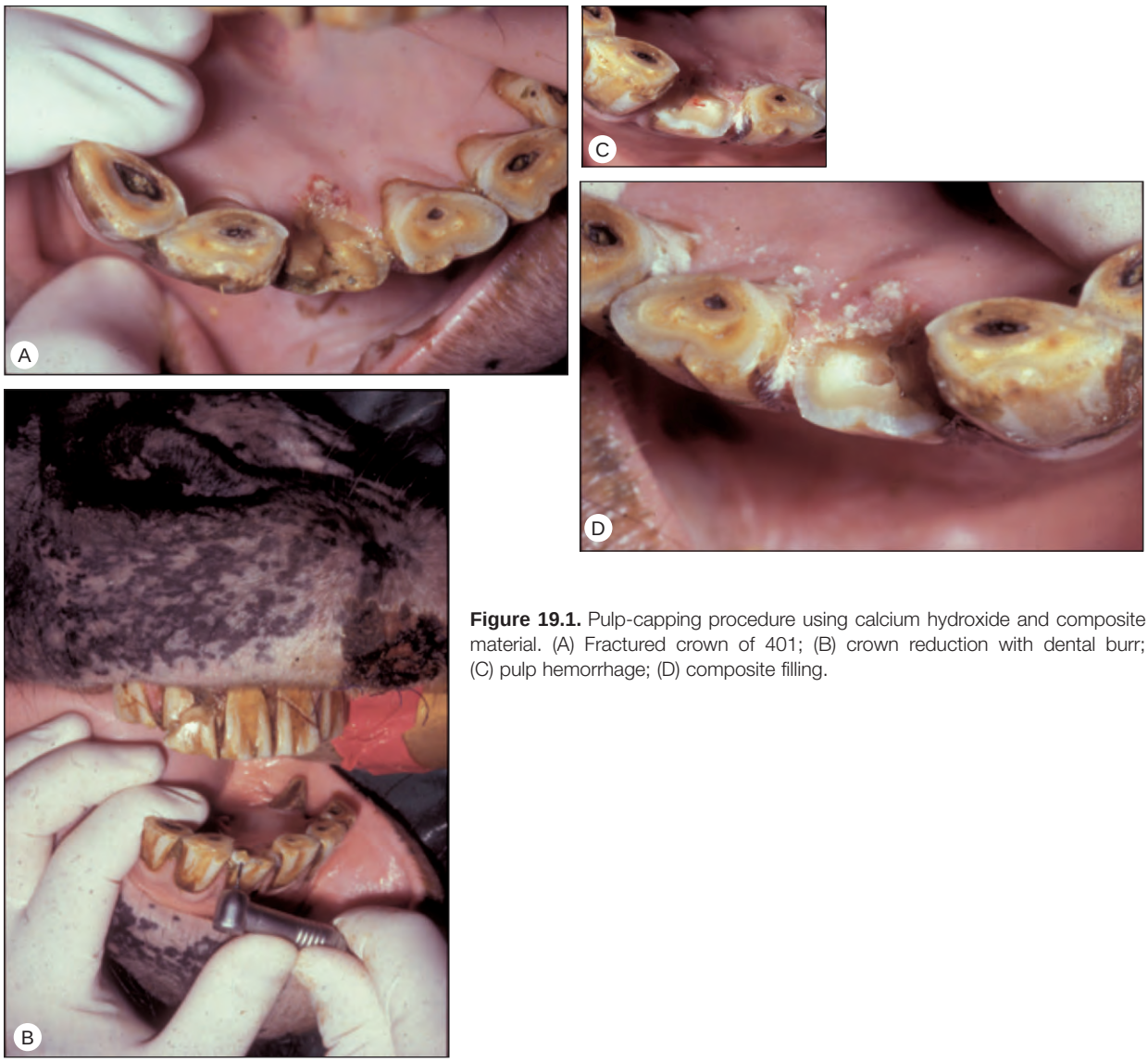


Figure 19.1. Pulp-capping procedure using calcium hydroxide and composite material. (A) Fractured crown of 401; (B) crown reduction with dental burr; (C) pulp hemorrhage; (D) composite filling.

Follow-up care requires that the tooth apex be monitored for periapical swellings and gingival ulceration and bone erosion (radiographic check-ups may be indicated). If complications arise it may be necessary to either complete a pulpectomy or extract the tooth.

Pulpectomy may also be used when there is chronic coronal pulp exposure. In such cases, the pulp chamber is opened from the crown or from the fracture site. The pulp chamber is cleaned out and prepared for filling (see root end resection below).

Surgical endodontics

In clinical practice most cases of pulpitis are not presented in the acute stage of the disease. The diagnosis of pulp infection is based on clinical findings and radiographic analysis. The selection of endodontic therapy versus tooth extraction in the management of equine dental disease is based upon the summation of the following observations: (a) the absence of other significant disease, (b) the localization of the periradicular disease process, (c) the availability of equipment and operative expertise and (d) client compliance and economic factors.

It is particularly important that the clinician evaluates if there are local factors indicating complications. Mandibular fistulae should be probed and pressure flushed to ensure there is no periodontal leakage. If there is active periodontal leakage or accompanying periradicular pathology, for example maxillary sinus empyema, this may inhibit the use of root end resection, endodontic therapy and tooth preservation. In young horses (4–6 years old), there may not be adequate maturation of the tooth roots to enable root filling to be achieved. There are only a few anecdotal stories of successful endodontic therapy in young horses. The technique of apexification, to promote root closure, is an important precursor of endodontic therapy. Apexification (surgical exposure, drainage of periradicular disease and application of calcium hydroxide), however, requires more than one general anesthetic and consequently may not be acceptable to the client. During interactions between clinician and client the potential of failure of endodontic techniques and ultimate extraction of the diseased tooth must be discussed. The client's compliance may be achieved if it is explained that endodontic success will avoid all the complications of tooth extraction, and if it fails then at least every effort has been made to save the tooth. The economics may then be weighed by both the veterinarian and the owner to allow a management plan to be agreed upon.

Technique for root end resection

The terms surgical endodontics, apicoectomy and root end resection are synonymous. In this account the term root end resection will be used. As has been described, it is commonly seen that the indications for endodontic therapy in the horse are the clinical signs associated with periradicular disease, swellings, drainage and radiological evidence of apical haloes (or bone lysis). A final check for conditions suitable for endodontic surgery are carried out under general anesthesia and include testing for periodontal leakage into and from the oral cavity as well as crown impactions. The relief, by interproximal grinding, of occlusal impaction should be carried out as the first procedure in the endodontic treatment surgery.

Equipment

In addition to the equipment and facilities for general anesthesia with monitoring, general surgical instruments and the use of intra-operative radiology, the following are required for root end resection and endodontic therapy:

1. suction
2. electrical hemostasis (diathermy)
3. bone trephines and rongeurs (angled and straight)
4. low- and high-speed drills with 10:1 reduction hand piece (Fig. 19.2)
5. diamond disks and abrasive burrs
6. short and long inverted cone burrs
7. lentula spiral
8. endodontic files and broaches
9. endodontic spoon excavator
10. periodontal probes and explorer
11. 2 per cent hypochlorite solution
12. paper points
13. zinc oxide powder and eugenol
14. glass mixing slab, syringes
15. plastic instruments and spatula



Figure 19.2. Dental drill with reduction gear hand piece.

16. gutta-percha (warm gutta-percha delivery system is ideal, e.g. Obtura II system)
17. endodontic spreaders
18. amalgam, mixer and carrier
19. an alternative to amalgam is Super EBA with alumina (Bosworth Co., Skokie, IL).

Equipment required for hemostatic support:

1. diathermy
2. suction
3. saline with 8 per cent epinephrine
4. retraction cord (SilTrax, Pascal Co. Inc. 2929 N.E. Northrup Way, Bellevue, WA 98004).

Surgical technique

Successful endodontic therapy in the horse, defined as the resolution of periradicular disease, and the retention and continued eruption of the diseased tooth, is dependent upon the application of good endodontic technique.

Access

After completing the oral examination, correction or relief of impactions and final radiographic analysis, drape off the surgical site after routine clipping, shaving and skin sterilization. Approach through a cruciate skin incision with retraction of the periosteum along the incision lines. The precise site is tooth dependent; it is easier to complete the root canal procedure on 107, 108, 207 or 208 if the horse is positioned on its back. Maxillary teeth are approached from a dorsolateral site. Bone over the disease root is removed by using a combination of a trephine, rongeurs and a Hall air drill. Complete exposure of the site is essential to afford complete excision of all periradicular disease. This is relatively easy in mandibular teeth but more difficult in maxillary teeth. In some cases of maxillary tooth disease associated with sinus empyema this may prove to be impossible and it may mean that the endodontic therapy treatment option is abandoned at this stage and the tooth is repelled.

After suitable exposure, the root end is divided using either a diamond disk attached to the dental drill and reduction hand piece or it may be cut off with a conical abrasive burr. The object is to produce an angled cut which is shorter to the outside on a 15°–20° plane.

Sterilization

It has been documented that inadequate sterilization of the diseased pulp chamber is the cause of long-term complications and failure of endodontics. Sterilization is achieved by the physical removal of all of the non-vital, necrotic pulp tissue, and this is done by shaving away portions of the dentinal walls with a file. Thorough and high-volume irrigation of the cavities with sterile saline and 2.5 per cent sodium hypochlorite solution is essential in this procedure. The complexity of the shape of the pulp chambers of the horse, however, makes endodontic curettage (filing etc.) particularly difficult and it means that complete cleansing of the pulp chamber is almost impossible. However, if carried out thoroughly with complete sealing and obturation, success is still achieved. This may be explained using the 'Dom Perignon' analogy, that is, if you place a perfect cork in the bottle, the result inside the bottle is champagne, not vinegar. Hence, the key to success is in sealing, obturation and root end closure.

Obturation

Obturation is the process of complete filling of the exposed pulp chamber. The objectives are first to eliminate all avenues of leakage from the oral cavity or the periradicular tissues into the root canal system. Second, effective obturation should seal within the system any irritants that cannot be fully removed during canal cleaning and shaping. This recognizes that microbial irritants (micro-organisms, toxins and inflammatory products from pulp residues) are the prime cause of pulpal disease and its subsequent extension in periradicular disease.[12] The American Association of Endodontists has defined obturation as the three-dimensional filling of the entire root canal system as close to the cementodentinal junction as possible. Biologically compatible root canal sealers are used in conjunction with the core filling material to establish an adequate seal.[13]

A plethora of materials have been used over the past 150 years for root canal filling in man and historically, gutta-percha has been the material of choice. Despite its relative lack of rigidity and adhesiveness and its ease of displacement under pressure, it is still the ideal material for use in retrograde filling, after root end resection, as done in the horse.

The characteristics of the ideal root canal filling, as modified after Grossman *et al.*[14] by Wiggs and Lobprise[15] are that it is:

1. easily manipulated and placed, allowing adequate working time prior to setting up
2. dimensionally stable, there is no shrinkage or expansion after compaction
3. conformable and adaptable to the various shapes and contours of the canal, while sealing it apically (coronally in retrograde filling) and laterally
4. non-irritating to periapical tissue (this is not significant in the horse where retrograde filling is the rule)
5. non-porous, impervious to moisture
6. unaffected and insoluble in tissue fluids; incapable of corroding or oxidizing
7. bacteriostatic
8. non-staining to dental or peridental structures (this is not significant in the horse where retrograde filling is the rule)
9. sterile or easily and quickly sterilized for immediate use
10. removable without difficulty
11. biologically unharmful
12. radiopaque.

In practice, the combination of gutta-percha and a mixture of zinc oxide and eugenol effects the best material. After exposure and preparation/sterilization, it is key for the endodontist to be able to work in a dry surgical field. The root

canal and pulp chamber is dried with paper points and warm air and the periradicular area is packed off to control osseous bleeding. The surgical assistant should have the primary responsibility of ensuring the integrity of the working area using suction and packing. The zinc oxide and eugenol mixture is mixed on the glass slab so that it will form a thick viscous mixture that can be injected by syringe through an 18-guage catheter. The pulp chamber is then lined with this material and filled with gutta-percha. This process is made easier using the entrance heating systems for intracanal softening of gutta-percha before compaction (such as the Obtura II system, Obtura Corp., Fenton, MO).[13] Canal and chamber obturation may be checked using lateral and oblique radiographs.

Apical seals

As the root canal filler sets up, the clinician uses either the short or the long inverted cone burr to construct an apical inlay cut to hold the apical sealer. The size of this apex after root end resection may well be quite large and require multiple mixes of standard amalgam to fill. This introduces the potential for an incongruity of amalgam sealer and subsequent failure of the seal. For this reason, although more expensive, other seals may be used such as glass ionomers or Super EBA. In the past 3 years, newer filling and sealing materials have been developed that can set up in the presence of moisture. Mineral trioxide aggregate (MTA) is such a material – it is essentially micropulverized, sterilized 'Portland cement.' As developed and packaged for use in man, the product is expensive (ProRoot MTA System costs $325.00 with $249.00 for refills).

Having completed the procedure, the apex is packed with a surgical dressing and the surgical site closed leaving drainage access.

Postoperative care includes analgesic and anti-inflammatory drugs (phenylbutazone) and antibiotics. The surgical site is checked daily and the wound pack removed after 3–4 days. Special diets are usually not needed in the care of horses after endodontic therapy (see Figs 19.3–19.11 for endodontic technique).

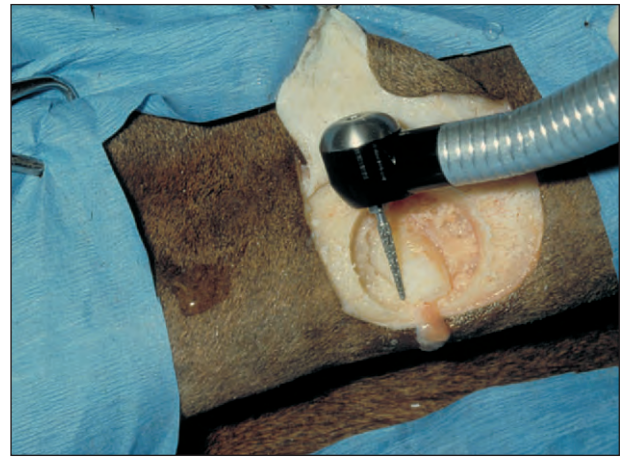


Figure 19.4. Endodontic technique: documented in anatomical preparations.

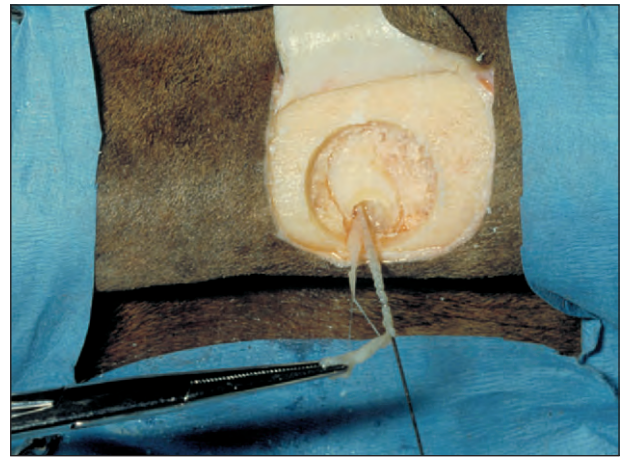


Figure 19.5. Endodontic technique: documented in anatomical preparations.

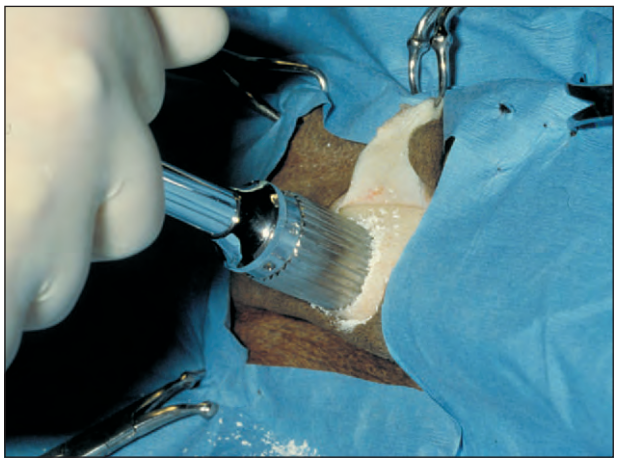


Figure 19.3. Endodontic technique: documented in anatomical preparations.

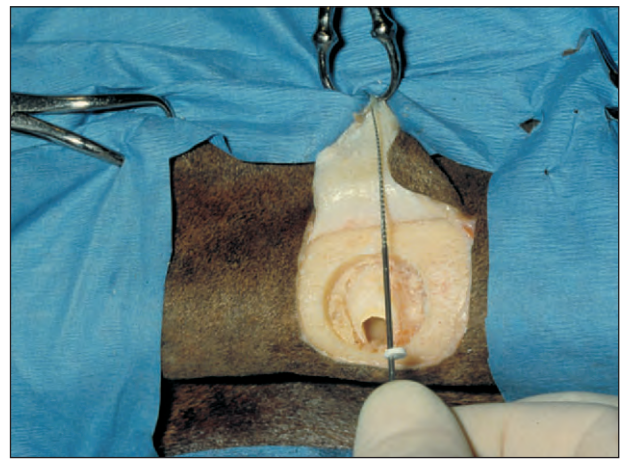


Figure 19.6. Endodontic technique: documented in anatomical preparations.

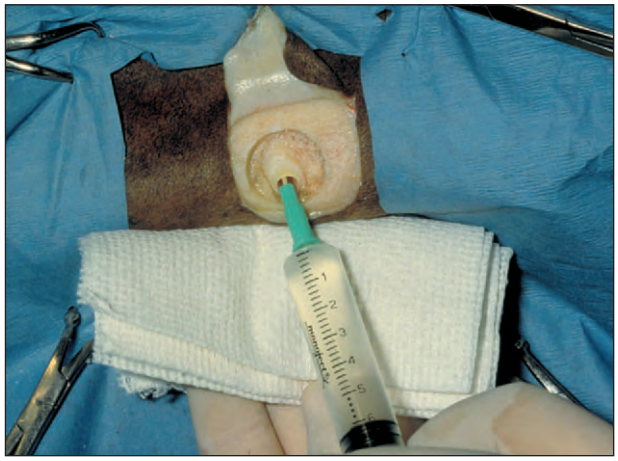


Figure 19.7. Endodontic technique: documented in anatomical preparations.

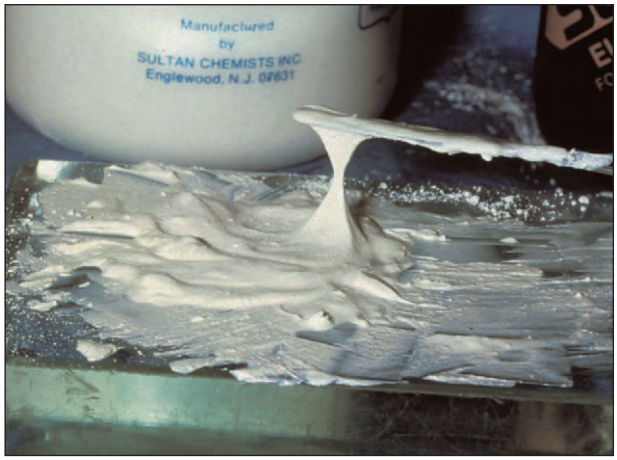


Figure 19.8. Endodontic technique: documented in anatomical preparations.

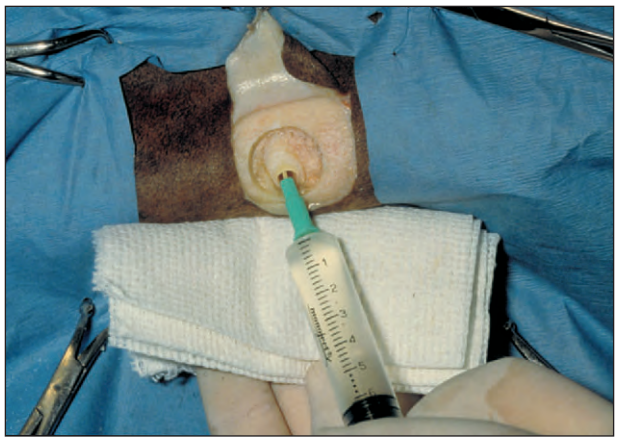


Figure 19.9. Endodontic technique: documented in anatomical preparations.

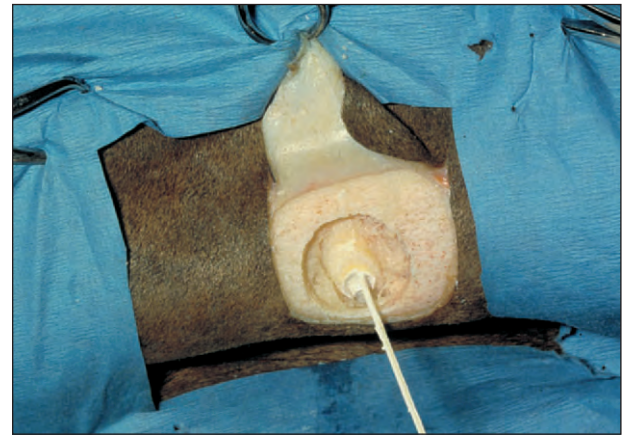


Figure 19.10. Endodontic technique: documented in anatomical preparations.

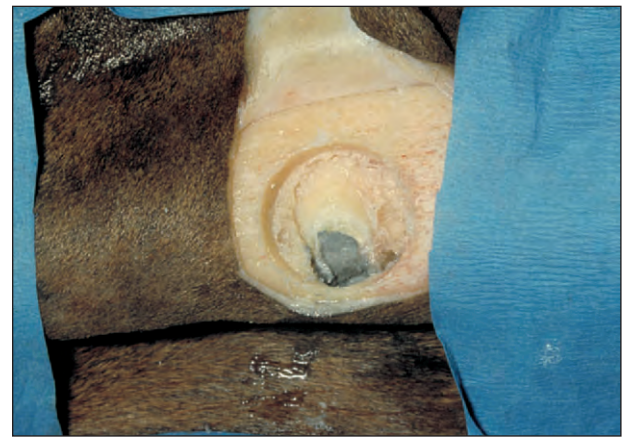


Figure 19.11. Endodontic technique: documented in anatomical preparations.

Results of endodontic therapy in the horse

The literature contains a number of descriptions for the use of endodontic techniques but there are no detailed accounts of long-term follow-up of the results and complications. It has been shown in experimental studies of endodontic therapy in the normal incisor teeth of horses that in 44 teeth that were treated and followed over an 18-month period, there continued to be normal eruption and wear. In personal communications with numerous veterinarians in the USA, Canada, Europe and Australia, the author estimates that with careful case selection up to 80 per cent success may be achieved. It appears that experience with endodontic technique and care in case selection, particularly in the maturity of the teeth affected, are the key factors to success.[16]

The following is an analysis of some 42 endodontic therapies carried out at the University of Illinois over the past 12 years. There were 38 horses, four of which had two separate teeth treated, either 306 and 307 or 406 and 407. Of the 42 treatments, only two were in horses under 3 years

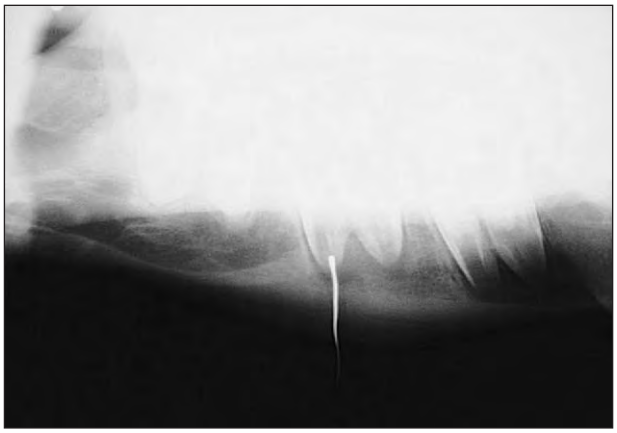


Figure 19.12. Mandibular dental fistula rostral root 307.

