Exotics Made Easy



Demystifying Rabbits and Small Mammals







Madonna Livingstone

5m

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Dr Madonna Livingstone BVMS MRCVS

5m Books

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For the animals who were my companions, friends, and teachers.
I dedicate this book to all the species because *they all matter*.

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Foreword

This is the book I would have loved by my side as I started my career 18 years ago. Exotics medicine is coming on leaps and bounds and may be daunting for anyone, from new graduates through to otherwise experienced clinicians.

I have known Madonna for many years, and she has always been just a phone call away to discuss cases and give advice. Madonna is a well-respected vet and an incredible teacher. This book is written as Madonna talks, so it is as if she is there talking you through the case, which I find very helpful. She has a unique style, her calm and logical approach to any situation will help you to treat any species with confidence and achieve a thorough understanding of what's going on and to provide options for diagnostics and/or a treatment plan, as after all, 'it is just a body system'.

This book is essential whether you are a student or a registered and practising veterinary nurse or surgeon. It guides you through the most common exotic mammals you are likely to see in practice. The step-by-step approaches described can be used for absolutely any species that may appear in your consulting room: furry, feathered or scaley.

When writing a foreword, one of the key points is to highlight why the author is the best person for the job. That is easy. This book is written based on Madonna's invaluable 22 years' experience working hands on with these species. Throughout the book there are helpful hints and figures illustrating how she approaches certain cases. Madonna is a dedicated, well respected and loved teacher to her vet students at Glasgow University and is the perfect author for a book on demystifying exotics. This is what she teaches on a daily basis, either in the clinic or in a lecture theatre. Madonna is a full-time clinical vet in an independent practice with an exotics (now often called non-traditional companion animals) caseload of over 70%. I love her ethos in that all species deserve the same high-level care and that the human–animal bond is not species specific. This is highlighted in every chapter throughout her book, that no matter the species, there is an animal, a family member, deserving the very best care. She is also a university lecturer teaching clinical skills and exotics medicine and surgery, nominated by her students and winning the external lecturer of the year award in 2021–2022 and 2022–2023, as well as the 2016 Aleen Cust Special Recognition Award for helping other vets with exotics too. In addition to this she is also a laboratory consultant for non-traditional companion animals, so someone who lives and breathes hands on clinical medicine, who loves teaching and has a full understanding in interpretating lab tests to help other vets achieve a diagnosis for their own cases. In her spare time, she often writes for peer-reviewed journals and provides bespoke in-house CPD for practices and speaks for various organisations on exotics.

Madonna has been awarded a distinction in her Parrot Studies and Behaviour Diploma and was also awarded the UK Rabbit Vet of the Year Award 2022 by Burgess and RWAF, with welfare being paramount in how she approaches her work. She has been striving to improve non-traditional companion animal welfare throughout her career and through her work as a committee member for the Scottish branch of the British Small Animal Veterinary Association (BSAVA).

I have asked colleagues to also comment on how she has impacted them, and the responses are unanimous.

> Madonna has been a huge influence on my professional career and prospects as a veterinary surgeon. Her unending passion and devotion to everything around her, clients, patients, students and co-workers is truly inspiring