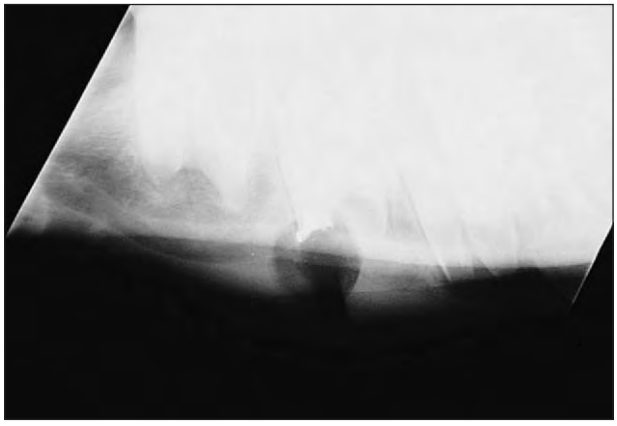


Figure 19.14. One month post root end resection.

of age, and only nine involved maxillary teeth. No success was achieved in five maxillary teeth in association with sinus empyema on initial therapies; in four of these cases the teeth were subsequently extracted and the other one had two follow-up periradicular drainage procedures with an eventual successful outcome. Four horses with periradicular disease associated with 108 or 208 had successful outcomes (Figs 19.12–19.15). The 33 other teeth were all rostral mandibular teeth. There were seven distal root 307s, five distal root 407s, nine mesial root 308s and four mesial root 408s, two each distal root 308 and 408 and two and one proximal root 309 and 409 respectively. Of the mandibular teeth, the mean duration of clinical signs was 6 months (range 2–14 months). Of the 33 mandibular endodontic treatments with follow-ups of 8 months and more, only three teeth required further surgery. Of these three, one tooth was preserved and two were extracted. The overall success rate as defined by absence of complications and preservation of the tooth was 32 out of 38 horses (84 per cent).

In a second series of 20 teeth (8 mandibular and 12 maxillary) in 19 horses aged 2–13 years (mean age 6.4 years) follow-up data were collected for 12 months on all 19 horses and 15 were available for long-term (more than 24 months) follow-up. In 8 of the 19 horses, signs of infection persisted long term. Follow-up radiography confirmed that the filling agent was retained in the pulp cavities in all cases and that the treated teeth continued to erupt. The technique used was silver amalgam in 17 teeth, intermediate restorative material in two teeth and glass ionomer cement in one tooth. Gentamicin-impregnated polymethylmethacrylate beads were placed in the tissues surrounding the treated tooth apices.[17] It was suggested that such endodontic techniques have a role in the management of dental infections in the horse, but that careful case selection is required.

Summary

Endodontic therapy, tooth preservation rather than extraction, is a viable technique for use in selected dental disease cases in horses. The keys to success are clear and effective client education leading to compliance, concentration on thorough techniques and adherence to proven endodontic practices.



Figure 19.13. Probe along dental track.



Figure 19.15. Six months post root end resection.

REFERENCES

1. Jensen JR and Serene TP (1984) *Fundamentals of Clinical Endodontics*. Kendall Hunt, Dubuque, Iowa, pp. 1–5.
2. Whitehouse W (1884) Dental decay. *British Dental Journal*, 27, 238–240.
3. Baker GJ (1985) Oral examination and diagnosis: management of oral diseases. In: *Veterinary Dentistry* (1st edn), ed. CE Harvey, WB Saunders, Philadelphia, pp. 217–234.
4. Weinberger BW (1948) *Introduction to the History of Dentistry*, CV Mosby, St Louis, p. 205.
5. Baker GJ and Kirkland DK (1992) Endodontic therapy in the horse. *Proceedings of the 38th Annual Meeting of the American Association of Equine Practitioners*, 329–335.
6. Dixon PM (1997) Dental extraction and endodontic techniques in horses. *Compendium on Continuing Education for the Practicing Veterinarian*, 19, 628–638.
7. Eisenmenger F (1959) Surgical treatment of alveolar periostitis in young horses. *Wienna Tierarzle Mochenschrift*, 46, 51–70.
8. Garcia F, Sanroman and Llonens MP (1990) Endodontics in the horse: an experimental study. *Journal of Veterinary Medicine*, 37, 205–214.
9. Cvek M (1981) Endodontic treatment of traumatized teeth. In: *Traumatic Injuries of the Teeth* (2nd edn), ed. JO Andreasen, WB Saunders, Philadelphia, pp. 318–349.
10. Seltzer S and Bender IB (1990) Pulp capping and pulpotomy. In: *The Dental Pulp Biologic Considerations in Dental Procedures* (3rd edn), eds S Seltzer and IB Bender, Ishiyaku EuroAmerica, St Louis, pp. 215–249.
11. Hermann BW (1930) Dentinobliteran der Wurzelkanale nach der Behandlung mit Kalzium. *Zahertzl Rund*, 39, 888.
12. Naidorf IJ (1974) Clinical microbiology in endodontics. *Dental Clinics of North America*, 18, 329.
13. Gutmann JL and Witherspoon DE (1998) Obturation of the cleaned and shaped root canal system. In: *Pathways of the Pulp* (7th edn), eds H Cohen and RC Burne, CV Mosby, St Louis, pp. 258–361.
14. Grossman LI, Obiet S and Del Rio C (1988) *Endodontics* (11th edn), Lea and Febiger, Philadelphia, pp. 226–245.
15. Wiggs RB and Lobprise H (1997) Basic endodontic therapy. In: *Veterinary Dentistry Principles and Practice*, eds RB Wiggs and H Lobprise, Lippincott-Raven, Philadelphia, pp. 280–328.
16. Emily P, Orsini P, Lobprise HB and Wiggs RB (1997) Oral and dental disease in large animals. In: *Veterinary Dentistry Principles and Practice*, eds RB Wiggs and H Lobprise, Lippincott-Raven, Philadelphia, pp. 559–579.
17. Schramme MC, Boswell JC, Robinson J *et al.* (2000) Endodontic therapy for periapical infection of the cheek teeth: A study of 19 horses. *AAEP Proceedings*, 46, 113–116.

20 Dental Materials in Veterinary Dentistry

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Introduction

Our goal as veterinary dentists is to maintain oral health and function in our patients by preventing disease and pain. Frequently, we improve oral health by correcting problems and treating disease, correcting malocclusions to increase the efficiency of mastication, relieving pain from fractured or abscessed teeth, and treating periodontal disease.

Dental materials are constantly changing. The basic categories of materials include restoratives and bonding agents, impression materials, casting materials, and periodontal materials. Understanding the biocompatibility, chemical, physical, and structural properties of dental materials is essential in providing the appropriate care for our patients.[1]

Restorative materials

Choosing a particular restorative material is based on the operator's personal preference, knowledge of the material's physical properties, and clinical data. Significant factors in product selection are the strength of a restorative material needed for a specific area in the mouth, the esthetics of the material, and other handling characteristics.

Direct placement restorative materials

The direct placement restorative materials are those that are placed into the prepared tooth defect without being fabricated in the laboratory prior to placement.[2] The materials are either metallic (amalgam) or polymeric (composites, glass ionomers, compomers, and acrylics).

Dental amalgam was introduced into the United States in 1833 and is still in use.[3] Half of the posterior single tooth restorations in humans today are dental amalgam. Amalgam is easy to place, is cost effective, and has a high resistance to wear and fracture. Its use is limited to the restoration of posterior teeth due in part to its non-esthetic gray color (Fig. 20.1A,B). Mercury contamination in the environment and the potential toxicity to some individuals are factors that should be considered when choosing to use amalgam.[3–5]

Dental amalgam is a mixture of a silver alloy (silver, tin, and copper) and mercury. Silver alloys have, historically, had copper contents of 2–4 per cent, which have now been replaced with copper of 13–30 per cent.[5–7] The higher copper content results in amalgams with greater strength and resulting in better longevity and less corrosion.[8]

Dental amalgam does not bond to tooth structure so undercuts in the cavity preparation are placed to retain it. To accomplish this, healthy dentin and enamel are removed which may slightly weaken the tooth.[8,9] Bonding agents and adhesives are now available to bond amalgam to tooth structure.[2] They are applied to the prepared dentin. Micromechanical bonds are formed in the dentinal tubules. This decreases dentin sensitivity and reduces marginal leakage, and in addition, provides support to the restoration and tooth crown.[8,10]

Amalgam was the restorative of choice until the development of the composite resins. Composite resins were introduced in the 1960s. However, it was not until the 1980s that they were comparable to amalgam with respect to wear, retention, handling, etc. Before dental composites, other esthetic materials were used, such as acrylic resins. They had a high coefficient of thermal expansion, and high polymerization shrinkage which predisposed them to recurrent caries. Silicate cements were also used during this time, but needed to be frequently replaced due to their solubility in the oral fluids. Once introduced, the composites replaced the acrylics and the silicate restorative materials.[6]

Composite resins are the materials of choice for directly placed restorations. They are smooth and esthetic, versatile, and appropriate for most situations. They do not have the fracture toughness that the metallic restorations have, but they are versatile and appropriate for most situations.

In the equine patient, cemental hypoplasia, or secondary infundibular decay of the maxillary cheek teeth results in a large cavity in the occlusal surface of the crown.[11–15] A composite restoration placed in this compromised structure lends support to the tooth and may prolong the life or use of the tooth (Fig. 20.2).

The occlusal cavity is prepared with air abrasion and/or a dental round bur to remove organic debris. The tooth is etched with phosphoric acid (37–40 per cent), rinsed thoroughly, and lightly air dried so that the tooth remains moist. A dental adhesive is applied and light cured according to manufacturer's instructions. The composite resin is placed in the prepared cavity, light cured, and contoured with a finishing bur.



Figure 20.1. (A) Occlusal caries in a canine maxillary molar. (B) Occlusal caries restored with amalgam.

The physical properties of replacement materials are very important when selecting them for restoration of teeth. Ideally, they should have the same characteristics as enamel. A few of the important properties of composites are low polymerization shrinkage, a coefficient of thermal expansion similar to tooth structure, and high wear resistance.

Polymerization is the process of making large polymer molecules from smaller monomer units. This process is activated by heat, chemicals, or light. A polymerized material is smaller in volume than its original components. This is referred to as polymerization shrinkage.[8] The consequence of this change in volume is the potential for leakage at the margin of the restoration. The coefficient of thermal expansion[6,8] is the shrinkage and expansion of a material with changes in temperature. This should approximate that of the dentin and enamel. When mismatched, differential shrinkage or expansion causes separation or stress between the tooth and the restoration.



Figure 20.2. Defect in the infundibulum of a maxillary molar restored with composite resin (courtesy of Dr David Klugh).

Another important characteristic of a composite material is the modulus of elasticity.[1,6,8,9] It is a measure of the stiffness of the material. As the modulus of elasticity increases, the stiffness of the composite resin increases. There are times when the use of a low-modulus material is more appropriate so that the restoration can flex with the tooth.

The components of a dental composite include a resin matrix and inorganic filler particles. Composite resins wear in the oral cavity due to occlusal forces. The higher the filler content of a composite resin, the harder and more wear resistant they are.[8] The organic resin matrix and the inorganic filler particles are bonded together by a coupling agent. The coupling agent that is commonly used is silane.

Polymerization of composite resin systems is initiated by either chemical or light activation. The chemically activated materials are a two-paste system. One paste contains a tertiary amine activator and the other a benzoyl peroxide initiator. When mixed together free radicals are released that initiate polymerization that results in a hard material.[1,5] Mixing the two pastes can trap air bubbles, which might decrease the material's strength. At the same time, these materials cure evenly, which results in a stress-free material when completely polymerized. The working time for placement is very short due to the rapid polymerization process, which makes them difficult to work with.

The light-cured composites, on the other hand, have a working time that is not limited, as polymerization does not begin until the material is activated by light. Once activated, the material polymerizes toward the light, which results in stresses that are not found in the self-cure (chemically activated) composites. A diketone and an amine in the composite are activated with visible blue light. Light energy in the range of 480 nm matches the chemical activator in most composite resins.

The resin matrix of a dental composite is bis-GMA or similar dimethacrylate monomer. The filler particles are inorganic particles such as ground and silanated glass. The size of the filler dictates the smoothness of the surface, or how well it polishes. Smaller particles have a smoother surface. Composites are categorized by their filler particle size. As the filler content of a composite increases (and the resin content decreases), shrinkage decreases, thermal expansion is more like tooth structure, and wear resistance increases.

The four categories of dental composites are the macrofill, small-particle, microfill, and hybrids, which are defined by the size of their filler particles. Of those, the hybrids and microfills are the most esthetic composites and are still used.[16] The most recent category of esthetic composite is the microhybrid. The range in particle size of the composites is 0.03 μm in the smallest particle of the microfills to 25 μm in the largest of the macrofilled composites. This thousand-fold difference is responsible for the difference in the characteristics of the various composite materials. All composites used today are bonded to tooth structures with dentin bonding agents and adhesives.

The first composite of the 1960s was the macrofilled composite. This material had quartz fillers with the particle size ranging from 10–25 μm (Fig. 20.3). The large filler size made them rough in appearance and more prone to plaque accumulation. The bacteria in dental plaque leads to gingivitis and periodontitis in some patients and must be taken into consideration. The macrofilled composites were used before the dentin bonding systems were developed thoroughly, and as a result there was leakage, dentin sensitivity, and recurrent caries. They are not used today for restorations, but have applications in orthodontics for bonding brackets, buttons, etc.

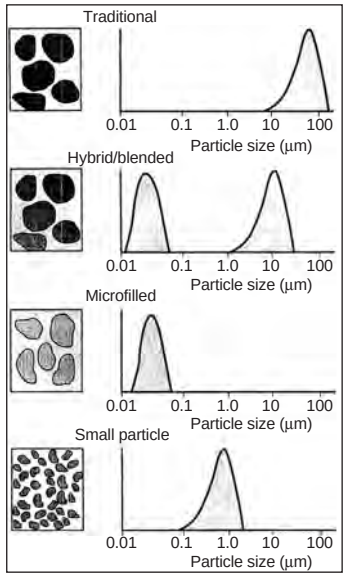


Figure 20.3. A classification of composites based on the partical size of the filler: macrofilled (traditional), hybrid, microfilled, small particle. (Redrawn from ref. 8, p. 111.)

In the 1970s the microfilled composites were introduced. The particle size in a microfill is uniformly small, 0.03–0.05 μm, which makes it polish like enamel (Fig. 20.3). The filler content is low at 40–50 per cent.[1,3,5,6,8] This increases the coefficient of thermal expansion and lowers the strength, but they are strong enough for use in low-stress areas. Their low modulus (of elasticity) makes them more flexible. They can be used to restore areas of enamel hypoplasia in the front of the mouth in areas that are not subjected to high biting forces.

The small-particle composites were developed in the 1980s in an attempt to find an acceptable composite in wear and strength that the microfills lacked. The particle size was 1–5 μm and the filler content was 80–85 per cent (Fig. 20.3).[1,3,5,6,8] Due to the range of particle sizes, the smaller particles fit in with the larger ones, requiring less resin. The resulting composites had less shrinkage, good wear and strength, and were esthetic. They were not as smooth as the microfill but were not nearly as rough as the macrofill. They were able to withstand occlusal forces and were therefore good for occlusal and interproximal restorations.

Late in the 1980s, the hybrid composites were developed. The range in particle size is from 0.5–1 μm and the filler content is 75–80 per cent by weight (Fig. 20.3).[1,3,5,6,8] They have the strength and the wear resistance of the small-particle composite. The surface finish mimics the microfill, but lacks the luster of the microfill due to the larger particle size. However, they are routinely used for esthetic restorations. The microhybrid is a blend of filler particles of the microfilled composite (~0.04 μm) and the hybrid composite (~0.5–3.0 μm). The smaller particles fit in between the larger particles, resulting in a filler concentration of 84 per cent by weight.[17] The result is a composite with the polishability of a microfill, and the strength and wear resistance of a hybrid (Fig. 20.4A,B).

In late 1996, a flowable composite was introduced.[18] The flowable composites are low-viscosity, light-cured composites that have been used successfully in areas of low stress. They have a very low modulus of elasticity, which makes them suitable for areas of flexion. They have high polymerization shrinkage, and low wear resistance because of their low filler content.[3,8,19] They are primarily used for liners under other restorative materials. Like the microfills, they can be used for restoring enamel defects in anterior teeth, but should not be used in areas of high wear.[18]

The most recent development is the introduction of the compactable composite. It was introduced after the flowable composite.[18] It is also referred to as a condensable or a packable composite. It can be placed in bulk like amalgam, has low polymerization shrinkage, and is more esthetic than amalgam. It has a low wear rate that is similar to amalgam. The compactable composite can be light cured at a depth greater than other composite resins.

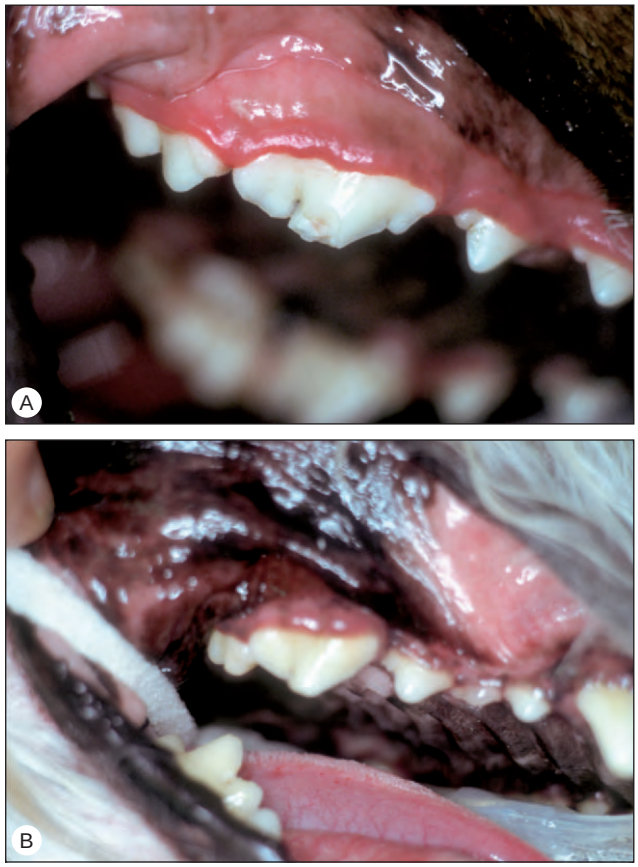


Figure 20.4. (A) Fractured crown with pulp exposure. (B) Fractured crown restored with a microhybrid composite resin after endodontic therapy (Filtek® Z250 – 3M ESPE).

The flowable composites and the compactable composites are not representative of advances in the properties of composite resins. They were developed in response to the proposed need for materials with special handling properties.

Glass ionomers were introduced in 1972. They are an adhesive material based on polyacrylic acid and powdered aluminosilicate glass. They set by an acid-base reaction, and chemically bond to dentin and enamel.[2] They release fluoride, which has a benefit as it is cariostatic.

The coefficient of thermal expansion of glass ionomers is close to tooth structure so there is minimal microleakage. In addition to chemical adhesion, the glass ionomers can be micromechanically retained through partially opened dentinal tubules that have been conditioned with polyacrylic acid. Initially the glass ionomers were chemically cured. In the late 1980s, resin was added to the glass ionomers to create a resin-modified glass ionomer that could be light cured. They had the advantage of free radical polymerization in addition to acid-base setting (Fig. 20.5).



Figure 20.5. Resin-modified glass ionomer (Vitrebond®). The addition of resin to the glass ionomer makes it light activated.

Glass ionomers are not strong and must be used in low-stress areas. They are opaque and not as good esthetically as composite restoratives. Therefore, their application is limited. They are brittle and are subject to fracture, they lack wear resistance, but they bond well to dentin. The type I glass ionomers are finely ground and are used for luting, or cementing, crown and bridges to teeth. Type II are used as restorative materials. The type III glass ionomers are used as a liner between materials such as the separation of root canal cement from a final composite restoration.[5]

Glass ionomers are of limited use in the equine patient due to their lack of strength when compared with the composites and compomers.

Compomers were introduced in 1995. They are a combination of a glass ionomer and a composite. They have a particle size of 0.8–5.0 μm and a filler volume of 42–67 per cent.[17] They are like a composite in that they have a resin matrix and bond to the tooth when used with the dentin bonding/adhesive system (Fig. 20.5). They are like glass ionomers in that they release fluoride, are not esthetic, and lack strength.[3,8,9]

A comparison of direct esthetic materials puts glass ionomers at one end of the spectrum with composites/dentin bonding systems at the other (Fig. 20.6).

Acrylic resins

Acrylic resins are used in dentistry for purposes other than tooth restoration. Acrylic resins set by polymerization as do the composite restorative materials with either heat or

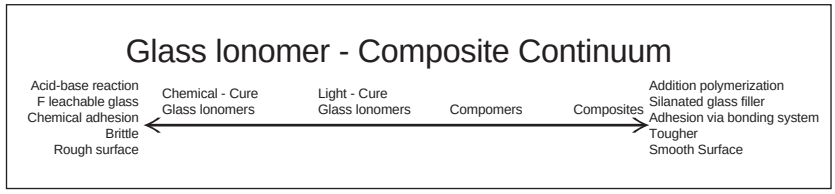


Figure 20.6. A comparison of direct esthetic restorative materials. (Modified from ref. 3, p. 67.)

chemical activation.[20] The initiator of the reaction is benzoyl peroxide. The heat-activated resins are generally used in dental labs to manufacture bases for full or partial dentures. Chemically activated acrylic resins are used to make custom trays, orthodontic appliances for our veterinary patients and their human owners, and temporary crowns.

In the equine patient, the acrylic resins have been useful in the manufacture of intra-oral splints for patients with Class II malocclusions (parrot mouth) (Fig. 20.7A).[11,14,15] The material is mixed and placed in the mouth to form a plate that covers the maxillary incisors and extends palatally to increase the area of contact by the lower incisors (Fig. 20.7B). This plate helps support the premaxilla during skeletal growth, thus decreasing the severity of the bony abnormality found in horses with this type of malocclusion (Fig. 20.7C).

The main problem with using acrylic resins in the mouth is the heat produced during polymerization. This has been addressed by using dental composite materials that are routinely used in human dentistry to make temporary crowns. They are very useful in veterinary dentistry to stabilize mandibular and maxillary fractures, and to orthodontically move teeth to correct malocclusions. They are non-exothermic, have a greater strength than some of the acrylics, are self-mixing, and are more viscous than the acrylics. This increase in viscosity makes them easier to place. ProTemp Garant® (3M ESPE), MaxiTemp® (Henry Schein), and Structure 2® (VOCO) are examples of these materials.

Dentin bonding agents and adhesive systems

The dentin bonding agents and adhesives used with dental composites are typically methacrylates (e.g. bis-GMA) and diluents. The bonding systems provide a means of bonding hydrophobic composites to hydrophilic dentine and the adjacent enamel.

An acid etching agent is used prior to the bonding agent. The acid opens the dentinal tubules by removing the smear layer and decalcifying the dentin surface layer. The dentin must remain moist to keep the interfibrillar spaces in the collagen network of dentin from collapsing during primer infiltration.[9,21] This is the concept of wet bonding (Fig. 20.8A,B).[22] Micromechanical bonds are formed from the flow of the dentin bonding agent (unfilled resin) into the dentinal tubules forming resin tags. In addition, a hybrid layer is formed by the interaction of the resin and the decalcified dentin. The composite resin is bonded to this hybrid layer interface (Fig. 20.9).

Many of the products used today combine the primer and adhesive in one bottle. The dentin and enamel are acid etched to remove the smear layer, after which the primer and adhesive are placed in one step. Examples of these products are One Step® (Bisco), Prime and Bond NT® (Dentsply Caulk), and Single Bond® (3M ESPE).

In 1999, the self-etching primer systems were introduced. They are an alternative to the conventional phosphoric acid treatment of dentin and enamel. The self-etching primers create a hybrid layer that incorporates the smear layer.[23] The etch and the primer are combined as a single agent. Examples of self-etching adhesive bonding systems are Clearfil SE Bond® (Kuraray) and Adper Prompt L-Pop® (3M ESPE).

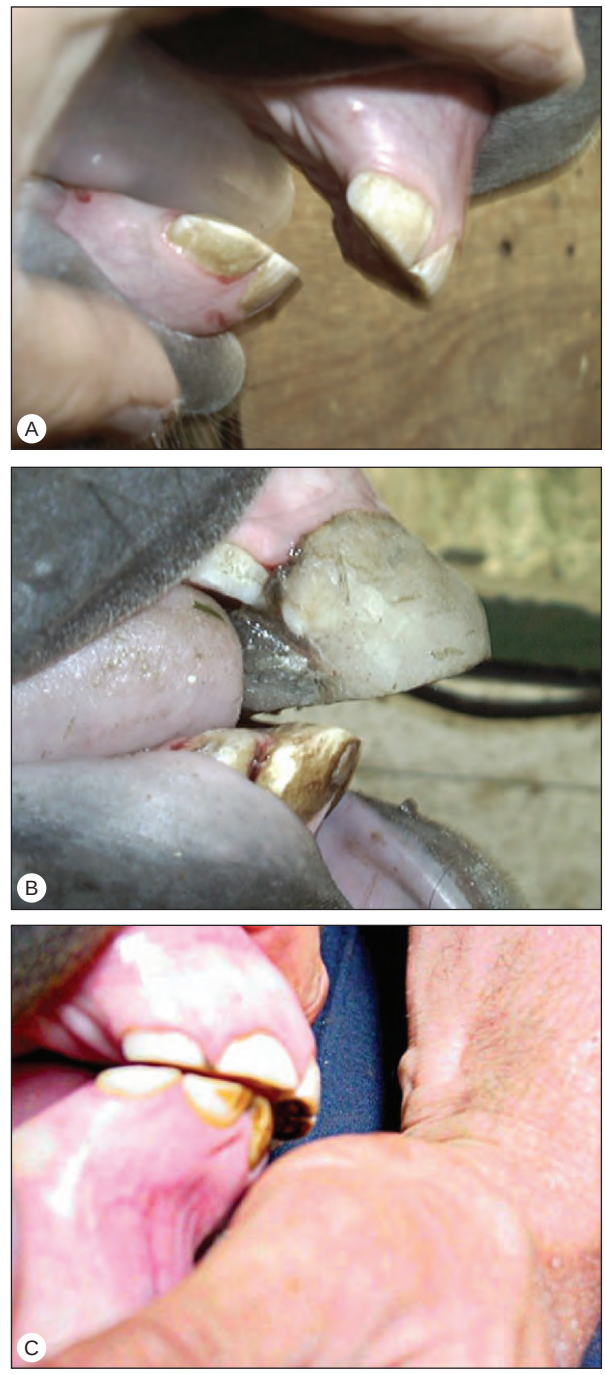


Figure 20.7. (A) Class II malocclusion (parrot mouth or mandibular brachygnathism) in a 5-month-old foal (courtesy of Dr David Klugh). (B) Acrylic resin appliance. The acrylic is placed directly on the maxillary incisors and extended palatelly. This facilitates contact of the rostral maxilla with the mandibular incisors (courtesy of Dr David Klugh). (C) Occlusion at age 26 months. The maxillary and mandibular incisors are in contact (courtesy of Dr David Klugh).

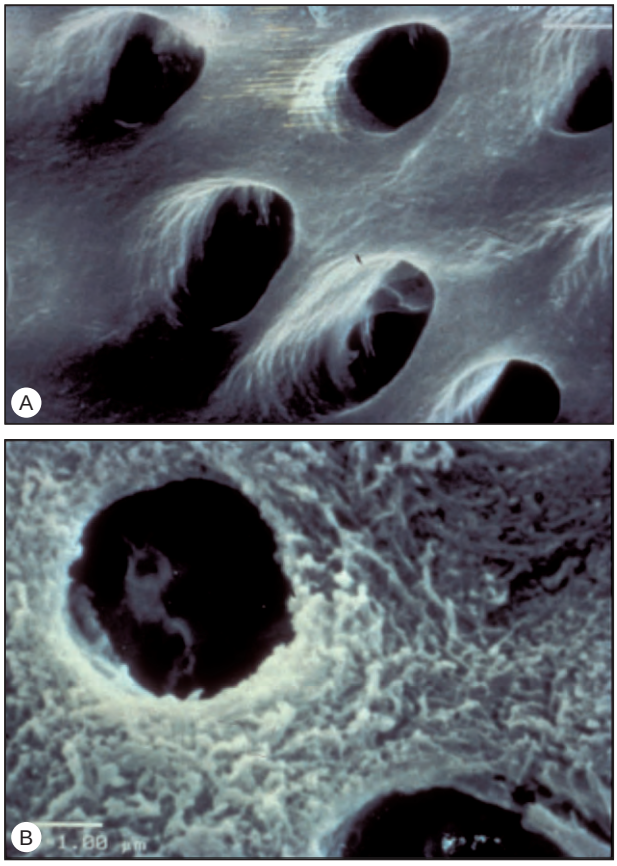


Figure 20.8. (A) Demineralized dentin after air drying. The collagen fibers are collapsed as a result of desiccation. (B) Demineralized dentin that has been kept moist. The collagen structure is preserved. (From ref. 8, pp. 160–161.)

Liners and bases

Liners and bases are materials that are used between cement or composite and the tooth. Liners decrease irritation to the pulp–dentin complex from cements and composite resins, but do not provide thermal insulation.[5] Bases, on the other hand, protect the pulp when a layer of approximately 0.75 mm is placed between the pulp and the cement. Bases also provide thermal insulation. Examples of bases are CaOH (calcium hydroxide), polycarboxylic acid, are glass ionomers. Liners and bases are not recommended for routine use as they interfere with bonding agents.

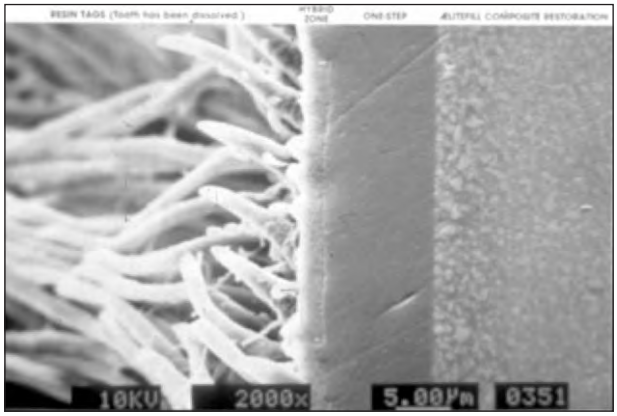


Figure 20.9. The bonded composite resin. Resin tags are formed from the flow of the dentin bonding agent (unfilled resin) into the dentinal tubules. A hybrid layer is formed by the interaction of the resin and the collagen fibers of the decalcified dentin. The composite resin is bonded to this hybrid layer interface (Fig. 20.8). (From ref. 3, p. 48.)

Cements

Cements are used to affix prosthetics on prepared teeth. Full gold crowns, porcelain fused to metal crowns, and post and cores are cemented to the prepared tooth in human and small animal patients.

Zinc phosphate cement consists of zinc oxide and phosphoric acid (Fig. 20.10).[10] It is rigid, has high compressive strength and acts as a thermal insulator. The low pH (pH 3) may be irritating to the pulp in a vital tooth. Zinc phosphate cement bonds mechanically to the tooth and not chemically.

Polycarboxylate cement (Durelon®, 3M ESPE) is zinc oxide and polyacrylic acid (Fig. 20.10).[10] This cement has half the compressive strength of zinc phosphate, has poor rigidity, and has a high solubility in water. It is potentially less irritating to the pulp, much like the glass ionomers. Polycarboxylate cement bonds to the calcium structure of enamel and to the dentin.

Reinforced zinc oxide eugenol (IRM®, Dentsply Caulk) is the least irritating of the dental materials due to its neutral pH (pH 7). When used with composite resins, it must be separated from the composite material with a liner since the eugenol interferes with polymerization.

The zinc phosphate and polycarboxylate cements have limited use today as the trend has shifted to the composite resin cements and the glass ionomer cements.

Glass ionomers are an aluminosilicate glass and polyacrylic acid (Fig. 20.10),[9,10] as discussed previously. They are similar to the silicate cements but the alumina-to-silicate ratio is higher in the glass ionomer material. They have an even higher compressive strength than zinc phosphate, but are less rigid, and they are not irritating to the pulp. They are used for luting, or cementing, posts and crowns to teeth.[5] Examples are Fuji® (GC America), Ketac® Cem (3M ESPE), and RelyX® Luting (3M ESPE).

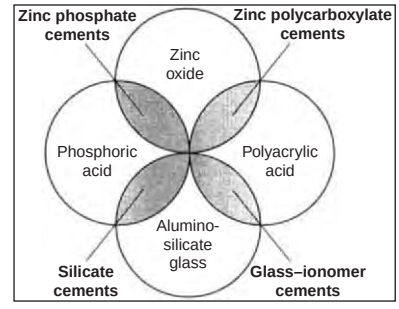


Figure 20.10. Basic components of dental cements. The powders are either zinc oxide or aluminosilicate glass. The liquids consist of phosphoric acid or polyacrylic acid. (Redrawn from ref. 8, p. 125.)

Composite resin cements are composed of dimethacrylate resins and glass fillers, as discussed previously. The resin cements are insoluble in water, are light- or dual-cured, and have high bond strengths to tooth structure.[17] Examples are Panavia F® (Kuraray) and RelyX® UniCem (3M ESPE).

EBA (orthoethoxybenzoic acid) has low compressive strength and high solubility in water similar to polycarboxylate cement. It is not irritating to the pulp however, and is a material that can be used in a wet field. SuperEBA® cement (Bosworth Co.) is a product that has grown in popularity as a filling material for root end surgical endodontics.[11]

Mineral trioxide aggregate (MTA) is a calcium silicate material (ProROOT MTA®, Dentsply Tulsa Dental) similar to Portland cement. It was introduced in 1998 and seems to be an ideal root end filling material. It is biocompatible, hydrophilic so it can be used in a moist field, and has excellent sealing capabilities.

Impression materials

Impression materials are used in dentistry to make a mold of the teeth and their surrounding structures which is then used to create a model. This can be a basic study model to evaluate occlusion, wear patterns, orthodontic considerations, etc. or can be a highly accurate model from which crowns and appliances are made. The model can be made of stone, plaster, or other similar materials. The type of model needed dictates which impression material to use.

The characteristics of an ideal impression material are accurate detail of the teeth and soft tissue, no distortion when removed from the mouth, and maintenance of dimensional stability before and during being cast into a model.

Impression materials are either inelastic or elastic. Generally, the elastic impression materials are used in veterinary dentistry. The two categories of elastic impression materials are the hydrocolloids and the synthetic elastomers.

The hydrocolloids are used for partial or full mouth impressions for study models or appliance manufacture. They are categorized as either reversible or irreversible. These materials do not have the dimensional stability of the elastomers. For the purpose intended they are practical and inexpensive. The reversible hydrocolloid is an agar. It is heated to a fluid state with a low viscosity. When it cools it becomes a gel. Agar is very accurate under the correct circumstances. The irreversible hydrocolloid is alginate. It is a soluble salt that reacts with calcium sulfate to become a gel of insoluble calcium ions. It is inexpensive, easy to use and generally appropriate for the veterinary patient.[5,6,8,24] The exception is when taking impressions for prosthodontics where greater detail is required. In this situation the elastomeric compounds are indicated.

The elastomeric impression materials are synthetic rubber and were originally referred to as rubber impression materials. These materials are composed of polymers that are cross-linked. There are four kinds of elastomeric impression materials that fall into two categories. The categories are defined by the type of reaction that occurs during polymerization. The addition-cured materials form a polymeric chain that lengthens in sequence without producing other chemical reactions. The addition-cured materials include vinyl polysiloxane and polyether rubber. The condensation-cured materials polymerize and produce a reaction by-product that is not part of the polymer. They include polysulfide rubber and condensation silicones.

The polysulfide rubber materials utilize a base paste and an accelerator that produce water as a by-product. This by-product contributes to continued polymerization shrinkage resulting in poor dimensional stability.[5,6,8] The material is inexpensive and includes products such as Coe-Flex® (GC America) and Permalastic® (Kerr).

The condensation-cured silicones have a by-product of ethyl alcohol, which leads to high polymerization shrinkage from evaporation. This results in poor dimensional stability like the polysulfide rubber materials. The silicones include Accoe® (GC America).

The polyether rubbers are a base paste and an accelerator. They provide excellent detail, have very good dimensional stability, and moderate tear strength.[5,6,8,24] They can be stored for 7 days but then start to absorb water that results in swelling, thus decreasing dimensional stability. It is more expensive than the polysulfides but less so than the vinyl polysiloxanes. An example of a polyether rubber is Impregum F® (3M ESPE).

The vinyl polysiloxanes are a base and catalyst paste, some of which are self mixing. They have excellent detail and dimensional stability, and high tear strength.[5,6,8,24] They can be stored for up to 2 weeks without affecting detail and stability. They are expensive but are the materials of choice for area specific impressions for crown manufacture in veterinary dentistry. There are many materials in this category some of which include Express® and Imprint® (3M), President (Coltene/ Whaledent), and Reprosil® (Dentisply Caulk).

The inelastic materials most commonly used in veterinary dentistry are impression waxes for a bite registration.[5]

Dental stone

Dental plasters and stone are used to make positive models of the teeth and soft tissue from the negative impression that was taken. Plasters and stone are made of calcium sulfate crystals.[20] The configuration of the crystals dictate the amount of water required to form dihydrate crystals. The more regular the crystal the less water is needed, which results in a model with greater compressive strength. Models made of dental stone are more resistant to abrasion than models made of plaster of Paris.

Periodontal materials

Periodontal disease is prevalent in all mammalian species. Periodontal disease results in destruction of the periodontium. The periodontium comprises the tissues that support the

teeth – the gingiva, the periodontal ligament, the alveolus, and the cementum. The accumulation of bacterial plaque on the teeth causes gingivitis. With the interplay of the host response, this condition can progress to periodontal disease.[4,5,11,13,25]

The goal in prevention of periodontal disease is to control the bacterial plaque. In the small animal patient, preventive measures are geared toward the daily mechanical removal of plaque coupled with regular professional dental cleaning. Mechanical plaque removal is achieved by daily tooth brushing, feeding dry diets that are geared toward plaque disruption, and encouraging the regular use of appropriate chew toys.

In the equine patient, the bacterial destruction of the periodontium results from the stasis of feed material. The feed is lodged in the interdental spaces and as it decomposes it produces by-products. Microbes and these by-products attack the periodontium and cause loss of root cementum, alveolar bone, and gingival attachment. In addition, many of these patients suffer from masticatory difficulties from lack of occlusal equilibration.[13–15] In the ideal world, prevention is preferable. Once the disease is established, the goal of treatment is to halt or slow its progression.

Some cases of periodontal disease require systemic antibiotic therapy. In many circumstances, however, we can utilize local or topical antimicrobial therapy.[26,27] We can deliver an antibiotic directly to the gingival sulcus of the diseased area, utilizing a flowable antibiotic solution, or use oral antibacterial solutions like chlorhexidine to aid in bacteria control.[28] A flowable doxycycline solution introduced directly into the periodontal pocket has a local antibacterial effect for up to 2 weeks. The concentration of doxycycline in the gingival sulcus is far greater than that achieved with systemic antibiotics. It is biodegradable so does not need to be removed from the gingival sulcus. This local antibacterial effect provides the right environment for the attachment of junctional epithelium from the soft tissue of the lining of the gingiva to the cementum. Doxirobe® (Pfizer) contains 8.5 per cent doxycycline. This product was developed for the veterinary patient. Atridox® (Atrix Laboratories) is the human counterpart and contains 10 per cent doxycycline.

Oral chlorhexidine solution (CET Oral Hygiene Rinse®, Virbac) is a broad-spectrum antibacterial agent that is very effective in reducing the overall concentration of oral bacteria.[5,25,28] It is locally effective, is not absorbed from the stomach when swallowed, does not cause gastric irritation, and is slowly released from the oral mucosal tissues for up to 12 hours. The concentration of chlorhexidine in these products is typically 0.12 per cent. Peridex® (Zila, Inc.) was the product first developed for the human patient. This is another way to avoid the overuse of systemic antibiotics.

The adherence of oral bacteria to the tooth surface may be reduced by applying a wax barrier. ProVSeal® (Kepa Corp.) is used in small animal dentistry. It is a hydrophobic wax that acts as a deterrent for colonization of the tooth surface by the hydrophilic bacteria. It is applied initially when the patient is under anesthesia and is then applied weekly by the pet owner. The wax is recharged with the less viscous weekly product. Daily tooth brushing, dental diets, and chew toys will not deplete the wax barrier. ProVSeal does not take the place of routine homecare techniques, but is used in addition to homecare.

The patterns of bone loss in advancing periodontal disease can be vertical, horizontal or a combination of the two. It is appropriate to consider bone replacement in some cases. Materials for bone replacement either stimulate new bone formation and incorporate into the bone (osseous induction) or act as scaffolding for regeneration of bone (osseous conduction). Osseous inductive materials are generally autogenous bone. The bone must be harvested from the patient to be placed in the bony defect. This is not always practical or convenient. Bone morphogenic protein (BMP) from decalcified freeze-dried bone allografts has osteogenic effects similar to autogenous bone. The demineralization process exposes components of the bone matrix that are osteogenic.[29] Osseous conductive materials are composed of hydroxyapatite, and various forms of glass and ceramics. Consil® (Nutramax Laboratories Inc.) is a glass bead polymer that is widely used in veterinary dentistry, as is Bioplant HTR® (Bioplant Inc.).

There are many new materials on the market that are reported to be osseous inductive. PepGen P-15® (Dentsply Friadent Ceramed) is the only material at present that stimulates new bone and is incorporated into the new bone. It is a bioengineered type-1 collagen from bovine that mimics the components of autogenous bone. The other materials are conductive in that they stimulate new bone formation. Emdogain Gel® (Biora Inc.) is an enamel matrix protein from porcine, Osteograf/N® (Dentsply Friadent Ceramed) is a bovine material, and AlloGro® (Dentsply Friadent Ceramed) is a decalcified freeze-dried bone allograft.

There are many materials available for those circumstances where surgery is necessary to halt the progression of disease in an attempt to regenerate lost bone.

Summary

As veterinary dentistry developed in the small animal patient in the 1970s it was imperative that we studied and understood the use of dental materials in the human patient. With this knowledge we were able to extrapolate and use materials in ways other than the way they were intended for use in the human.

We have found that temporary crown materials can be used for intra-oral splints in horses, dogs, and cats for the purpose of orthodontics and fracture stabilization. The periodontal materials serve the same purpose in our companion animals as they do in humans. Those materials that are biodegradable are far superior to those that need to be removed, as our patients require sedation or general anesthesia for placement and removal.

Steps were taken to adapt dental techniques, materials, and instruments used for the human patient to our small animal patients. Similar steps are now being taken in developing equine dentistry.

REFERENCES

1. Anusavice KJ (1996) *Phillip's Science of Dental Material* (10th edn). WB Saunders.
2. DuPont G (2001) The bonded composite restoration. In: *Proceedings 15th Annual Veterinary Dental Forum,* pp. 57–60.
3. Gladwin M and Bagby M (2000) *Clinical Aspects of Dental Materials*. Lippincott, Williams and Wilkins.
4. Harvey CE and Emily PP (1993) *Small Animal Dentistry.* Mosby-Year Book, St Louis.
5. Wiggs RB and Lobprise HB (1997) *Veterinary Dentistry: Principles and Practice.* Lippincott-Raven, Philadelphia.
6. Leinfelder KF and Lemons JE (1988) *Clinical Restorative Materials and Techniques*. Lea & Febiger, Philadelphia.
7. Oakes AB (1994) Amalgam in veterinary dentistry. *Compendium of Continuing Education for the Equine Practitioner*, 16(12), 1531–1538.
8. van Noort R (2002) *Introduction to Dental Materials* (2nd edn). Mosby, St Louis.
9. DuPont G (1998) Modern restorative materials. In: *Proceedings 12th Annual Veterinary Dental Forum,* pp. 41–44.
10. Nakabayashi N and Pashley DH (1998) *Hybridization of Hard Tissues.* Quintessence Publishing Co.
11. Baker GJ and Easley J, eds (1999) *Equine Dentistry.* WB Saunders, London.
12. Baker GJ and Allen M (2000) Infundibular embryology, morphology, pathology and clinical signs in horses. In: *Proceedings 14th Annual Veterinary Dental Forum,* pp. 296–297.
13. Gaughan EM and DeBowes RM, guest eds (1998) Dentistry. *Veterinary Clinics of North America: Equine Practice*, 14(2).
14. Honnas CM and Bertone AL, guest eds (1993) The equine head. *Veterinary Clinics of North America: Equine Practice,* April.
15. Kertesz P (1993) *A Colour Atlas of Veterinary Dentistry and Oral Surgery.* Wolfe, London.
16. Brannan R (2000) Restorative dentistry. In: *Proceedings 14th Annual Veterinary Dental Forum,* pp. 74–78.
17. Craig RG, Powers JM and Wataha JC (2000) *Dental Materials Properties and Manipulations* (7th edn). Mosby, St Louis.
18. Bayne SC, Thompson JY, Swift EJ and Stamatiades P (1998) A characterization of first-generation flowable composites. *Journal of the American Dental Association,* 129(5), 567–577.
19. Labella R, Lambrechts P, VanMeerbeek B and Vanherle G (1999) Polymerization shrinkage and elasticity of flowable composites and filled adhesives. *Dental Materials,* 15(2), 128–137.
20. Phillips RW (1984) *Elements of Dental Materials for Dental Hygienists and Assistants* (4th edn). WB Saunders, Philadelphia.
21. Brannan R (1998) Dentin bonding agents and adhesives. In: *Proceedings 12th Annual Veterinary Dental Forum,* pp. 91–93.
22. Kanca J (1992) Resin bonding to wet substrate I. *Quintessence International,* 23, 39–41.
23. Tay FR, Carvalho R, Sano H and Pashley DH (2000) Effect of smear layers on the bonding of self-etching primer to dentin. *Journal of Adhesive Dentistry,* 2(2), 99–116.
24. Verstraete FJM (1999) *Self-Assessment Color Review of Veterinary Dentistry* (4th edn). Manson Publishing, London and Iowa State University Press, Ames.
25. Carranza FA and Newman MG (1996) *Clinical Periodontolgy* (8th edn). WB Saunders, Philadelphia.
26. Walker C (1999) The supplemental use of antibiotics in periodontal therapy. *Compendium on Continuing Education in Dentistry,* 20(4 Suppl), 4–12.
27. Newman MG and van Winkelhoff AJ (2001) *Antibiotic and Antimicrobial Use in Dental Practice.* Quintessence Publishing.
28. Killoy WJ (1999) Local delivery of antimicrobials: a new era in the treatment of adult periodontitis. *Compendium on Continuing Education in Dentistry,* 20(4 Suppl), 13–18.
29. Emily P (2003) Bone augmentation. In: *Proceedings 8th World Veterinary Dental Congress,* Kyoto, Japan, pp. 132–135.

Manufacturers

3M ESPE, St Paul, MN
Atrix Laboratories Inc., Ft Collins, CO
Bioplant Inc., South Norwalk, CT
Biora Inc., Malmö, Sweden
Bosworth Co., Skokie, IL
Coltene/Whaledent, Mahwah, NJ
Dentsply Caulk, Milford, DE
Dentsply Friadent Ceramed, Lakewood, CO
Dentsply Tulsa Dental, Tulsa, OK
GC America, Alsip, IL
Henry Schein Inc., Melville, NY
Kepa Corp., Upland, CA
Kerr, Romulus, MI
Kuraray Medical Inc., Okayama, Japan
Nutramax Laboratories Inc., Baltimore, MD
Omni, Gravette, AR
Pfizer Animal Health, Exton, PA
Virbac Corp., Fort Worth, TX
VOCO, Cuxhaven, Germany
Zila Inc., Phoenix, AZ

21 Mandibular and Maxillary Fracture Osteosynthesis

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Introduction

Fractures of the mandible and maxilla are commonly encountered in equine practice.[1,2] Many of these fractures tend to be open and some are comminuted. Despite this, excellent blood supply and the fact that the mechanical forces placed on these bones are not excessive allow relatively fast healing when compared with fractures of the distal limb. The goals of repair are early return to normal alimentation, resolution of infection, and cosmesis. Multiple options have been utilized in resolution of these injuries including conservative management, wire fixation with or without addition of intra-oral splints, internal fixation and external skeletal fixators. Although the complication rate for mandibular and maxillary fractures is high, the long-term prognosis is generally favorable.[3]

Fractures of the mandible may be categorized by anatomic location including the incisive portion of the body, the interdental space, the molar portion of the body and the vertical ramus, including the temporomandibular joint and coronoid process.[4] Fractures of the mandible rostral to the cheek teeth represent up to two-thirds of fractures,[3] while fractures of the ramus are rare.[5,6]

Incidence

The mandible is reported to be the most commonly fractured bone of the horse.[1] The inquisitive nature of the horse and the use of the lips and nose to gather information about the environment predispose the horse to trauma of the upper and more commonly the lower jaw. The rostral mandible is not protected by large amounts of soft tissue, rendering the bone more susceptible to trauma. Fractures of the mandible on solid objects or, in cases of rostral fractures, catching the incisors on stationary objects and pulling back are common scenarios.[1,5,7] Compression fractures can result from a kick or from the horse running into a stationary object.[3] In a recent review of 67 cases of mandibular and maxillary fractures in horses presented to the Texas A&M University Large Animal Clinic over the last 5 years, the most common etiologies included kicks from other animals, becoming entangled in a fence, running into a fence, and pulling back with incisors on a stationary object.[8] Of these cases 61 per cent occurred rostral to the premolars. Only two of the 67 cases involved the vertical ramus of the mandible. There was no significant age difference seen between rostral and caudal fractures with the average age of each group being 5 years. Pathologic fractures of the mandible can occur secondary to progressive dental disease with infection and tumors.[9] Fracture of the molar portion of the mandible is a possible complication of surgical repulsion of the premolars and molars.

Anatomy

The equine mandible is composed of two halves that are mirror images of one another. Each hemimandible contains a horizontally oriented body and a vertically oriented ramus. The body is composed of two parts. Rostrally the incisive portion contains the incisor teeth and caudally the molar portion contains the premolar and molar teeth. The interdental space lies between the third incisor tooth and the second premolar tooth. The vertical ramus contains the coronoid and condylar processes at its dorsal aspect (Fig. 21.1). The condylar process comprises the mandibular portion of each temporomandibular joint and articulates with the squamous portion of the temporal bone via an interposing articular disk. Fusion of the two halves of the mandible occurs along the mandibular symphysis at 2–3 months of age.

The mandibular alveolar nerves enter the mandibular foraminae on the medial aspect of each vertical ramus. The mandibular alveolar nerves course through their respective mandibular canals, ventral to the mandibular cheek teeth, innervating the teeth and gingiva.[10] Each mandibular alveolar nerve is accompanied by a mandibular alveolar artery.

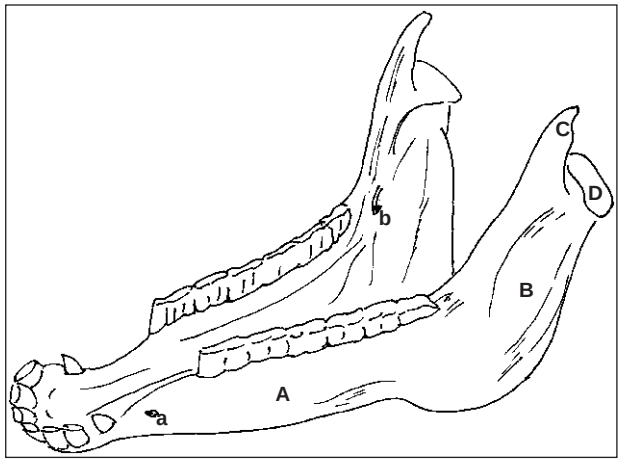


Figure 21.1. Anatomy of the mandible. A – body; B – vertical ramus; C – coronoid process; D – condylar process; a – mental foramen; b – mandibular foraminae.

The mandibular alveolar nerves bifurcate, giving rise to the mental nerves which emerge from the mental foramina, and the inferior alveolar nerves which course rostrally in the incisive portion of the mandible and innervate the canine and incisor teeth and gingiva.[11]

Overlying the lateral aspects of the mandible, the facial artery, facial vein and parotid salivary duct course from medial to lateral across the ventral surface of the bodies of the mandible and turn to course dorsally over the lateral aspects of the mandible along the rostral margin of the masseter muscles. The facial nerve courses rostrally across the lateral aspects of the face, approximately at the level of the cheek teeth. Ventral to the eye the facial nerve divides to supply motor innervation to the ipsilateral upper and lower lips.

Related anatomy of surgical concern on the ventral aspect of the mandible includes the mandibular lymphocenter and the facial artery, facial vein and parotid salivary duct. These structures course rostrally along the medial aspect of each hemimandible before turning to run laterally and dorsally along the rostroventral margin of the masseter muscles.

The mandible changes in shape as the mandibular teeth erupt. The incisive and molar portions of the body of the mandible are thicker in the young horse and thin progressively as reserve crown is reduced as the teeth erupt.[12]

Patient evaluation and decision-making

Clinical signs exhibited by affected horses vary with the site of mandibular fracture. Oral pain is typically manifest by difficulty or unwillingness to eat. Ptyalism, halitosis and incisor malalignment are the most common clinical signs (Fig. 21.2).[1,13] Fracture of the incisive portion of the mandible may be overlooked due to lack of overt external signs (Fig. 21.3). The majority of rostral mandibular fractures result in avulsion of teeth with subsequent impaction



Figure 21.2. Appearance of a foal that sustained a fracture of the mandible through the interdental space of each hemimandible. Because of ventral deviation of the rostral mandible resulting in inability to occlude the incisors, the tongue continually protrudes out the mouth. Neurologic function of the tongue is normal.

of feed and debris in the fracture gap, which is accompanied by foul odor. Fractures of the interdental space are commonly bilateral and result in instability of the rostral mandible that is readily palpable (Fig. 21.4). Bilateral fractures tend to be more common in younger horses.[3,8] Hemorrhage from the oral cavity may be present. Clinical signs of facial swelling, soft-tissue trauma, ptyalism, dysphagia, weight loss and malalignment of teeth should prompt an investigation for the presence of a caudal mandibular fracture.[4] Horses with disease of the temporomandibular joints may exhibit external swelling, pain upon palpation of the temporomandibular joint, unwillingness to open the mouth, crepitation, rostral or caudal displacement of the mandible, and malocclusion of the caudal teeth, preventing apposition of the incisors. Oral examination should always be performed and will enable diagnosis of rostral mandible and interdental space fractures (Fig. 21.3). Mucosal laceration, broken, loose or displaced teeth and malocclusion are oral signs indicating that mandibular fracture should be suspected. Although temporomandibular joint injuries are uncommon, palpation of joints is advisable.

Radiography is indicated for fractures caudal to the incisor teeth. Radiography of rostral fractures involving only the incisors may not be indicated in every case based on findings of a physical examination. Lateral, dorsoventral and oblique views allow fracture definition and comparison with the



Figure 21.3. Fracture of the incisive portion of the mandible. The horse looked normal on clinical presentation and had no externally obvious clinical signs that a fracture had occurred.

contralateral portion of the mandible. Intra-oral placement of radiographic cassettes will allow better definition of rostral mandibular fractures. Improved definition of the condyle and coronoid process of the ramus is obtained by taking a lateral projection with the mouth held open with a speculum.[2,14] Fractures in the molar portion of the mandible can be difficult to define radiographically due to superimposition of tooth roots. Computed tomography, if available, may be useful in characterizing these fractures in the caudal mandible, especially if internal fixation is being considered.[2] Positive contrast fistulography may be performed in the evaluation of fractures associated with draining tracts.[2]

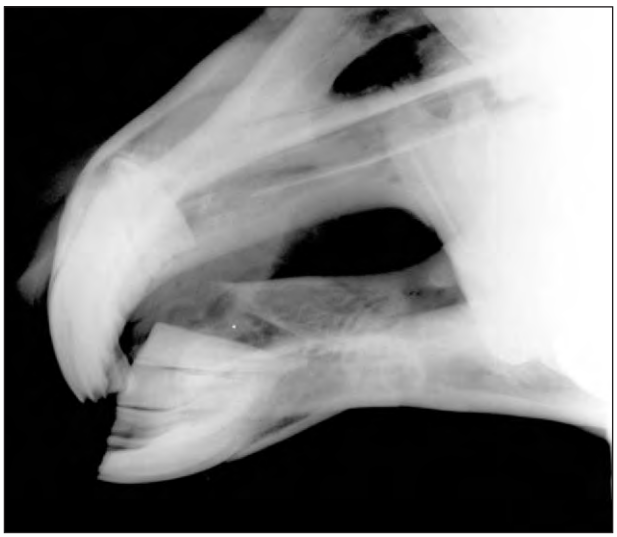


Figure 21.4. Lateral radiographic projection of a bilateral fracture of the mandible through the interdental space resulting in ventral deviation of the mandibular incisors.

The overall health of the horse and cardiovascular status should be assessed, especially in horses with fractures that cause inability or unwillingness to eat or drink. Evaluation should include complete assessment of the horse's neurological status and determination of any airway or ophthalmologic compromise. Fixation of mandibular fractures can be delayed with little impediment to healing so as to allow stabilization of the horse with fluids. Administration of antimicrobials, non-steroidal anti-inflammatory drugs, and tetanus prophylaxis is indicated. Penicillin is a suitable antimicrobial for most fractures. Broader spectrum antimicrobials are indicated when internal fixation is used. The decision whether to complete fracture repair in the standing sedated horse with local anesthesia or under general anesthesia is dependent on the site and character of the fracture and temperament of the horse. Most incisive fractures as well as some fractures of the interdental space can be reduced effectively in the standing horse.

The method chosen to repair mandibular and maxillary fractures is dependent on the horse's temperament and the anticipated stress they will place on the repair. Additionally, fracture configuration, degree of contamination, age of the horse, and time interval between occurrence of the fracture and repair should be considered. Simple fractures of the rostral mandible and maxilla that are easily reduced can be repaired satisfactorily with intra-oral wiring. Rostral fractures that are unstable or have severe comminution are candidates for intra-oral splinting or some type of external fixator. Internal fixation has limited utility in rostral fractures due to the contaminated nature of many of these injuries and the presence of reserve crown, which precludes screw placement.

Fractures involving alveoli can result in infectious periodontitis and pulpitis, necessitating removal of the tooth. The decision to remove affected teeth should be deferred until the fracture has healed.[5] Premolar and molar teeth may lend stability to the mandible during healing, compared with an empty alveolus. Detached and broken teeth are candidates for removal.[1] Loose incisors may be wired in place and can be used to bolster repairs in some cases.[15] Exposed permanent teeth that are unerupted should be left in place and manipulated as little as possible. If viability of a tooth is in question, it may be left in place and removed, if necessary, after bony healing has occurred.[1]

Fractures involving the incisive portion of the mandible or maxilla

Fractures of the rostral incisive portion of the mandible and maxilla are common and amenable to fixation in most

settings. Evaluation and fixation of these fractures may be completed in the standing sedated horse. Anesthesia of the mandibular alveolar nerves at either the mandibular alveolar foramina or the mental foramina will facilitate debridement and fixation of these fractures in the standing horse.

Oral examination reveals the extent of the fracture and teeth involved. 'Corner fractures' (Fig. 21.5) running from midline near the first or second incisor to the area of the ipsilateral third incisor are common. Radiographs can be used to define fractures of the incisive portion of the mandible but are often not necessary in most cases.

Following sedation and local or general anesthesia, therapy of open fractures begins with cleansing and debridement of the fracture line. Attention to debridement of the fracture line will facilitate reduction.[15] Feed, blood clots and bone fragments are removed from the fracture and a dilute antiseptic solution (0.01 per cent povidone iodine or 0.05 per cent chlorhexidine) is used to thoroughly lavage the wound. Alignment of the incisor occlusal surfaces is used as a guide during reduction of the fracture. Alignment of the fracture may be maintained by an assistant during surgical fixation or by wiring the incisors of the incisive bone and the mandible together.[5] Lacerations and tears of the gingival mucosa may be sutured using polypropylene or polydioxanone in a simple continuous horizontal mattress pattern, or allowed to heal by second intention.

Wire fixation is widely used to stabilize rostral mandibular fractures involving the incisors[3,8] Wire fixation carries the advantage of providing a stable fixation along the tension surface of the mandible and is less complicated to perform than other repair methods. The risk of chronic infection is less with wire fixation than when internal fixation is utilized. A minimum of one wire is used on each side of the fracture with overlapping of the wires.[15] Exact placement of wires is dependent on the configuration of the fracture. Holes are placed between the incisive teeth at the gingival margin



Figure 21.5. Dorsoventral x-ray projection of a rostral incisor ('corner') fracture.



Figure 21.6. Cannulation of predrilled holes with a 14 gauge needle facilitates wire placement when repairing rostral maxillary or mandibular fractures.

using a Steinman pin or 3.2 mm drill bit. Cannulation of the holes with a 14 guage needle facilitates placement of the wire between teeth (Fig. 21.6). Fourteen gauge needles may be used in young horses without prior drilling of holes. The occlusive surfaces of the incisors are aligned to attain proper fracture reduction.[5] Stainless steel wire, 1.2 mm or larger, is placed through the holes, twisted together on the labial surface of the mandible and bent down into the gingiva. Fencing pliers are useful for tightening the wires. Wires should be pulled away from the gingiva as they are twisted together to avoid wire breakage during the tightening process. Notches may be cut into the caudal margin of the third incisor teeth or canine teeth to allow the wire to be anchored around these structures. Holes may be drilled through the mandible in the interdental space, permitting wire placement from the incisors in a caudal direction (Fig. 21.7).[5] Screws placed in the diastema of the mandible can also serve as anchors for wires.[9] Suturing mucosal lacerations prevents continued gross contamination of the fracture site but is not necessary in most cases.

Fractures of the rostral mandible involving the incisors can propagate caudally into the interdental space, creating large bone fragments. A combination of wire fixation and pins or cortical bone screws is indicated in these instances to provide stability that cannot be obtained with wire fixation alone.[13,16] Following preparation of the fracture, reduction is achieved and may be maintained with reduction forceps during implant placement. Pins or cortical bone screws placed in lag fashion are positioned strategically to provide compression across the fracture line.[16] Stab incisions are made in the gingiva and a drill guide is used to protect surrounding soft tissues. Care should be taken during curettage of open fractures and while placing implants avoid tooth roots.[16] The size and configuration of the fragments will dictate the number of implants used. Concurrent wire fixation is used as needed to counter rotational instability.[16]



Figure 21.7. Repair of the corner fracture in Fig. 21.3 was accomplished with wire placed on either side of the fracture. This photograph was taken 6 weeks postoperatively when the horse presented for removal of wire implants. Healing was uncomplicated with a good cosmetic result.

After fixation of rostral mandibular incisive fractures the horse may be fed hay and grain. Hay should be pulled apart so the horse does not have to prehend with the repaired teeth. Grazing grass or eating other feeds requiring cutting with the incisors should be avoided until the fracture has healed. Daily examination of the wires is indicated to identify loose or broken wires. Wire loosening or breakage was reported in 22 per cent of cases in one study.[3] The mouth may be rinsed with water to remove feed material trapped in wires or to lavage open wounds. Wires are removed from the oral cavity 4–8 weeks postoperatively. The prognosis for healing of these fractures is good to excellent. The ultimate cosmetic quality of the dentition is dependent on the damage that has occurred to the teeth and permanent tooth buds during the original injury.

Fractures of the interdental space

Fractures through the interdental space commonly involve the left and right sides of the mandible, causing instability of the rostral aspect of the mandible.[4] Fractures of the maxillary interdental space can also occur but are not as commonly encountered, often with significant comminution.[3] A full radiographic series, including dorsoventral, lateral and oblique views, is indicated to delineate the fracture and identify involvement of adjacent teeth and the presence of osseous fragments. Fractures of the mandibular interdental space are most often transverse or short oblique in configuration with minimal comminution.[2] Bilateral fractures are common in horses less than 1 year and unilateral fractures of the interdental space are more commonly seen in older horses.[8] Traumatic fractures of the premaxilla may have concurrent injury to the nasal septum, nasal process, or facial bones.[8,15]

Fixation of fractures involving the interdental space is performed under general anesthesia. Intravenous anesthesia or nasotracheal intubation increases space for the surgeon to work in the oral cavity and is recommended as it permits the surgeon to access both sides of the mandible. Methods of fixation of fractures of the interdental space include tension-band wiring, oral acrylic splints, U-bars, external fixators and bone plating.[1,3–6,9] Unilateral fractures which are minimally displaced, not causing pain or inappetance, may respond well to conservative therapy.[3,8,9]

Fractures of the interdental space that are not significantly displaced and have sufficient interdigitation at the fracture line, limiting movement, may be repaired using tension-band wiring.[2,4–6] The tension surface of the mandible lies along the dorsal border (oral surface) allowing tension-band wiring to be successful when minimal displacement is present. The use of wires does not require specialized equipment, is inexpensive and decreases the risk of osteomyelitis with open contaminated fractures.

The horse is anesthetized and placed in dorsal recumbency and the oral cavity, left and right external cheek surfaces are prepared for surgery; holes are drilled between mandibular or maxillary incisors as described above. Stab incisions are made bilaterally through the cheek over the space between the second and third mandibular premolar teeth. Hemorrhage is minimized by incising through the skin and using blunt dissection to separate underlying soft tissues.[4] The buccal mucosa is incised and the drill bit is positioned between the second and third premolars just ventral to the gingival margin.[5] Soft tissues are protected during drilling by use of a drill guide. Wire is threaded through the holes between the premolar teeth and directed rostrally to be laced through holes previously made between the incisors. Differing patterns for wire placement may be used incorporating one or two wires.[4–6] The wire spanning the interdental space is twisted together to achieve compression at the fracture line.[4,6] The stab incisions through the cheeks are left to heal by second intention or closed with a single suture. The wires are removed 6–8 weeks after fixation.

Intra-oral acrylic splints provide stable fixation of interdental space fractures and are technically easy to use.[2,8,17] Following anesthesia and preparation of the oral cavity and cheeks the fracture is reduced. Polymethylmethacrylate or cold-curing acrylic is mixed and molded to fit the oral surface of the mandible or premaxilla, extending from the incisors to the second premolar, taking care to avoid the frenulum of the tongue. A thickness of 6–8 mm is sufficient for most splints; the acrylic may be thickened at sites of wire incorporation to reduce fatigue and breakage of the splint. The splint is removed after curing and rough edges are smoothed. Multiple 1.2 mm-diameter wire loops are placed through holes drilled into both sides of the mandible or maxilla between the incisors and in the interdental space, rostral and caudal to the fracture line. The splint is drilled to match the holes in the mandible or maxilla and the wires are twisted together and bent down into the gingiva. Wire loops may also be placed around the second premolar teeth to provide anchorage at sites caudally.[4] Holes for these wires are

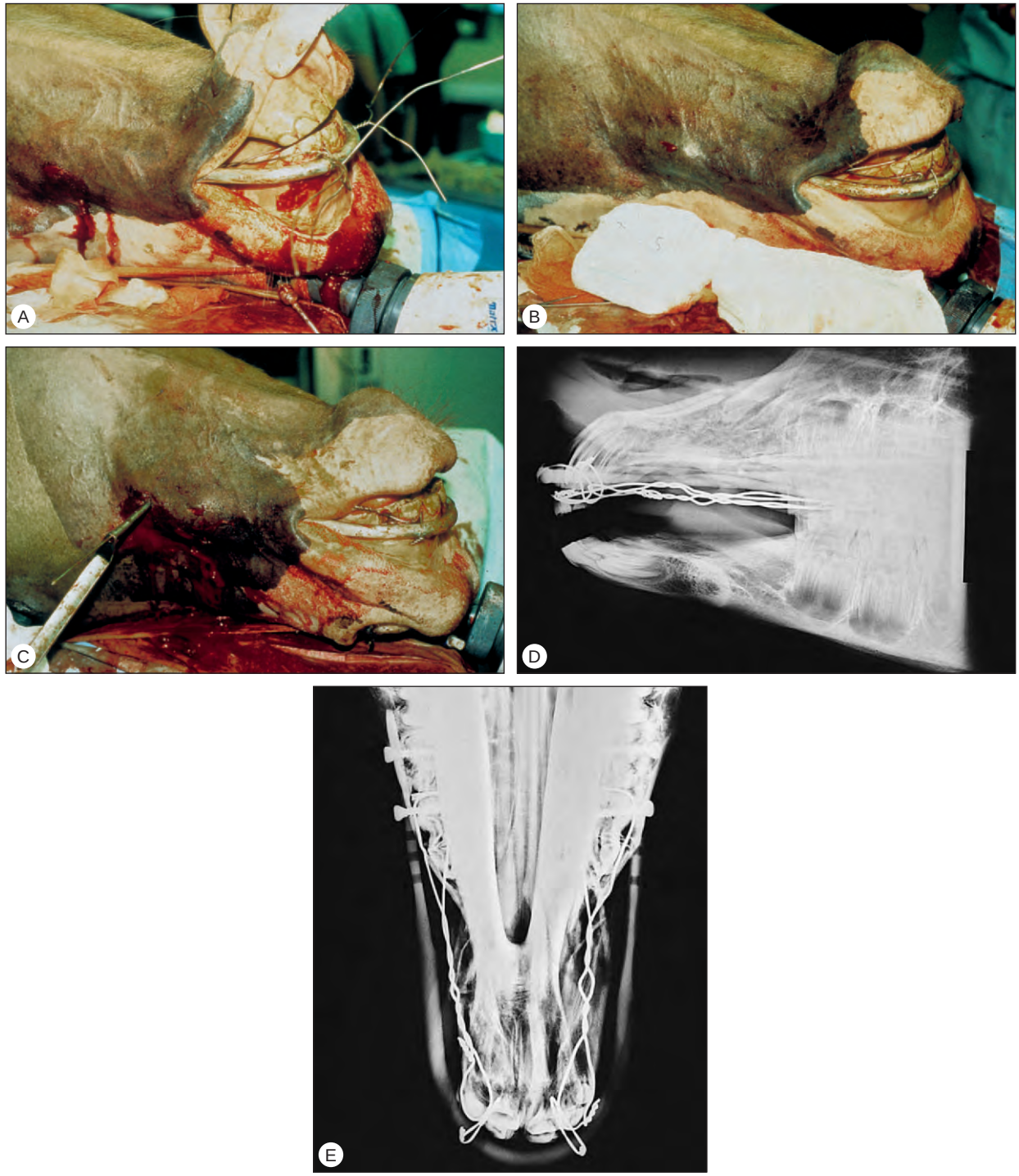


Figure 21.8. Stabilization of a bilateral fracture through the maxillary interdental space with a U-bar that has been attached to the rostral premolars with cortical bone screws. With the horse in dorsal recumbency and nasally intubated, the U-bar is contoured to fit around the labial and buccal surfaces of the incisors and rostral premolars. (A) Holes are drilled between the incisors and wire threaded between the teeth and around the bar. (B) The wires are twisted around the rostral portion of the U-bar to anchor the appliance. (C) Stab incisions are made through the cheeks, the fracture is reduced and the incisors aligned prior to drilling 3.2 mm holes through the second and third premolars on each side of the maxilla. The holes in the teeth are tapped and a 4.5 mm screw inserted to attach the U-bar to the premolars. (D) and (E) Tension-band wiring spanning the interdental space was also employed in this case.

placed as described above for tension-band wiring. Alternatively the methylmethacrylate splint can be formed over preplaced tension band wires between the incisors and premolars.[18] This technique allows the fracture to be reduced completely before application of the splint.

Intra-oral acrylic splints require minimal surgical invasion of the mandible; avoid the risk of damage to tooth buds or roots and provide fixation along the tension side of the fracture.[13] Acrylic splinting can be used successfully in foals that have not erupted incisors suitable for placement of wires.[17] In foals a wire loop can be passed around the mandible in the diastema as an additional anchorage point for the splint. Care should be taken to pass the wire close to the bone to avoid strangulation and necrosis of the soft tissues. The oral cavity should be flushed daily and the splint removed 6–8 weeks after surgery, with the patient sedated and standing.

Intra-oral placement of U-bars with fixation around the teeth using wire has been described to treat fractures of the interdental space.[1,5,19,20] These are especially useful for comminuted fractures in which increased stability is required. Aluminum rod, 6.3–9.5 mm is contoured to fit around the labial and buccal aspect of the mandibular incisors and second and third premolar teeth, respectively. Holes are drilled into the bar corresponding to sites between the rostral second and third cheek and incisor teeth. The rod may be flattened to make drilling through the bar easier. Holes are drilled between the mandibular premolar teeth and the incisors, as previously described. Stab incisions in the cheeks are necessary to access the site between the premolar teeth. If accessible, the fracture site is debrided, lavaged and reduced. The formed rod is placed in the oral cavity around the mandibluar or maxillary teeth and the holes in the bar are aligned with holes between the teeth. Wire (1.2 mm) is placed through the bar, around an adjacent premolar or incisor tooth, and then back through the bar. Wire ends are twisted together on the buccal and labial aspect of the teeth and bent down into the gingiva to prevent laceration.

Placement of wire through holes in the bar and between teeth is difficult due to lack of visualization and limited working space. As an alternative, screw fixation of the U-bar to the teeth has been performed (Honnas, unpublished data, 1997) (Fig. 21.8).[21] The bar is contoured prior to surgery, as described previously, and 5.0 mm holes are drilled in the bar at a site corresponding to the rostral two premolar teeth. Stab incisions are made in the cheeks over the premolar teeth and the bar is positioned following reduction of the fracture. Holes are drilled into the premolar teeth with a 3.2 mm drill bit and are tapped to accept a 4.5 mm cortical bone screw. Wire is used to attach the rostral portion of the bar to the mandible, as previously discussed. The U-bar is removed 6–8 weeks after surgery.

U-bars allow fixation of mandibular fractures with minimal surgical invasion and places the fixation on the tension side of the fracture.[13] Long-term outcome of the effects on the premolar teeth that have been drilled for screw fixation of the U-bar has not been reported. To date, results using this fixation have been good and resultant dental disease has not been a problem (Honnas, personal observation).

Type II Kirschner–Ehmer frames, with intramedullary pins placed completely across the mandible rostral and caudal to the fracture line, can be successfully used to stabilize interdental space fractures (Fig. 21.9).[1,2,4,5] Type I external fixators which engage only one side of the mandible have also been used but provide less stablization than a Type II fixator. Stab incisions are made into the skin overlying the mandible and pilot holes are drilled across the mandible. Radiographic guidance is used to avoid damaging teeth. Protection of soft tissues during drilling and pin placement decreases the incidence of pin tract infection. Pins ranging in size from 3.1–6.3 mm diameter are used depending on the size of the horse. Use of two pins on either side of the fracture is recommended when possible.[2,5,14] Pins should be placed in a straight line to allow connection to the sidebar. Pins are cut approximately 4 cm from the skin surface. Clamps and metal rods or acrylics are used as sidebars to connect pins placed through the mandible (Fig. 21.10). The fixator should be bandaged to reduce the risk of the horse catching the sidebars on objects in the environment (Fig. 21.11).[14] Sidebars made of acrylic may be molded to decrease entanglement of the external fixator with objects in the environment. Approximately 6 weeks will be needed to allow sufficient healing of the mandible fracture. Following removal of the external fixator, pin tracts are lavaged daily until healed.

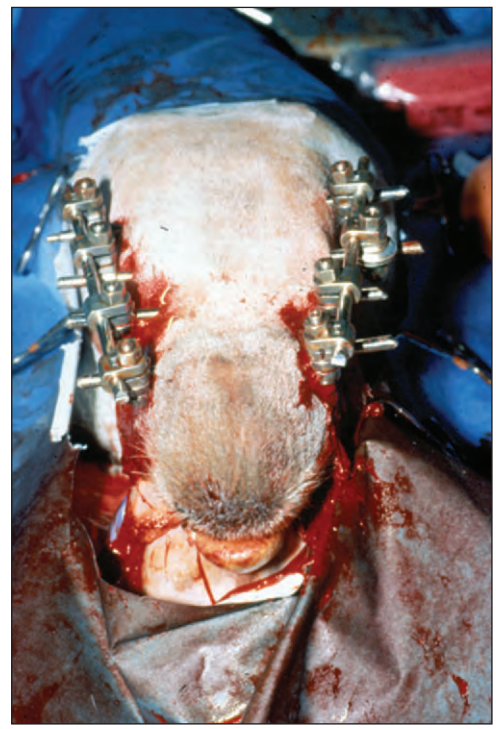


Figure 21.9. View of ventral mandible with Type II external fixator using metal clamps to attach the sidebars.



Figure 21.10. View of ventral mandible with Type II external fixator using flexible plastic tubing filled with acrylic to connect the sidebars.

Advantages of a Kirschner–Ehmer frame include: the ability to obtain purchase into rostral fragments of the mandible when limited space for plate fixation is present; relative ease of application compared with plating; decreased amount of tissue dissection needed for placement; lower risk of disseminating infection; patient acceptance and decreased risk of damage to teeth, especially in younger horses.[4,5,14,15] The use of intra-oral wiring in combination with an external fixator significantly increases the strength of the repair of interdental space fractures.[22]

Bone plating can provide a successful repair of fractures of the interdental space and mandibular body and is recommended in adult horses when increased stability is needed.[2,15] In vitro testing of osteotomized mandibles has shown that compression plating provides the most stable fixation when compared with wiring and external fixation techniques.[22] Bone plating is discussed below under caudal mandibular fractures.

The strength of each of the repair methods mentioned above has been compared in a study using osteotomized cadaver mandibles. By testing the repaired mandibles in a monotonic bending device, dynamic compression plating was shown to provide the most stable fixation. External fixators without the augmentation with interdental wiring was found to provide inferior stability.

Rarely, fractures of the rostral mandible, involving either the incisive area or interdental space, are accompanied by severe damage to the bone and overlying soft tissues, resulting in a poor prognosis for healing and survival of the tissues of the rostral mandible. Rostral mandibulectomy or maxillectomy can be used in these cases and is performed by excising the rostral fragments, debriding the area of the fracture and soft tissues, and apposing mucosa over the mandibular stump when possible (Fig. 21.12). Rostral mandibulectomy has been reported for the treatment of rostral mandibular tumors.[23] The ability of the horse to eat hay and grain is not inhibited and the cosmetic results are good in most cases.

Fractures of the caudal mandible

Fractures of the mandibular body and the vertical ramus have been reported to be uncommon.[2,3,5] In a recent review of our referral hospital population, however, these fractures were encountered quite frequently, representing 39 per cent of cases.[8] Radiography is used to define involvement of teeth, temporomandibular joints, the hyoid apparatus and to



Figure 21.11. Following surgery, the sidebars on an external fixator should be bandaged to prevent the horse from catching the apparatus on objects in the environment.



Figure 21.12. Rostral maxilectomy performed to treat a comminuted maxillary fracture, which failed to respond to conservative treatment.

assess for comminution, and guides the surgeon to applicable treatment options and implant positioning.[15] Fractures of the molar portion of the mandibular body are typically unilateral with minimal displacement and comminution.[2,4,8] Reported fractures of the vertical ramus include transverse, oblique and degloving fractures of the angle of the mandible.[2,24] Caudal mandibular fractures can be treated using conservative therapy, compression plating or external fixation.[2,4,5,25,26]

Fractures of the caudal region of the body or ramus of the mandible resulting in minimal instability, malocclusion and pain that permit the horse to eat are candidates for conservative therapy.[13,15] Sixty-eight per cent of unilateral fractures of the caudal mandible were treated conservatively in our case series.[8] In these cases, the overlying pterygoid and masseter muscles confer stability to the fracture.[24] Anti-inflammatory medications and lavage of the oral cavity increase patient comfort, and feeding mashes of complete pelleted feeds provides nutrition in an easily consumed fashion. Antimicrobial therapy is indicated when the fracture is open to the oral cavity. Care providers should ensure that the horse continues to eat and drink. Radiographic evaluation of the fracture should be performed periodically. Evidence of lack of progression of healing within 6 weeks post fracture or bony lysis denoting osteomyelitis at the fracture site are indications for internal fixation of the fracture.[13]

Fracture of the caudal angle of the mandible with degloving of the overlying soft tissues has been described.[2] The injury results from placement of the head between stationary objects and then pulling back. Communication of these fractures with the oral cavity or adjacent alveoli is uncommon. Surgical removal of the small fractured segments of the mandible can be performed with minimal functional or cosmetic disturbance.[2] Internal fixation may be indicated for larger fragments.

Indications for fixation of caudal mandibular fractures are instability, malocclusion preventing prehension or mastication, pain with unwillingness to eat and bilateral involvement.[2,4,5,13] Internal or external fixation can be used to stabilize caudal mandibular fractures. Bone plating provides a strong bone-implant construct and can be used successfully in unilateral or bilateral body fractures and fractures of the vertical ramus (Fig. 21.13).[24,26] Bone plating is recommended for adult horses in which healing may be delayed.[15] External fixation provides stable fixation of fractures of the mandibular body and carries benefits of decreased surgical dissection and increased latitude in placement of implants.

A ventrolateral approach to the mandible is used for bone plating mandibular fractures. The facial artery, facial vein, parotid salivary duct and mental nerve are identified and preserved during surgical dissection. Elevation of the masseter muscle from the mandible is necessary and is accomplished by transection of the attachments of the muscle at the ventral border of the mandible and reflecting the muscle proximally. The size of plates used ranges from 3.5 to 4.5 mm narrow or broad, depending on fracture configuration and size of the patient. Positioning of the bone plate on the ventrolateral aspect of the mandible is preferred.[2] Alternative plate positions include ventral and ventromedial.[1,2] The plate is contoured and attached to the bone; a minimum of three screws on either side of the fracture is recommended.[14] Teeth roots should be avoided when applying plates. The use of masonry drill bits to drill through teeth roots without causing tooth loss has been reported.[4]



Figure 21.13. Comminuted fracture of the vertical ramus of the mandible that was repaired with dynamic compression plate. The masseter muscle was elevated and reflected dorsally to access the mandible for plate application. The fracture healed without complication.

Wilson and co-workers have described a surgical approach to the ramus of the mandible for internal fixation.[24] An incision is made 2–3 cm rostral and parallel to the caudal border of the mandible from the zygomatic arch to the point where the facial artery crosses the ventral border of the mandible. The transverse facial artery and vein, facial nerve and auriculotemporal nerve are retracted dorsally and the insertion of the masseter muscle along the caudal mandible border is sharply incised, allowing subperiosteal elevation of the masseter muscle. Plates used rostrally and caudally on the lateral surface of the ramus provide secure fixation. Care is warranted when tapping and placing screws in the thinner rostral portion of the ramus.[24] Following internal fixation, the masseter fascia and periosteum are closed followed by subcutaneous and skin closure.

Although bone plating provides a very stable construct, extensive surgical dissection is required. Fractures open to the oral cavity can be expected to become infected, necessitating removal of plates after bone healing.[1,14] Bone plating may be a necessity despite the presence of an open fracture when treating unstable fractures in adult horses and when buttress plating is needed in conjunction with corticocancellous bone grafting.[1,2,4–7,13]

External fixators, including type II Kirschner–Ehmer frames previously discussed, can be used in treatment of caudal fractures of the body.[4] The use of a pinless external fixator has been reported in cattle for fixation of mandibular body fractures.[25] In the place of pins, special bone clamps were used in series along the ventral border on the mandible. The clamps embed into the cortex of the mandible without penetrating the medullary cavity. The clamps were tightened in place using a nut on a central hinge. An additional short bar ran laterally from the central hinge and enabled all clamps to be connected to a common connecting bar coursing longitudinally along the mandible. Stab incisions followed by blunt dissection of soft tissues were necessary for placement of the clamps into the mandibular body. Fixators were removed 5–8 weeks after fixation and stability of the healed mandible allowed immediate return to mastication. Draining tracts around the implants healed with local wound therapy and complications encountered with healing of the mandibular fractures were not attributable to the external fixator.

Prognosis for healing of caudal mandibular fractures is guarded to good.[2,4,14] The majority of horses treated at Texas A&M for caudal mandibular fractures survived.[8] Complications did occur however, and were generally associated with communication with the oral cavity and involvement of alveoli. When possible, aggressive debridement of fracture line with thorough lavage, closure of oral mucous membranes and antimicrobial use are the best means of prevention of osteomyelitis and sequestration. Treatment of osseous infection developing postoperatively follows the guidelines of debridement, autogenous cancellous bone grafting when indicated and antimicrobial therapy. Involvement of alveoli with consequential infectious periodontitis and pulpitis will require removal of affected teeth.[2,5,14] Removal of a tooth or destruction of a permanent developing tooth mandates regular dental care to prevent serious malocclusion. Implant-associated infection requires removal of plates or pins, debridement of soft tissues, lavage and antimicrobials. Resolution of infection after implant removal often proceeds without further complication.[14]

Failure of fixation is an additional potential complication. Assessment of the initial fixation, degree of stability and presence of infection should be considered in planning further treatment. Use of a more stable means of fixation and concurrently addressing any additional problems such as infection will provide the best prognosis for complete healing. Adjunctive therapy, including autogenous cancellous bone grafting and antimicrobial impregnated beads may be indicated.

Fractures involving the temporomandibular joint

While fractures involving the temporomandibular joint are rare, they are associated with a higher degree of morbidity.[2,27,28] Radiography, including lateral, oblique and dorsoventral views, is indicated. Lateral projections with the mouth held open with a speculum may give better definition to the temporomandibular region as the condylar and coronoid processes will be subluxated.[2,14]

Internal fixation of fractures involving the temporomandibular joint has not been reported to the author's knowledge. Conservative therapy or condylectomy are the treatment recommendations that have been published.[27,28] Bilateral mandibular condylar fractures have been reported in a stallion.[28] Treatment of the horse in that report included floating the caudal cheek teeth to allow improved prehension and mastication as premature occlusion of the molars prevented occlusion of the incisors.

Condylectomy of the mandible has been advocated for treatment of fractures involving the temporomandibular joint.[27] Branches of the auriculopalpebral nerve are retracted ventrally and portions of the parotid gland are retracted caudally. A horizontal incision is made in the lateral mandibular ligament and joint capsule over the meniscomandibular compartment of the temporomandibular joint. A vertical periosteal incision is made in the lateral surface of the condyle and the periosteum is liberally elevated. Condylectomy is performed 2.5 cm ventral to the articular surface of the condyle. The joint capsule, subcutaneous tissues and skin are closed.

Temporomandibular joint fractures carry a guarded prognosis as degenerative joint disease and resultant inhibitions to prehension and mastication render the horse susceptible to weight loss.[28,29] The prognosis after condylectomy to treat degenerative disease of the temporomandibular joint appears to be guarded to good.[29]

Complications and aftercare

Complications are common with mandiblular and maxillary fractures that have been treated with conservative management and those repaired with internal or external fixation. Short-term complications including soft-tissue infection and wire loosening or breakage were reported in 27 per cent of horses with rostral mandibular fractures.[3] Loosening of implants that goes unnoticed can lead to delayed fracture healing and possible malocclusion.[30] Drainage is most effectively dealt with by timely removal of implants, providing adequate drainage, and systemic antibiotics. Horses with persistent drainage should be examined radiographically for signs of bony sequestrum formation. Pin tract infections are seen frequently with external fixators. This complication can be lessened by minimizing soft-tissue trauma during

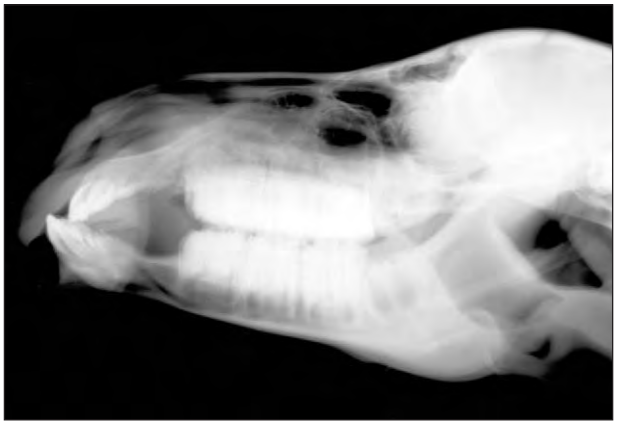


Figure 21.14. Lateral radiograph of a foal which sustained a compressive fracture of the premaxilla several weeks previously. Treatment consisted of wiring the mandibular incisors to the mandibular premolars to retard growth, allowing the maxilla to grow to an equal length.

placement and decreasing the likelihood of pin migration through the use of positive profile threaded pins. Malocclusion of incisors following rostral mandibular and maxillary fractures was reported in 14 per cent of horses.[3] In foals, brachygnathism following bilateral fracture can occur. This complication can be dealt with by placement of braces on the mandible or maxilla using previously described wiring techniques between the inicisors and premolars to retard growth until the fractured jaw comes into alignment (Fig. 21.14). Infection of the tooth roots of the premolars and molar can be seen following fracture of the horizontal ramus of the mandible and usually requires extraction or repulsion of the teeth. The importance of follow-up dental care should be emphasized to owner, especially in horses who have lost teeth as result of fracture. The majority of horses do not require a special diet following fracture stabilization but they should be monitored closely to ensure adequate feed and water consumption.[30]

Summary

Because the mandible and to a lesser degree the maxilla of the horse is frequently fractured, knowledge of mandibular surgical anatomy and the most common types of mandibular fractures encountered is essential to the equine veterinarian. This chapter reviews pertinent surgical anatomy, clinical signs and diagnosis of fractures of the mandible and discusses their treatment on an anatomical basis.

Fractures of the mandible are defined by physical and radiographic examination. Use of fixation methods that counter the tensile forces on the dorsal or oral surface of the mandible should provide the best opportunity for successful healing. The prognosis for the majority of mandibular fractures is good when thorough debridement and use of proper bone-implant constructs are employed.

REFERENCES

1. Sullins KE and Turner AS (1982) Management of fractures of the equine mandible and premaxilla (incisive bone). *Compendium Continuing Education for the Practicing Veterinarian,* 4(11), S480–S489.
2. DeBowes RM (1996) Fractures of the mandible and maxilla. In: *Equine Fracture Repair,* ed. AJ Nixon, WB Saunders, Philadelphia, pp. 323–332.
3. Henninger RW, Beard WL, Schneider RK *et al.* (1999) Fractures of the rostral portion of the mandible and maxilla in horses: 89 cases (1979–1997). *Journal of the American Veterinary Medical Association,* 214, 1648–1652.
4. Schneider RK (1990) Mandibular fractures. In: *Current Practice of Equine Surgery,* eds. NA White and JN Moore, JB Lippincott, Philadelphia, pp. 589–595.
5. Meagher DM and Trout DR (1980) Fractures of the mandible and premaxilla in the horse. *Proceedings of the American Association of Equine Practitioners,* 26, 181–192.
6. Little CB, Hilbert BJ and McGill CA (1982) A retrospective study of head fractures in horses. *Australian Veterinary Journal,* 62(3), 89–91.
7. Monin T (1977) Tension band repair of equine mandibular fractures. *Journal of Equine Medicine and Surgery,* 1, 325–329.
8. Knox PM and Honnas CM (2003) Unpublished data.
9. Auer JA (2000) Mandible, maxilla and skull. In: *AO Principles of Osteosynthesis,* ed. GE Fackelman, JA Auer and DM Nunamaker, AO Publishing, Stuttgart, pp. 35–56.
10. Godinho HP and Getty R (1975) Equine cranial nerves. In: *Sisson and Grossman's The Anatomy of the Domestic Animals* (5th edn), ed. R Getty, WB Saunders, Philadelphia, p. 656.
11. Kainer RA (1993) Clinical anatomy of the equine head. *Veterinary Clinics of North America Equine Practice,* 9(1), 1–23.
12. Hillman DJ (1975) Equine skull. In: *Sisson and Grossman's The Anatomy of the Domestic Animals* (5th edn), ed. R Getty, WB Saunders, Philadelphia, pp. 335–336.
13. Watkins JP (1991) Diseases of the head and neck. In: *Equine Medicine and Surgery* (4th edn), eds PT Colahan, IG Mayhew, AM Merrit and JN Moore, American Veterinary Publications, Goleta, CA, pp. 1461–1464.
14. Blackford JT and Blackford LW (1992) Surgical treatment of selected musculoskeletal disorders of the head. In: *Equine Surgery,* ed. JA Auer, WB Saunders, Philadelphia, p. 1075.
15. Ragle CA (1993) Head trauma. *Veterinary Clinics of North America Equine Practice,* 9(1), 171–176.
16. DeBowes RM, Cannon JH, Grant BD *et al.* (1981) Lag screw fixation of rostral mandibular fractures in the horse. *Veterinary Surgery,* 10, 153–158.
17. Colahan PT and Pascoe JR (1983) Stabilization of equine and bovine mandibular and maxillary fractures using an acrylic splint. *Journal of the American Veterinary Medical Association,* 182, 1117.
18. Dart AJ and Pascoe RR (1987) Treatment of a bilateral mandibular fracture in a mare using an intraoral acrylic splint. *Australian Veterinary Journal,* 64, 382–384.
19. Gabel AA (1969) A method of surgical repair of the fractured mandible in the horse. *Journal of the American Veterinary Medical Association,* 155, 1831–1834.
20. Krahwinkel DJ, Heffernan HJ and Ewbank RL (1969) Surgical repair of fractured maxillae and premaxillae in a horse. *Journal of the American Veterinary Medical Association,* 154, 53–57.
21. Von Saldren FC and Gawaz M (1993) The dental intraoral fixation of mandibular fractures with dynamic compression plates (DCP) in the horse. *Proceedings Annual Meeting of the European College of Veterinary Surgeons,* 2, p. 29.

22. Peavey CL *et al.* (2003) Fixation technique influences on the monotonic properties of equine mandibular fracture constructs. *Veterinary Surgery,* 32(4), 350–358.
23. Richardson DW, Evans LH and Tulleners EP (1991) Rostral mandibulectomy in five horses. *Journal of the American Veterinary Medical Association,* 199, 1179–1182.
24. Wilson DG, Trent AM and Crawford WH (1990) A surgical approach to the ramus of the mandible in cattle and horses. *Veterinary Surgery,* 19, 191–195.
25. Lischer CJ, Fluri E, Kaser-Holz B *et al.* (1997) Pinless external fixation of mandible fractures in cattle. *Veterinary Surgery,* 26, 14–19.
26. Murch KM (1980) Repair of bovine and equine mandibular fractures. *Canadian Veterinary Journal,* 21, 69–73.
27. Barber SM, Doige CE and Humphreys SG (1985) Mandibular condylectomy, technique and results in normal horses. *Veterinary Surgery,* 14, 79–86.
28. Hurtig MB, Barber SM and Farrow CS (1984) Temporomandibular joint luxation in the horse. *Journal of the American Veterinary Medical Association,* 185, 78–80.
29. Patterson LJ, Shappell KK and Hurtig MB (1989) Mandibular condylectomy in a horse. *Journal of the American Veterinary Medical Association,* 195, 101–102.
30. Beard W (1999) The skull, maxilla, and mandible. In: *Equine Surgery,* eds. JA Auer and JS Stick, WB Saunders, Philadelphia, pp. 887–899.

Appendix

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A Glossary of Equine Dental Terminology

Ablations Taking away, wearing down, erosion of an area.

Abrasion Mechanical wearing away of teeth by abnormal stresses. This could result from abnormal stresses on the teeth.

Abrasive points Burrs, rotary instruments that have an abrasive coating on the operative head.

Abscess Localized collection of pus, generally as a result of an infection.

Absorbent points Cones of porous paper used to dry the root canal after instrumentation.

Accessional Permanent teeth that do not replace deciduous teeth but rather become an accession (an addition) to the deciduous or succedaneous teeth or to bone types.

Acellular cementum Cementum that has no cells trapped in it.

Achondroplasia Developmental abnormality of cartilage. Failure of cartilage formation.

Acid etching The microscopic roughening of the enamel, dentinal or cemental surface with a dilute acid to increase its mechanically retentive property to restorative materials. Usually the procedure preferentially removes the enamel prism cores and leaves the prism peripheries intact.

Acquired Pertaining to something obtained by itself, not inherited.

Acrodont Tooth that is attached to the crest of the jaw through ankylosis and lacks a root formation.

Acrylic Plastics, basically methyl methacrylate, mixed from a powder (polymer) and liquid (monomer). Referring to synthetic compounds that contain acrylic acid, which is formed by the oxidization of acrolein. These were once used extensively as restoratives.

Adamantinoma A benign but locally aggressive epulis. Obsolete term for ameloblastoma.

Aerobic bacteria Bacteria that thrive in the presence of oxygen.

Ala (pl. Alae) Latin for *wing*, referring to the sides of the nostrils of the nose.

Alginate Irreversible hydrocolloid impression material used frequently in veterinary dentistry, especially when partial–or full–mouth models are needed that require decent, yet not excellent detail.

Alignment Arrangement (e.g., of teeth) in a row.

Allele An alternative form of a gene.

Alveolar Of or pertaining to the sockets of the teeth.

Alveolar bone Bone forming the sockets for the teeth.

Alveolar crest Highest part of the alveolar bone closest to cervical line of the tooth.

Alveolar mucosa Loosely attached mucous membrane covering bone that contains the teeth.

Alveolar nerve Branches of mandibular nerve entering mandibular foramen and innervating all mandibular teeth.

Alveolar process The part of the maxilla or mandible that contains the sockets of erupted teeth or the crypts of developing unerupted teeth.

Alveolus The socket within the jaws in which the reserve crown or roots of a tooth lie.

Amalgam An alloy or combination of finely powdered metals that are mixed, or triturated, with mercury to form a condensable mass.

Amalgamation The process of combining an alloy with mercury (e.g. silver or copper alloy triturated with mercury makes up an amalgam).

Amalgamator A mechanical device or a ground glass mortar and a glass pestle used to triturate an alloy.

Amelo-(prefix) Indicating enamel or tissues of the epithelial odontogenic origin.

Ameloblast Germ cell originating from epithelium from which the enamel is formed.

Ameloblastic odontoma Dental tumor originating from dental laminar epithelium.

Ameloblastoma The most common tumor of the dental laminar epithelium. It is slow-growing but often demonstrates a multiple cystic structure and can extend into bone.

Amelodentinal junction Junction between enamel and dentin.

Amelogenesis The process of enamel formation.

Amylase Enzyme in saliva and pancreatic juice. Converts starch into sugar.

Amyloid *Pathology*, Abnormal, complex, insoluble glycoprotein-like substance, the formation of which has been connected with immunoglobins.

Amyloidosis A degenerative condition where amyloid is deposited in the parenchymal organs (e.g. kidneys, liver and spleen) rendering the cells nonfunctional. The condition often follows a chronic suppurative focus in the body when it is termed secondary or reactive amyloidosis.

Anachoresis Exposure to bacteria through a hematogenous or lymphatic route.

Anaplastic sarcoma Undifferentiated tumor of mesenchymal origin.

Anatomical crown That part of the tooth where enamel constitutes a portion of its external or internal structure.

Anatomical root That part of the tooth where enamel does not constitute a portion of its external or internal structure and is covered with cementum.

Anelodont teeth Teeth with a limited period of growth, the root canals progressively narrow and the apical foramina become constricted with age. Subdivided into hypsodont and brachydont.

Angle of the mandible Junction of the horizontal and vertical ramus of the mandible.

Angular process Portion of the vertical ramus of the mandible.

Anisognathism Condition of having unequal jaw widths in which the mandibular molar occlusal zone is narrower than the maxillary counterpart. It is seen in the equine, feline, canine, bovine, and other species.

Ankylosis Fusion of cementum of a tooth with alveolar bone. Joining together of bone and bone, or tooth and bone by direct union of the parts, resulting in rigidity.

Anodontia Condition in which most or all the teeth are congenitally absent. The absence of teeth, partial or total and qualified accordingly, usually indicating a failure of tooth development.

Anomaly Any noticeable difference or deviation from that which is ordinary or normal.

Anorexia Loss of appetite.

Anterior Rostral. Situated in or toward the front. This term is commonly used to denote the incisor and canine teeth or the area toward the front of the mouth.

Anterior teeth Collective term for the incisors and canines.

Antral Relating to an antrum.

Antrum An air-filled natural cavity, usually in bone. Also called a sinus (e.g. maxillary antrum=maxillary sinus).

Apexification Maturation of root ends.

Apical Relating to the apex of a tooth, i.e. the root area of a mature tooth (or similar area of an immature tooth before root formation).

Apical foramen, foramina (pl) Entrance(s) to the pulp cavities of a tooth (where the blood vessels, nerves and lymphatics enter pulp).

Apicoectomy Endodontic treatment that involves amputation of the root tip.

Approximal See Proximal. Interproximal. *Dentistry*, collective term that refers to surfaces of teeth that face adjoining teeth of the same dental arch.

Aradicular hypsodont A subdivision of hypsodont, long-rooted, describing dentition without true roots (sometimes called open-rooted) that produces additional crown throughout life. As teeth are worn down, new crown emerges from the continually growing teeth, as in lagomorphs and the incisors of rodents.

Arcades Refers to the arches of teeth in some brachyodont species. In horses the straight *rows* of cheek teeth are separated from the incisors by the physiological diastema ("bars of mouth") so all of the teeth do not form an arch.

Articular disc Fibrous disc between the mandibular condyle and the temporal fossa.

Articular process Portion of the vertical ramus of the mandible that is part of the temporomandibular joint.

Attrition Process of normal wear on the crown, usually due to prehension and mastication.

Auto-immune disease Immune-mediated inflammatory reaction to the host's own tissues.

Avulsion Tearing away of a part, such as a tooth.

Axial Pertaining to the longitudinal (long) axis of a structure.

Bacterial plaque Also, dental plaque. Soft mass of micro-organisms, cellular material and food debris that adheres to the surfaces of teeth and/or gingiva.

Basal cell carcinoma Tumor originating from basal cell layer of epidermis.

Beak Dental overgrowth resembling the beak of a bird.

Bell stage Third embryonic stage of enamel organ formation in which the crown form is established.

Benign Nonmalignant. Such lesions do not destroy the tissue from which they originate or spread to other parts of the body (metastasize).

Bifurcation Division into two parts or branches, e.g., two roots of a tooth.

Bisecting angle Technique of taking radiographs to minimize linear distortion by aiming the beam perpendicular

to the line that bisects the angle formed by the long axis of the tooth and the film.

Bishoping To burn. Tampering with the dental appearance of an animal, normally a horse, to make it look younger for fraudulent reasons. Making an old horse appear younger by burning or drilling and staining an artificial concavity in the dentine of the incisor tablets in an attempt to mimic the infundibulum of a younger animal.

Bit Mechanial device held in the mouth and attached to the reins.

Bite plane Appliance in which the incline is designed to prevent occlusal closure.

Biting force The pressure (N/cm^2) exerted by teeth when engaged by the muscles of mastication.

Blast *Histology*. Embryonic cell or formative layer.

Blind wolf tooth Unerupted wolf tooth.

Body of the mandible Horizontal portion of the mandible, excluding the alveolar process.

Bolus of food A ball of food that has been chewed and mixed with saliva and is ready to be swallowed.

Bone Hard connective tissue that forms the skeleton of the body. The hardness is attributable to the hydroxyapatite crystals.

Brachy- (prefix) Indicating something short.

Brachycephaly Condition in which individuals have short, broad facial profiles.

Brachygnathic Having an abnormally short mandible.

Brachydont Teeth with a shorter crown:root ratio (e.g. primates, dogs, cats, and carnivores in general).

Brachygnathism Overjet (parrot mouth). A congenital deformity in which the upper incisors overlap the lower incisors.

Branchial arches I and II Developmental sections of the facial region present around day 21 of fetal development.

Bruxism Abnormal grinding of the teeth.

Bucca Latin for "cheek."

Buccal Pertaining to or directed toward the cheek (outside/lateral aspect of mouth). Also called facial. Surfaces of the posterior teeth and adjacent tissues facing the buccinator muscle of the cheek.

Bucco- (prefix) Signifying buccal, cheek.

Buccostomy The formation of a surgical opening through the side of the face that is kept patent beyond the duration of the operation.

Buccotomy Surgical incision made through the side of the face, usually performed in herbivores, to accomplish an intra-oral procedure that is inaccessible through a labial approach.

Bud state First stage of development of the enamel organ that develops from the dental lamina.

Bullous pemphigoid Autoimmune disease frequently causing lesions at the oral mucocutaneous junction.

Bundle bone Extra thickness of bone added to the cribriform plate.

Burrs Rotary dental instruments with cutting blades or abrasive surfaces as an active part of the operative head.

Cachexia Condition of weakness of the body that results from a debilitating chronic disease.

Calcification Process by which organic tissue becomes hardened by a deposit of calcium salts within its substance. Literally, the term denotes the deposition of any mineral salts that contribute toward hardening and maturation of tissue.

Calculus Mineralized dental plaque that adheres to tooth surfaces and prosthetic dental materials.

Campylorrhinus lateralis Twisted maxilla and nasal septum. Wry nose. Developmental abnormality.

Canal Long tubular opening, e.g., through a bone or tooth root.

Cancellous bone Less dense bone situated between surrounding denser cortical bone plates.

Canine teeth The teeth found between the incisors and cheek teeth usually in male horses; the fighting teeth of a horse (Triadan 104, 204, 304, 404).

Cap The remnant of a deciduous cheek tooth that covers an erupting permanent cheek tooth (premolars only) and is later shed.

Cap stage Second stage of enamel organ development.

Capsule (joint) Fibrous band of tissue surrounding a joint and limiting its motion.

Carbide Carbon mineral used for grinding and floating.

Caries Demineralization of calcified dental tissues and destruction of their organic parts through the acid produced by micro-organisms.

Cariogenic acid Acid produced by cariogenic bacteria.

Caudal Relating to the posterior aspect of a structure.

Caudal infraorbital block Intraoral regional anesthetic nerve block achieved by injecting the infraorbital nerve at the caudal aspect of the infraorbital canal.

Cellular cementum Cementum that has cells (cementocytes) trapped in it.

Cellulitis Diffuse inflammation often purulent, of the soft tissues.

Cement Used to apply orthodontic brackets, appliances, crowns, or other prosthodontic devices. A plastic material that is used to affix dental restorations. A type of filling material.

Cemental hypoplasia Commonly seen in maxillary tooth infundibula. Incomplete cementogenesis.

Cementoblasts Cells that form cementum.

Cementoclasts Cells involved with the resorption of cementum.

Cementocytes Cementoblasts that have become entrapped within cementum.

Cementodentinal junction (CDJ) Junction where the cementum and dentin contact.

Cementoenamel junction (CEJ) Junction of enamel of the crown and cementum of the root, (e.g., cervical line).

Cementogenesis Process of cement formation.

Cementoid Term meaning cementum-like.

Cementoma Benign proliferation of the connective tissue that produces cementum or cementum-like tissue.

Cement, cementum A bone-like calcified component of teeth, includes peripheral cementum which composes a significant portion of the equine clinical crown and infundibular cementum.

Cemental hypoplasia A developmental disorder commonly seen in maxillary cheek teeth infundibula due to incomplete cementogenesis.

Centric occlusion (central occlusion) Relationship of the occlusal surfaces of one arch to those of the other when the jaws are closed and the teeth are in maximum intercuspation.

Cephalic Relating to the skull or head.

Cephalometrics Anatomical measurements of skull structures.

Cheeks Lateral boundaries of the oral cavity.

Cheek teeth *Veterinary*. Usually an equine term to describe all the premolar and molar teeth but sometimes used to describe those teeth of other animals.

Cheilitis Inflammation of the lips.

Choke Esophageal obstruction.

Chondrosarcoma Malignant tumor of cartilage.

Chronic A process continuing over a long period of time (e.g., many months). The opposite of acute.

Cingulum A bulbous convexity on the cervical third of the lingual surface of a tooth.

Cleft palate Lack of joining together of hard or soft palate.

Clinical crown (erupted crown) The part of the crown that lies outside of the alveolus and gingiva (e.g., the part that is visible in the oral cavity).

Colic Pain related to alimentary disease.

Commissure Junction of the upper and lower lip at the angle of the mouth. Band of tissue joining two parts or organs together.

Complex or compound odontoma Mixed odontogenic tumor composed of both epithelial and mesenchymal cells in a disorganized mass that contains no tooth-like structures. Can have a cystic component.

Composite Type of restorative typically composed of an organic polymer matrix of high molecular weight, usually bisphenol A-glycidyl methacrylate (bis-GMA) resin, with or without fillers.

Concha Any organ of the body that resembles a shell in shape. Nasal turbinate bone.

Condylectomy Excision of condylar portion of the mandible.

Condyloid process That portion of the vertical ramus of the mandible that is part of the temporomandibular joint.

Congenital Denoting a condition usually abnormal, present at or before birth, but one that is not necessarily hereditary.

Contrast Relates to the variation in the black and white density on areas of radiographic film.

Coprophagy The act of eating feces.

Coral formation *Veterinary*. Metaplastic calcification of the conchal cartilage through chronic infection.

Corona Tooth crown.

Coronal Direction toward the crown. Relating to or towards the crown part of a tooth.

Coronoid process Bony projection at the upper anterior portion of the vertical ramus. It is the attachment location for the temporal muscle.

Cortex The external layer of an organ or bones; hence, cortical.

Cranial nerves Nerves of the head.

Craniofacial deformity Skull and face deformities. May be developmental or acquired.

Crest As pertains to radiation, the height of the wave.

Crib-biting (cribbing) Destructive behavior when horses bite their food container resulting in an abnormal wear pattern to their incisors and the ingestion of air.

Cribriform plate Dense bone that forms the actual wall of the tooth socket.

Crown 1) The part of the tooth which contains enamel (both clinical crown and reserve crown), i.e., all of the tooth except the roots, which by definition contain no enamel. 2) A restorative that covers part or all of the clinical crown.

Crypt Term used to describe the early tooth socket.

Cryosurgery Surgical destruction by freeze/thaw cycles. Using liquid nitrogen.

Cup Hollow structure with open top.

Curvature of Spee Rising slopes of distal aspects of mandible.

Cusp 1) A pronounced elevation on the occlusal surface of a tooth terminating in a conical, rounded, or flat surface. 2) Any crown elevation that begins calcification as an independent center.

Cuspid (canine teeth, fang teeth) One of four pointed teeth situated one on each side of each jaw, immediately distal to the corner or lateral incisors.

Cyanoacrylate Adhesive material usually self-curing in the presence of moisture in an anaerobic environment.

Cyst Sac of fluid lined by epithelial cells; it may grow to varying sizes.

Debridement *Dentistry*. The removal of debris from a cavity in a tooth, extraction socket or root canal. *Medicine.* The surgical removal of cellular debris from the surface of a wound.

Decay The decomposition of organic matter.

Deciduous teeth The first dentition; milk teeth. See Primary teeth.

Deglutition Action of swallowing.

Dehiscence *Medicine*. The spontaneous breakdown of a surgical wound.

Dens Tooth.

Dens in dente (tooth within a tooth) Formed when the top of the tooth bud folds into itself, producing additional layers of enamel, cementum, dentin, or pulp tissue inside the tooth as it develops.

Dens invaginatus A developmental anomaly involving an invagination on the lingual or palatal surface of an incisor.

Dental arcade *Veterinary*. A term for the arrangement of all the cheek teeth and associated tissues in any one of the dental quadrants of the horse. The term appears to have originated from the meaning of 'arcade': 'a line of shops in a covered passage,' as the cheek teeth of the horse are also closely abutting, similar units in a straight line.

Dental arch All teeth forming an arch in either the maxillary or mandibular jaw.

Dental attrition Increased amount of wear or loss of tooth substance due to normal masticatory forces.

Dental cap *Colloquial*. **Crown prosthesis** *Veterinary*. The coronal remains of a horse's deciduous molars once the roots have been resorbed.

Dental lamina Embryonic downgrowth of oral epithelium that is the forerunner of the enamel bud. Ectodermal-origin epithelium pushes into mesodermal tissues underneath.

Dental papilla Mesodermal structure partially surrounded by the inner enamel epithelial cells. The dental papilla forms the dentin and pulp.

Dental sac Several layers of flat mesodermal cells partially surrounding the dental papilla and enamel organ. It forms the cementum, periodontal ligament, and some alveolar bone.

Dental star Occlusal appearance of secondary dentin in incisors.

Dentes canini Canine, cuspid, eye or fang teeth.

Dentes decidui Deciduous teeth.

Dentes incisivi Incisor teeth.

Dentes molares Molar teeth.

Dentes permanentes Permanent teeth.

Dentes premolares Premolar teeth.

Denticles Small, tooth-like structures.

Dentigerous Containing teeth or tooth-like structures.

Dentigerous cyst Developmental branchial arch dental follicle remnant with ectopic, systic tooth-like structure on the temporal bone.

Dentin (dentine) A soft (ivory like) component of the mineralised tooth and that increasingly fills the pulp chamber with age (e.g., with secondary dentin).

Dentinal tubules Space or tubes in the dentin occupied by the odontoblastic process.

Dentinocemental junction (DCJ) *See* CDJ.

Dentinoenamel junction (DEJ) Juncture within the crown of the tooth where the dentinal and enamel walls meet.

Dentinogenesis imperfecta A hereditary condition in which dentin is abnormally formed.

Dentition General character and arrangement of the teeth, taken as a whole, as in carnivorous, herbivorous, and omnivorous dentitions. Primary dentition refers to the deciduous teeth; secondary dentition to permanent teeth. Mixed dentition refers to a combination of permanent and deciduous teeth in the same dentition.

Dermatitis Inflammation of the skin and subcutis.

Developer Solution to make the latent image on an exposed x-ray film visible.

Diarthrodial joint Movable joints.

Diastema A space between teeth. In the horse refers both to the physiological space between the incisors and premolars (the interdental space) or more commonly to the pathological presence of a gap between adjacent teeth. (pleural Diastemata). Pathological diastema has also been termed "Valve Diastema".

Digastricus muscle Paired muscles from jugular process of occipital bones to mandible. Opens the mouth.

Digestive tract In the embryo, the roof of the entodermal yolk sac enfolds into a tubular tract forming the gut. Initially, a blind tract closed at both ends (anal-cloaca and oral-stomodeum).

Diphyodont The feature of having two sets of teeth, one designated deciduous or primary and the other permanent. Most domesticated animals (e.g., cats, dogs, cows, horses, etc.) and humans are diphyodont.

Disarming *Veterinary*. Procedure where one or more teeth are either extracted or shortened in order to prevent animals from inflicting injuries.

Distal Farthest away from the median line of the face.

Dolichocephaly Condition marked by a long, narrow facial profile.

Domestication Adjustment of animals to living with humans; taming.

Dominant An allele that produces an effect on the phenotype even when present in a single dose.

Dorsal Toward or situated on the top.

Dorsum of the tongue Top surface of the tongue.

Duplicidentata Double-row dentition.

Dysmastication Difficulty chewing.

Dysphagia Difficulty swallowing.

Dysphrehension Difficulty grasping food with lips and teeth.

Dysplasia Abnormal development of a part or organ.

Dyspnea Difficulty in breathing.

Ectoderm Outer embryonic germ layer that forms skin, salivary glands, hair, sweat glands, sebaceous glands, nerves, etc.

-ectomy *Medicine*. Excision of a part.

Edentate Animals that are normally devoid of anterior teeth or, animals that are normally edentulous (toothless).

Elevation Raising the mandible or closing the mouth.

Elevator Instrument used to elevate the tooth or root section out of the alveolus during extraction.

Elongation Distortion of a radiographic image because of too little vertical angulation.

Empyema The accumulation of pus in a hollow organ or body cavity.

Emphysema The abnormal presence of air in a part of the body.

Enamel A calcified dental tissue which is the hardest substance in the body, and provides great wear resistance for teeth. It covers the dentin of the crown portion of the tooth.

Enamel cuticle (Nasmyth's Membrane) A thin membrane that covers the crown of a tooth at eruption.

Enamel hypoplasia Condition in which the enamel layer is thin or reduced.

Enamel lamellae Imperfections or cracks in enamel caused by trauma or imperfect enamel formation.

Enamel organ Ectodermal epithelial structure that leads to the formation of tooth enamel. Interspersed with enamel plates in Equine Type I enamel.

Enamel pearls (enamelomas) Small enamel growths on the root of the teeth; considered abnormal structures.

Enamel prisms Basic enamel unit running from the dentinoenamel junction to the surface enamel.

Enamel rod Individual pillars of enamel formed by ameloblasts.

Endo- (prefix) Within.

Endoderm Inner germ layer of an embryo that forms the epithelial lining of organs such as the digestive tract, liver, lungs, and pancreas.

Endodontics Branch of dentistry involved with treating the pulp and root canals of teeth.

Endoscope Instrument used for examining inside hollow organs and the abdominal cavity.

Endoliths Calcifications within the endodontic system; sometimes referred to as pulp stones.

Epiglottis Mucosal-covered cartilage that helps cover the laryngeal opening during swallowing.

Epistaxis Hemorrhage from the nose.

Epithelial attachment Interface at the base of the gingival sulcus or periodontal pocket that unites the gingiva to the tooth.

Epithelium Thin cellular layer that covers the external and internal surfaces of the body or organs.

Epulis The most common form of benign growth arising from the periodontal ligament. It may be fibromatous, ossifying, or acanthomatous.

Erosion External loss of tooth hard tissue due to a chemical process without active bacterial involvement.

Eruption of teeth The process of movement of tooth from the alveolus into the oral cavity.

Eruption cysts (pseudocysts) Enlarged areas of soft tissue at the developing apices of immature permanent cheek teeth. These cysts can cause bony swellings on the ventral surface of the mandible, less commonly on the maxillary bones in 2–4 year old horses.

Eruption times General times for anticipated eruption of teeth.

Eruptive stage Period of eruption from the completion of crown formation until the teeth come into occlusion. The prefunctional eruptive stage occurs at the beginning, before the teeth move into occlusion.

Exfoliation Shedding or loss, e.g., of a primary tooth.

Exodontia Extraction of teeth.

Exostosis Local deposition of new bone that projects beyond the normal limits of the skeleton.

Exostoses Small extra growths of bone on a surface; usually seen on the buccal cortical plate.

Exothermic A chemical reaction that generates heat.

External fixation Any method by which fractured bones are supported by means outside the body.

External fixator Method by which fractured bones are immobilized with percutaneous pins that are joined outside the body.

Extirpate To completely remove or destroy a part or organ.

Extirpation Complete surgical removal or destruction of a part, such as a pulp.

Extra-oral Outside the mouth.

Extract To pull out or remove.

Extrinsic Originating outside a structure.

Extrusion Movement of a tooth further out of the alveolus, typically in the same direction as normal eruption.

Eye teeth *See* cuspid.

Facial Term used to designate the outer surfaces of the teeth collectively (buccal or labial).

Facial nerve Cranial nerve VII, innervating the facial muscles of expression and caudal belly of the digastric muscle.

Facultative anaerobes Bacteria that can live in either aerobic or anaerobic conditions.

Familial Used to describe conditions that affect a family to an extent that is considered greater than expected by random chance or circumstance.

FDI system System for tooth identification promulgated by the Federation Dentaire Internationale (International Dental Federation).

Fetid Having a smell of decaying matter.

Fibroma Benign tumor of mesodermal origin.

Fibrosarcoma Malignant tumor comprised of fibrous connective tissue.

Fibrous dysplasia Incomplete differentiation of fibrous tissues. Replacement of bone as a result of parathyroid dysfunction.

Filiform papillae Small pointed projections pointing caudally that heavily cover most of the dorsum of the anterior two-thirds of the tongue.

Filing Grinding or rasping of dental tissues.

Fissure A small crack, e.g., an enamel fracture in a cheek tooth.

Fistula A tract (duct) leading from an internal cavity in the body surface or from one body cavity e.g. from a paranasal sinus to the mouth (oromaxillary fistula) or from the oral to the nasal cavity (oronasal fistula); usually formed following by the incomplete closing of a wound or abscess.

Fixer solutions Chemicals used to preserve and enhance the latent image on the radiographic film.

Flap Portion of mucous membrane or skin separated from the surrounding tissues except for at least one edge.

Floating *See also* Rasping. *Veterinary*. The process of smoothing down the sharp buccal or lingual enamel overgrowths (points) on the cheek teeth of horses. The act of using rasps to remove sharp edges from teeth.

Fluorosis Disruption in the mineralization of forming teeth due to excess ingestion of fluoride, often seen as chalky white spots or discoloration of the enamel.

Focal film distance (FFD) Distance from the focal spot on the tube's target to the film.

Follicle Fibrous sac which surrounds the developing tooth germ and by which it is attached to the oral mucosa.

Follicular cyst Dentigerous cyst or dilation of the follicular space around the crown of a tooth that is unerupted or impacted.

Foramen A small circular opening or passage e.g. where the mental nerve leaves the mandible.

Frenulum Fold of alveolar mucosa forming a noticeable ridge of attachment between the lips and gums. *See* frenum.

Frenum Fold of skin or lining tissue that limits the movement of an organ (e.g., tissue under the tongue).

Fulcrum A device used to increase leverage of dental equipment.

Functional occlusion Active tooth contact during mastication and swallowing; also called dynamic occlusion.

Furcation Point at which roots diverge. Being divided into parts. Teeth with multiple roots have bi- or trifurcation, as the point where the roots meet coronally.

Gag An instrument to prevent the closure of the mouth during oral examination or surgery.

Galvayne' Groove A groove in the labial surface of 103/203 (upper permanent corner incisor of the horse) which begins to appear at approximately 10 years, is half way down incisor at 15, fully down at approximately 20, half worn away at 25 and absent at 30 years of age. This feature has been shown to be an inaccurate indication of age.

Gamma radiation Radiation of the same approximate wavelength as x-radiation that is naturally occurring rather than man-made.

Gemination Disorder in which the developing bud attempts to split but fails to do so completely, resulting in duplication of part of the tooth but not total twinning.

Gene A unit of information in DNA that codes for a particular disease or trait.

General anesthesia Controlled, drug-induced unconsciousness, whereby pain, voluntary muscle movement and an effective swallowing reflex are eliminated.

Genetic Term describing the condition of being hereditary.

Genotype The genetic makeup of an animal.

Gingiv- or **gingivo-** (prefix) Denoting the gingiva.

Gingiva Keratinized oral membrane that immediately surrounds the teeth and alveolar bone.

Gingival Of or pertaining to the gums.

Gingival crest Most occlusal or incisal extent of gingiva.

Gingival crevice (sulcus) Subgingival space that under normal conditions lies between the gingival crest and the epithelial attachment to the adjacent peripheral cementum.

Gingival fibers Periodontal fibers in the gingiva.

Gingivitis Inflammation of the gums.

Glass ionomers Dental restorative compounds that chemically bind to enamel and dentin by ions forming salts that bond to the calcium in the tooth, even if slight moisture is present.

Glossectomy Surgical removal of part or all of the tongue, or a lesion of the tongue.

Glossitis Inflammation of the tongue.

Glossoplegia Paralysis of the tongue, either unilateral or bilateral.

Gnathic Relating to the jaw, meaning the mandible in modern usage.

Gomphosis Type of fibrous joint in which a conical object is inserted into a socket and held.

Granuloma Localized mass of reactive connective tissue associated with an area of chronic suppuration and/or healing.

Gutta percha An ionomer of rubber extracted from the sap of certain tropical trees. Endodontic filling agent that is about 60 per cent crystalline and slightly viscoelastic.

Gum Oral mucus membrane, the firm specialized epithelium covering the oral aspect of the jaws and attached to the cementum of the base of the clinical crowns just above the alveolar crest.

Halitosis Malodorous or foul breath.

Hard palate Bony vault of the oral cavity proper covered with soft tissue.

Hard tissue Calcified or mineralized tooth tissues or bone.

Haussman gag A metal-framed, ratchet-operated device used to keep the mouth of herbivores, especially horses, open for examination or treatment.

Haversian system System of blood vessels located within the bones to provide nourishment.

Hemimandibulectomy Excision of half of the mandible.

Hemisection To cut in half.

Hemostasis To arrest hemorrhage.

Hereditary Genetically determined; passing or capable of passing from parents to offspring.

Hertwig's epithelial root sheath Downgrowth of the inner and outer enamel epithelium that outlines the shape and number of the roots.

Heterodont The feature of having more than one type (size, shape) of tooth represented in the dentition, such as incisors, cuspids, premolars, and molars.

Homodont The feature of having all teeth that are of the same general shape or type, although size may vary.

Hook A sharp narrow overgrowth developed on a tooth through abnormal wear, e.g. On 1st or 6th cheek teeth. Also note 7 or 9 year incisor hooks, now shown to be inaccurate for age determination.

Horizontal ramus That portion of the jaw composed of the body and symphyseal area of the mandible.

Hydroxyapatite Calcium and phosphate containing crystals found in hard substances of the body such as bone, cementum, dentin, and enamel; sometimes spelled hydroxylapatite.

Hyoid apparatus Bony structure originating from 2nd and 3rd branchial arches. Attached to petrous part of temporal bones and supports the root of the tongue, pharynx and larynx.

Hyper- (prefix) Exaggerated, excessive.

Hypercementosis Increased thickness of cementum, usually seen at the apex of the root.

Hyperemia Congestion of blood, seen in pulp.

Hyperplasia Enlargement or overdevelopment of organ or tissue through increased production of cells.

Hyperplastic Affected by hyperplasia.

Hyperptyalism Excess salivation.

Hypodontia Condition in which some teeth are missing.

Hypoplasia Reduced or inadequate tissue formation.

Hypoplastic enamel Thin enamel, commonly seen in conjunction with enamel hypocalcification. *See* enamel hypoplasia.

Hypsodont Teeth which have a limited growth period but prolonged eruption throughout the life of the animal (In contrast, anelodont teeth, as in rabbits have permanent growth and eruption throughout life, brachydont teeth such as humans have a limited growth and eruption time).

Iatrogenic Physician induced injury that is caused by or created by treatment e.g., fractures of 311 or 411 using a guillotine or fractures of cheek tooth caused by dental shears in a young horse.

Idiopathic Disease of unknown origin.

Impacted teeth Teeth which have been prevented from erupting by mechanical obstruction, usually compression from the two adjacent teeth. Impactions may cause large eruption cysts to develop at the apex of the impacted tooth and focal hard swellings ("3 and 4 year old bumps") on the mandible or maxilla.

Implant *Dentistry*. Intra-osseous, biocompatible structure placed in the alveolar bone which is used as a support in prosthodontics.

Impression Mold taken of the teeth and/or intra-oral contours of the jaw for the preparation of a replica model.

Impression material A plastic substance used in the making of a mold of the teeth and/or the contours of the jaw.

Impression tray Receptacle usually custom-made in veterinary use, to fit the jaw being worked on, for carrying the impression material.

Incisal Coronal portion or direction in incisors.

Incisal bone The premaxilla, rostral most area of upper jaw that accommodates the maxillary incisors and is formed solely by the medial nasal process; also known as the primary palate.

Incisivomaxillary suture Articulation of the incisive bone and the maxillae.

Incisors Teeth found at the front of the horse's mouth (e.g. all teeth embedded in the premaxilla are incisors by definition as are those situated in the rostral mandible). Incisors are used for grasping (prehension) of food. In horses there are normally 12 deciduous and 12 permanent incisors (Triadan 101-3; 201-3; 301-3; 401-3).

Incisive bone (premaxilla) The bone attached to the rostral aspect of the maxilla which bears the upper incisors.

Incline planes Orthodontic appliances designed to make contact with the cusps or incisal edges of the teeth of the opposing occlusion to stimulate tooth movement directed by the incline.

Inferior Indicating the relative position of a structure that is lower than others specified when the body is in an anatomical position.

Inferior border of mandible Lower edge of the lower jaw.

Inflammation Reaction of living tissue to infection or injury.

Infra- (prefix) In *anatomy*, indicating a position beneath the structure being qualified. In *dentistry*, indicating a position apical to the structure being qualified.

Infundibulum Enamel infoldings found in centers of incisor and the upper cheek teeth that are filled (or partially filled) with cementum. The single incisor infundibulum is termed the "cup." There are two infundibulae in each upper and none in the lower cheek teeth.

Insidious Slow or gradual onset. Refers to a disease indicates that it does not exhibit early symptoms of its onset or progress, (e.g. has a gradual and slow onset).

Interceptive orthodontics Generally considered to be the extraction or recontouring (crown reduction) of primary or permanent teeth that are contributing to alignment problems of the permanent dentition.

Interdental Located between teeth.

Interdental (interproximal) space The space between two adjacent teeth. Also used to describe the space between incisors and cheek teeth (e.g. "bars of the mouth").

Internal fixation Surgical stabilization of fracture with pins, plates, screws, etc. within the affected bones.

Interproximal Between the proximal surfaces of adjoining teeth in the same arch.

Interproximal space The space between two adjoining surfaces such as teeth (can be used interchangeably with interdental).

Interradicular septa Bony partitions between adjacent teeth.

Intradental oral cavity (IDOC) Space whose boundaries are the lingual and palatal margins of the teeth.

Intrinsic Lying entirely inside a structure.

Intrinsic muscles of the tongue Unit of muscles that produces the complicated protrusion and prehensile movement of the tongue. They are innervated by the hypoglossal nerve.

Isognathism Condition of having equal jaw widths, in which the premolars and molars of opposing jaws align with the occlusal surfaces facing each other, forming an occlusal plane.

Jaw The upper jaw is formed by the premaxilla (incisive) and maxillary bones and the lower jaw is formed by the mandibular bones, both jaws contain the teeth.

Juvenile ossifying fibroma Benign, locally invasive gingival tumor.

Keratin Substance that makes up the surface cells of skin, hair and hooves.

Labium (pl. labia) Latin for "lip".

Labial Of or pertaining to the lips. Also, as a direction, towards the lips or the rostral aspect of mouth.

Lamina dura Radiographic term denoting the cribriform plate, bundle bone, and the dense alveolar bone surrounding reserve crown and roots.

Lampas Physiologically normal swelling of the mucosa of the hard palate, often greatest just behind the upper incisors especially in young horses. This results from venous distension during tooth eruption.

Lateral Away from the median plane (is the opposite of medial) refers to the buccal aspect (outside) of teeth. A position farther from the midline of the body or median plane. Opposite of medial. (e.g. mandibular cheek teeth are lateral to the tongue). The hard palate is medial to maxillary cheek teeth.

Lateral excursion Lateral movement of the mandible relative the maxilla.

Lateral excursion to separation A measure of cheek teeth occlusion and occlusal angulation. This term refers to the point during lateral excursion of the mandible with the jaws closed when the angled occlusal surface of the cheek teeth causes separation of the incisors.

Ligament Regularly arranged group of collagen fibers that attach bone to bone.

Lingual Referring to the tongue; also, as a direction, towards the tongue used to refer to medial aspect of the mandibular cheek teeth (palatal refers to identical aspect of maxillary cheek teeth).

Lingual arteries Primary blood supply to the tongue.

Lingual frenum Fold of tissue that attaches the undersurface of the tongue to the floor of the mouth.

Lingual mucosa Thick, rough, cornified mucous membrane covering the dorsum of the tongue.

Lips Most rostral extent of the oral cavity. The upper and lower lips converge at the angles of the mouth to form its commissures.

Lophodont The feature of having teeth that have a lamellar structure of longitudinal layers of enamel and dentin that become fused with cementum, with cusps that connect to form ridges, as in the cheek teeth of the rhinoceros and elephant.

Luxation Partial or complete dislocation from a joint, as in the temporomandibular joint or a tooth, or of a tooth from its alveolus.

Lysis To dissolve or break down.

Macrodontia Teeth that are disproportionally large.

Macroglossia Oversized or large tongue.

Malar Relating to the zygoma, cheek bone.

Malignant Term to describe tumors that show an uncontrollable growth and destructive growth pattern of the tissue of origin and that may exhibit metastasis.

Malocclusion Faulty occlusion; abnormal contact of opposing upper and lower teeth.

Maleruption Improper eruption of tooth/teeth.

Malformation Failure to develop properly.

Mandible The lower jaw bone formed by the fusion of the two hemimandibles at the symphysis.

Mandibular Pertaining to the mandible.

Mandibular alveolar block Intraoral regional anesthetic nerve block achieved by injecting at the lingual mandible near the base of the coronoid process.

Mandibular arch First pharyngeal arch that forms the area of the mandible and maxillae. It is the lower dental arch.

Mandibular condyle Rounded top of the mandible that articulates with the mandibular fossa.

Mandibular foramen Opening on the medial surface of the ramus of the mandible for entrance of nerves and blood vessels to the lower teeth.

Mandibular fossa Depression on the inferior surface of the skull in the temporal bone that articulates with the condyle of the mandible.

Mandibular symphysis Point at which the two hemimandibles merge, forming the mandible.

Masseter muscle Muscle of mastication arising from the zygomatic arch and inserting on the lateral ramus of the mandible. It acts to close the mandible.

Mast cell Mesodermal cell contains granules that release histamine in inflammatory reactions.

Mastication The grinding of food by the teeth.

Masticatory mucosa Mucosa (parakeratinized or keratinized) of the hard palate and gingiva.

Masticatory surface Occlusal surfaces of teeth.

Maxilla The paired bones of the upper jaw which contains the two rows of upper cheek teeth and also contains the maxillary sinuses and contributes to the hard palate.

Maxillary Of or pertaining to the maxilla.

Maxillary cheek teeth row Upper cheek teeth.

Maxillary sinuses Paired paranasal sinuses located in the maxillae medially and dorsally to the maxillary fourth premolars.

Maxillofacial Structures including and covering maxillary and facial bones.

Meatus A naturally occurring canal or channel.

Medial Opposite of lateral.

Medial pterygoid muscles Muscles of mastication arising from the sphenoid bone and inserting on the condyle and articular processes of the mandible. They serve to close the mandible.

Median line Vertical line that divides the body into right and left (e.g., the median line of the face).

Median plane A plane running vertically (dorsoventrally) down the midline of a horse from nose to tail.

Median raphe Midline of the palate dividing the right and left sides.

Melanoma Mesodermal origin tumor containing pigment bearing melanocytes.

Mental Relation to the chin.

Mental foramen Foramen on the lateral side of the mandible, below the premolars.

Mental regional block Intraoral regional anesthetic nerve block achieved by injection at the largest mental foramen. It provides analgesia to the incisors, canines, and first two premolars.

Mesaticephaly Condition marked by a head shape of medium proportions.

Mesenchymal cells Embryonic connective tissue that begins the development stage of the dental papilla and the dental sac.

Mesenchyme Connective tissue derived from mesoderm.

Mesial Toward or situated in the middle (e.g., toward the midline of the dental arch).

Mesial drive Phenomenon in which the permanent molars continue to move mesially after eruption.

Mesocephaly Condition marked by a balanced facial profile, somewhere between dolichocephalic and brachycephalic.

Mesoderm Middle germ layer of the embryo that forms connective tissue, muscle, bone, cartilage, blood, etc.

Metaplasia The transforation of one type of tissue into another.

Metaplastic calcification Pathological deposition of calcium in soft tissues.

Metastasis Dissemination of tumor cells to other parts of the body.

Methyl methacrylate Liquid monomer used in the manufacture of acrylic resins by mixing it with a powder polymer.

Microdontia Teeth that are disproportionally small.

Microglossa A small tongue. Also known as bird tongue.

Midline Imaginary line that divides the body into right and left halves.

Midsagittal plane Imaginary plane that divides the body vertically into right and left halves.

Mixed dentition The feature of having primary and permanent teeth in the dental arches at the same time.

Molarization Morphogenic appearance of premolar teeth to resemble molar teeth.

Molars Grinding cheek teeth that have no deciduous predecessors in the dental arcades (e.g., the last three grinding cheek teeth). (Triadan 109-11; 209-11; 309-11; 409-11). The term molars is also wrongly used to refer to all of the 6 cheek teeth.

Monkey mouth Sow mouth. Protruding mandible.

Monophyodont The feature of having only one set of teeth that erupt and remain functional throughout life, i.e. there are no deciduous teeth.

Morphology Study of the form and structure of an organism or part of it.

Mottled enamel Enamel that is opaque or chalky and may be discoloured due to its porous nature.

Mouth Entrance to the oral cavity.

Mouth speculum Mechanical device used to hold the mouth open.

Mucobuccal fold Point at which the oral mucosa and the top or bottom of the vestibule turn toward the alveolar ridge.

Mucocele *See* sialocele.

Multiple root Tooth with multiple roots.

Nares Nostrils.

Nasal septum Wall between the left and right sides of the nasal cavity, made up of the ethmoid and vomer bones.

Nasmyth's Membrane Membrane covering the surface of the tooth crown at the time of eruption.

Necrosis The death of organis tissue; hence, necrotic.

Neoplasia A new growth or tumor.

Neoplasm Benign or malignant tumor, morbid mass of tissue growing at an abnormal rate.

Newborn gingival cyst Cyst arising from the remnants of dental lamina in newborn animals.

Nonsuccessional (nonsuccedaneous) teeth Permanent teeth (classically molars) that do not succeed a deciduous counterpart.

Object film distance (OFD) Distance between the film and the object during radiography. Minimizing OFD can reduce distortion.

Obturation The process of filling, packing as in endodontic filling of pulp cavities.

Occluding Contacting opposing teeth.

Occlusal Articulating or biting surface; the coronal surface of some premolars and molars.

Occlusal plane Side view of the occlusal surfaces.

Occlusal relationship Way in which the maxillary and mandibular teeth touch each other.

Occlusal surface Surface of a tooth within the marginal ridges, that contacts the corresponding surfaces of antagonists during closure of the mouth.

Occlusion Surface-to-surface contact between opposing teeth.

-odontics (suffix) Indicating a dental subject or discipline.

Odonto- or odont- (prefix) Relation to teeth; indicating toothed.

Odontoblast Dentin-forming cell that originates from the dental papilla.

Odontoblastic cell layer Layer in the pulp that is closest to the dentinal tubules.

Odontoblastic process Cellular extension of the odontoblast, extending along the full width of the dentinal tubules.

Odontogenic cyst or tumor Lesions arising from cellular components of the developing tooth structure.

Odontoma Mixed odontogenic tissue tumor containing both epithelial and mesenchymal cells. It may be either compound (disorganized mass) or complex (with denticles).

Oligodontia The absence of one or more teeth.

-oma (suffix) Indicating a tumor.

Opaque Not easily able to transmit light.

Open fracture A fracture where there is a breach in the overlying skin or mucous membranes.

Operculum Persistence of a thick, fibrous gingiva over a partially or even fully erupted tooth.

Oral cavity (cavum oris) Area extending from the lips to the oral pharynx at the level of the palatine tonsil.

Oral cavity proper Area extending from the alveolar ridge and teeth to the oral pharynx. It does not include the vestibule.

Oral mucosa Stratified squamous epithelium running from the margins of the lips to the area of the tonsils and lining the oral cavity; also known as oral mucous membrane.

Oral mucous membrane *See* oral mucosa.

Organic matrix Noncalcified framework in which crystals grow.

Oro- (prefix) Combining form indicating oral, mouth.

Oronasal fistula An opening between the oral and nasal cavities - usually is a complication of extraction or periapical abscess of the 1st three upper cheek teeth.

Oropharynx Area between the soft palate and the base of the tongue.

Ortho- (prefix) Straight.

Orthodontic acrylics Materials used to form a framework or base structure from which various inclines, springs, arch wires, or expansion devices can be attached.

Orthodontics That area of dentistry concerned with the supervision and guidance of the growing dentition and correction of the mature dentofacial structures. It involves those conditions that require movement of teeth and/or correction of malrelationships of the jaws and teeth and malformations of their related structures.

Orthognathic surgery Surgery of mandibles to correct tooth alignment.

Osseoconductive Characteristic of a product that aids in regenerating new bone in an osseous site. Almost all guided-tissue regeneration products are osseoconductive.

Osseoinductive Characteristic of a product that aids in the generation of new bone in any site, even muscle tissue.

Autogenous bone grafts and bone morphogenic protein can do this; however, freeze-dried bone and irradiated bone are not osseoinductive because the necessary cells have been killed by treatment of this product.

Osseointegration Process in which a material's surface becomes attached or bonded to bone; also known as functional ankylosis. In the process, metal oxides on the surface of an implant bond to bone.

Osseous wiring Placement of wires in direct contact with bone to provide reduction and support to segments of a bony fracture.

Ostectomy Removal of osseous defects and infrabony pockets by the removal of bony pocket walls.

Osteo- (prefix) Indicating bone.

Osteoblasts Cells that form bone.

Osteoclasts Multinucleated cells responsible for destroying bone, as well as cementum and dentin.

Osteocytes Osteoblasts that have surrounded themselves with bone.

Osteogenic Bone producing.

Osteoid Bone-like.

Osteoma Benign bone tumor.

Osteomyelitis Infection of bone marrow.

Osteoplasty Shaping of bone to restore its physiologic contour.

Osteosarcoma Osseous tumor of the mandible or maxilla that is locally invasive but has less metastatic potential than its counterpart in the appendicular skeleton.

Osteotome Bone cutting chisel.

Osteotomy Surgical operation of cutting through a bone.

Overbite Relationship of the teeth in which the incisal edges of the maxillary anterior teeth extend below the incisal edges of the mandibular anterior teeth when the teeth are placed in a centric occlusal relationship.

Overjet *See* overlap, horizontal.

Overlap, horizontal (overjet, overjut) Facial projection of the upper anterior and/or posterior teeth beyond their antagonists in a horizontal direction.

Overshot See Retrognathism.

Palatal Pertaining to the palate or roof of the mouth.

Palatal surface Lingual (medial) surface of the maxillary teeth.

Palate Roof of the mouth is hard palate to level of 111, 211 where it then joins the soft palate.

Palatine artery Large artery that lies just medial to the upper arcades of cheek teeth at the edge of the hard palate.

Palatine rugae *See* rugae.

Palliative Treatment that alleviates the severity of pain or disease without curing it.

Papilla Wart-like lesion.

Paranasal Around the nose, as in paranasal sinuses.

Parotid duct Duct formed by two to three tributaries from the parotid salivary gland. It opens at the parotid papilla at the level of the caudolateral root of the maxillary fourth premolar.

Parotid salivary gland "V"-shaped serous gland located beneath the ear and behind the posterior border of the mandible and the temporomandibular joint. It has superficial and deep portions. *See* parotid duct.

Partial anadontia Condition in which some but not all of the teeth are missing.

Parrot mouth Overbite, overjet, mandibular brachygnathism.

Passive eruption Condition in which the tooth does not move but the gingival attachment moves farther apically.

Pathologic movement Orthodontic tooth movement that occurs when a heavy force is exerted, resulting in necrosis of periodontal tissues on the pressure side and poor to no deposition of bone on the traction side.

Peg tooth A small tooth with a cone-shaped crown. *See also* microdontia.

Pellicle A thin film of salivary proteins found on the clinical crown of teeth.

Percutaneous Through the skin.

Percutaneous skeletal fixation Use of pins or wires extending from fracture fragments and secured externally with an additional device (e.g., rod or acrylic tubing).

Peri- (prefix) Around.

Periapical Around the apex of a tooth root.

Periapical abscess Active infection around the root tip or apex, with suppuration.

Periapical cyst Cystic reaction around the root tip, often developing from epithelial cells from the rests of Malassez.

Periapical granuloma Granulomatous reaction around the root tip or apex without demonstrable bacteria.

Pericoronitis Inflammation of the gingiva.

Periodontal Literally means "around or near the teeth;" surrounding a tooth; usually used to refer to gums or the other soft tissues (periodontal membrane/ligaments) which attach the teeth to the socket. Also refers to the socket (alveolus).

Periodontal disease Inflammation of the gingiva or periodontium, their active ressive alternation or their alternation state with or without disease.

Periodontal ligaments Tough fibers which secure the cement on the periphery of the tooth to the bony alveolus; act as shock absorbers to dampen occlusal pressures.

Periodontal membrane Collagen fibers attached to the teeth roots and alveolar bone, serving as an attachment of the tooth to the bone. (See Periodontal ligament)

Periodontal pocket Space created by periodontal erosion of gingival sulcus.

Periodontal probes Flat or round-tipped instruments that have various lengths in millimeters marked on them.

Periodontitis An active disease state of the periodontium.

Periodontium Supporting tissues surround the teeth.

Periodontology or Periodontics Area of dentistry concerned with the study and treatment of the diseases involving the gingivae and the supporting tissues of the teeth.

Periosteum Tough elastic membrane covering the surface of bones; fibrous and cellular layer covering bones and containing cells that can become osteoblasts.

Periradicular osteomyelitis Radiographic osteopenia and expansion effects of the alveolus seen in some cases of chronic pulpal inflammation.

Peritubular dentin Dentin immediately surrounding the tubule. It is slightly more calcified than the rest of the dentin.

Permanent teeth (dentes permanetes) Final or lasting set of teeth that are typically of a very durable and lasting nature (opposite of deciduous).

Phenotype External appearance or performance of an animal.

Phy- (prefix) To generate.

Physiologic mobility Degree of tooth movement that can be considered normal, limited to the width of the periodontal ligament.

Physiologic movement Movement in orthodontic treatment that occurs when a light-to-mild force is applied and acts as a stimulus to initiate cellular resorption on the pressure side and deposition of bone in the tension side.

Pica An intense desire to ingest nonfood items.

Plaque A soft coating essentially bacteria together with some mucins and proteins that invariably forms on teeth; remains thin if they receive natural cleaning, as most parts do in the horse; builds to great thickness in areas that are not naturally cleaned and is the cause of calculus build up in these areas. Precursor to build-up of dental calculus and tartar; bacterial/organic/inorganic matrix involved in mineral leaching process.

Pleurodont Tooth that has no root but is attached to the lingual or palatal surface of the jaws.

Plexus A complex network of nerves, blood vessels or lymphatics.

Pocket An abnormally deep defect between the gingiva and the crown or root surface of the tooth.

Posterior Behind or toward the back/caudal part. Situated toward the back, such as premolars and molars.

Polydontia Condition of having more than one supernumerary tooth.

Posterior teeth Teeth of either jaw to the rear of the incisors and canines.

Prefunctional eruptive stage See eruptive stage.

Prehensile Adapted for grasping.

Premaxilla Bony area of the upper jaw that includes the alveolar ridge for the incisors and the area immediately behind it in primates. Also called the incisive bone.

Premolars Permanent teeth that replace the primary molars, designed to help hold and carry, like cuspids and break food down into smaller pieces, like molars. Also known as bicuspids. Cheek teeth that have deciduous predecessors (Triadan 106-8, 206-8, 306-8, 406-8). Have evolved to be similar to molars of horses. Premolar 1 "wolf tooth" has no predecessor.

Primary dentin Dentin formed from the beginning of calcification until tooth eruption.

Primary dentition Deciduous teeth; also known as first set of teeth (baby teeth, milk teeth).

Primary teeth See Deciduous teeth.

Primordial cyst Cyst resulting from the degeneration of the stellate reticulum of the enamel organ found in place of a tooth.

Prognathism (underjet) Protrusive jaw ("sow mouth," "Monkey mouth"); the mandibular incisors are more rostral than the upper incisors. The opposite of brachygnathism.

Prophylaxis, prophylactic Preventive care; in equine dentistry means regular dental maintenance. Also may refer to tetanus antitoxin and antibiotic administration when required.

Prosthesis Artificial device to replace missing natural parts. *Dentistry*. Crown denture or bridge.

Proximal See approximal. *Anatomy*. Situated close to the center of the body, the median plane or the point of origin of an organ or limb.

Ptyalism Excessive flow of saliva.

Pulp (dental) Highly vascular and innervated connective tissue contained within the pulp cavity of the tooth. It is composed of arteries, veins, nerves, connective tissues and cells, lymph tissue and odontoblasts.

Pulp canal Canal in the root of a tooth that leads from the apex to the pulp chamber. Under normal conditions, it contains dental pulp tissue.

Pulp cavity Entire cavity within the tooth, including the root canal, pulp chamber and horns. See Pulp chamber.

Pulp chamber or pulp cavity Canals in the central portion of tooth that houses connective tissue, nerves and blood vessels and give vitality to the tooth.

Pulp exposure Unnatural opening of the pulp chamber by pathologic or mechanical means.

Pulp stones Small dentin-like calcifications found in the pulp.

Pulpal necrosis Partial or total pulpal death.

Pulpectomy Extirpation of the entire pulp.

Pulpitis Inflammation of the pulp that may be reversible or irreversible.

Pulpotomy Surgical removal of a portion of the pulp in a vital tooth.

Purulent Condition involving the presence of pus.

Pus Yellow, white or green fluid that is the product of inflammation composed mainly of dead leucocytes, plasma and liquefied tissue cells.

Pyorrhea A lay term denoting periodontal disease.

Quadrants One-fourth of the dentition. The four quadrants are divided into right and left, maxillary and mandibular.

Quidding The term used to describe the dropping of partially masticated boluses of food from the mouth.

Radicular ankylosis Loss of part or all of the periodontal ligament, resulting in fusion of root cementum and socket bone.

Radicular hyposodont Subdivision of hyposodont dentition, sometimes called closed rot, in which true roots erupt additional crown through most of life. These teeth eventually close their root apices and cease growth. As teeth are worn down, new crown emerges from the reserve or submerged crown of the teeth.

Radiolucent Offering little or no resistance to the passage of x-rays.

Radio-opaque Offering resistance to the passage of x-rays.

Ramp Pathological exaggeration of distal upward slope of mandibular cheek teeth.

Ramus The vertical ramus is the portion of the mandible that is covered by the masseter muscles and forms the angle of the jaw and temporomandibular joint. The horizontal ramus houses the cheek teeth.

Ranula Salivary retention cyst (sialocele) located under the tongue caused by blockage of the sublingual duct or gland.

Rarefaction Loss of bone substance that creates an area of radio-opacity on radiographic examination.

Rasping Floating of teeth.

Recession Migration of the gingival crest in an apical direction away from the crown of the tooth.

Recessive An allele that produces an effect on the phenotype only when present in a double form.

Removal appliances Orthodontic devices designed to be easily and routinely removed and then reinserted.

Reparative dentin (tertiary dentin) Localized formation of dentin in response to local trauma such as occlusal trauma or caries. This type of dentin forms at the tubule access once a dead tract is formed, as well as being formed by differentiated mesenchymal cells that migrate from the pulp.

Repulsion Exodontia by means of forces applied to root apices.

Reserve crown The portion of the crown which is yet to erupt into the oral cavity.

Resorption Physiologic removal of tissues or body products as of the root of deciduous teeth or of some alveolar process after the loss of the permanent teeth.

Restorative dentistry Area of dentistry that is concerned with treatment, repair and conservation of teeth broken down through trauma or caries.

Retro- (prefix) from behind, backwards.

Retrognathism Anatomical relationship where the mandible lies in an excessively caudal/retrusive position to the upper jaw. *Veterinary*. Overshot.

Retarded eruption Delayed eruption of teeth from a variety of influences.

Retrograde Reverse approach. In endodontics indicates root filling from an apical approach.

Reversible pulpitis Inflammation of the pulp that can be resolved, returning the pulp to a healthy state.

Rhinitis Inflammation of the mucous membrane lining of the nasal passage.

Ridge A linear elevation. May be marginal, triangular, cusp, incisal, oblique or transverse.

Root The enamel-free area at the apex of a tooth. That portion of the tooth normally embedded in the

alveolar process and covered with or fully composed of cementum.

Root bifurcation That point at which a root trunk divides into two separate branches.

Root canal The apical opening(s) of the pulp chamber(s) of the tooth. These openings are wide in younger teeth (open roots) but constrict due to secondary dentin deposition in older teeth (closed roots) and move away from the apical aspect of the tooth as root cementum is laid down beneath this site in the older tooth. See pulp canal.

Root sliver Portion of root left in place after exodontia.

Root resection Cutting off of a root but not its associated portion of crown.

Root trifurcation That point at which a root trunk divides into three separate branches.

Rostral (anterior) Toward the front of the body (e.g., toward the muzzle).

Rostral hook Focal overgrowth of the rostral aspect of the 06s (usually uppers in horse with overjet, but occasionally on lowers 06s in horses with underjet.

Rugae Small ridges of tissue extending laterally across the anterior of the hard palate.

Rule of dental succession No successional and deciduous precursor should be erupted simultaneously or in competition for the same dental arcade space at the same time.

Sagittal Anatomical plane running parallel to the median (midline) plane (e.g. sagittal fracture of a cheek tooth through the infundibula).

Salivary glands Glandular system secreting saliva, a serous and mucus-like fluid that assists in the lubrication and digestion of food.

Salivary mucocele Localized collection of saliva in tissues other than a salivary gland or duct. Secondary dentin Normal physiologic dentin formed throughout the pulp cavity following eruption.

Secondary dentition Permanent dentition.

Section The process of cutting; a division or segment of a part.

Sedation Drug-induced calmed state, diminished physical activity and a reduced response to stimuli, where pain is not eliminated and an effective swallow reflex is maintained.

Selenodont dentition The feature of having cheek teeth with cusps that connect to form a crescentic outline, quarter-moon or concavoconvex ridge pattern as in the even-toed hoofed animals (order Artiodactyla) except swine.

Sensible dentin Term used to imply that a tooth is vital.

Sequestrum A detached piece of necrotic bone that is devoid of its blood supply.

Seven year hook Overgrowth of lateral corners of 103 and 203 seen at seven years of age.

Seroma Localized accumulation of serous exudate associated with surgical dead space.

Sharpey's fibers Calcified part of the periodontal ligament embedded in cementum or alveolar bone.

Shed Term used for exfoliation of deciduous teeth.

Shear mouth A wear disorder of cheek teeth where the angulation of the occlusal surfaces are increased (e.g. 45° to the horizontal plane).

Shell teeth A hereditary and/or congenital disorder of teeth in which there is a crown but little to no root development.

Sialocele Retention cyst of salivary fluids.

Sialolith Salivary stone; calcifications found in salivary glands or ducts.

Sinus Air cavity connected with the nose. *Medicine*. Epithelially lined tract between an area of suppuration and a mucous membrane surface or the skin.

Sinusitis Inflammation of a paranasal sinus or sinuses that can be due to apical infections of caudal 4 maxillary cheek teeth as well as to non-dental causes such as primary infections, cysts or tumors.

Slant mouth, slope mouth A disorder of wear where the incisor occlusion surface angle deviates from horizontal (e.g. due to eating with only one side of the mouth) is due to a cheek teeth disorder or wry nose.

Smooth mouth Age-related dental attrition with loss of clinical crowns.

Soft palate Unsupported soft tissue that extends back from the hard palate free of the support of the palatine bone.

Soft tissue Noncalcified tissues such as nerves, arteries, veins and connective tissue.

Sow mouth Monkey mouth. Protruding mandible.

Speculum Mechanical device used to hold the mouth open.

Spreader Forceps used to separate cheek teeth.

Squamous cell carcinoma (SCC) Malignant tumor of the squamous epithelium.

Star, dental star The exposed secondary dentin-filled portion of the pulp chamber on the occlusal surface of the incisors; was widely and often inaccurately used in the estimation of age of the animal.

Static occlusion Relationship of the teeth when the jaws are closed in centric occlusion.

Steinmann pin Cylindrically shaped metal rod with threaded or trochar points used as an intramedullary splint in fracture repairs.

Stellate reticulum Ectodermal and epithelial-derived middle layer of the enamel organ. It serves as a cushion for the developing enamel.

Step mouth A cheek teeth row with one or more rectangular 'step-like' occlusal abnormalities. Usually due to loss of a tooth with overgrowth of its occlusal counterpart.

Stomatitis Inflammation of the soft tissues of the oral cavity or mouth.

Stomodeum Depression in the facial region of the embryo that is the beginning of the oral cavity; the primitive mouth.

Sublingual caruncle Small elevation of soft tissue at the base of the lingual frenum that is the opening for the mandibular duct.

Sublingual fold Fold of tissue extending backward on either side of the floor of the mandible above the mylohyoid line in the canine region.

Sublingual salivary gland Smaller of the major salivary gland pairs; monostomatic portion lines within the mandibular gland; major sublingual duct to sublingual caruncle (or near); polystomatic portion of 6–12 small scattered lobules of sublingual salivary tissue; several minor sublingual ducts into lateral sublingual recess.

Subluxation Incomplete dislocation of a joint such as the temporomandibular joint or a tooth from its alveolus.

Submandibular Referring to the region below the mandible; a group of lymph nodes around the submandibular gland.

Submerged teeth Teeth covered by bone.

Successional lamina Lingual extensions developed during primary dentition from the dental lamina buds.

Successional (succedaneous) teeth Permanent teeth that replace or succeed a deciduous counterpart, typically certain diphyodont incisors, cuspids or premolars.

Sulcus Elongated valley in the surface of a tooth formed by the inclines of adjacent cusps or ridges that meet at an angle.

Supererupption Condition in which teeth have the cementoenamel junction erupted above normal. It may be either supraclusion or supraversion.

Superior Indicating the relative position of a structure that is higher than others specified when the body is in the anatomical position.

Supernumerary roots Those roots beyond the normal complement of a tooth.

Supernumerary teeth Those beyond the normal complement (extra).

Suppurate To discharge pus.

Supra- (prefix) Above.

Supraeruption Eruption of a tooth beyond the occlusal plane.

Symphysis The central rostral point of the mandible where the two parts of the jaw joint. This may remain a fibrous joint throughout life or it may ossify at birth.

Synarthrosis Any immobile or fused joint that lacks a synovial capsule; it is usually formed by fibrous tissue, cartilage or a mixture of both.

Tartar Eruption of salivary deposits on clinical crowns. Calciumhydroxyappetite.

Tattoo Mechanism of identification of horses.

Temporalis muscle Muscle of mastication arising from the temporal fossa and inserting on the coronoid process of the mandible to close the mandible.

Temporary teeth Dentes temporaui, considered to be the first set of temporary teeth that are shed at some point and replaced by permanent teeth.

Temporomandibular joint (TMJ or JAW joint) The articulation of the mandible and temporal bones of the skull.

Temporomandibular ligament Thickened part of the temporomandibular joint capsule on the lateral side.

Teratoma Tumor or group of tumors composed of tissues that would not normally occur at that site. Derived from germ cells and often containing teeth or hair.

Tertiary dentin *See* reparative dentin.

Tetracycline stain Intrinsic grey, green, yellow or brown discoloration of the dentine and enamel caused by systemic treatment with a tetracycline-based antibiotic at the time of development of that part of the tooth.

Thecodont The feature of having teeth that are firmly set in sockets.

Theory of periodontal ligament force Eruption theory that the periodontal ligaments force for occlusal maintenance also contributes to eruption.

-tomy (suffix) Surgical cutting of a part.

Tomes process (Tomes fibers) Ameloblast processes.

Tongue A mobile prehensile structure of the oral cavity used for grooming and intake of food and fluids.

Tooth A calcified structure containing dentine attached to the jaws of vertebrates occurring in or at the mouth; or in the alimentary canal of some invertebrates.

Tooth bud The formative structure of a tooth in the dental follicle.

Tooth eruption Emergence and movement of the crown of the tooth into the oral cavity.

Tooth germ Soft tissue that develops into a tooth.

Tooth migration Movement of a tooth through the bone and gum tissue.

Tooth resection Cutting off of a portion of the crown with or without its associated root structure.

Trabeculae Interlacing meshwork that makes up the cancellous bony framework.

Transverse Across a longitudinal anatomical plane or direction from medial to lateral (e.g., lingual to buccal).

Trephination Process of making an opening into a bone with a trephine (e.g., for surgical exposure of the sinuses or repulsion of a tooth).

Trephine To perforate with a trephine (see trephination). A cylindrical saw for cutting a circular piece of bone out of a skull.

Triadan nomenclature System for the precise numbering of teeth and their position. Modified and applied to many species.

Trifurcation Division of three tooth roots at their point of junction with the root trunk.

Trigeminal nerve Cranial nerve V that innervates many of the muscles of mastication.

True temporomandibular joint ankylosis Inhibited jaw movement due to a bony union across the temporomandibular joint surface.

Tushes, tusks (see canines)

Twinning disorder Condition in which there has been a complete cleavage of the splitting germination bud with the extra tooth being a mirror image of the original, not a separate tooth bud.

Twitch A loop of cord attached to a stick used to control horses during veterinary examination or treatment through pinching the upper lip by tightening the cord with the twisting action of the stick.

Ulcer Break in the skin or mucous membrane resulting in the exposure of deeper structures.

Underbite, Underjet, Undershot Sow mouth. Protruding mandible.

Ventral Anatomically, that which is below (e.g., opposite of dorsal).

Vestibule Space between the lips or cheeks and the teeth.

Vestige, vestigial The remnant of a structure that functioned in a precursor of that species (e.g. the wolf teeth of horses, canine teeth in mares).

Vice A bad habit.

Vincent's infection Acute Necrotizing Gingivits, Trench Mouth. Fusiform and spirochete (Borrelia vincentii) gum infection in man.

Vital *Dentistry*. Tooth or pulp tissue with intact innervation and vascular supply.

Vomer Bone that forms the lower part of the nasal septum.

Wave mouth An acquired disorder of wear of the cheek teeth where their occlusal surfaces have a wavelike appearance in a rostro-caudal direction.

Wry nose Campylorrhinus lateralis.

Wolf teeth (Triadan 105, 205, 305, 405). Vestigial teeth in the horse; the first premolar; small teeth mesial or lingual to the second premolar.

Wolff's law of transformation of bone "The law of transformation of bone is to be understood as the law under which, as a result of primary alteration in the form and function or of function alone of bone, there follows certain definite changes, determined by mathematical laws in its inner architecture and as certainly according to the same laws of mathematics, secondary changes in its external form".

Wry mouth Condition which one of the four jaw quadrants is grossly out of proportion to the other three causing a facial deviation from the midline.

Zygomatic arch Arch of bone on the side of the face or skull formed by the zygomatic bone and temporal bone.

Zygomatic bone Bone that forms the cheek area.

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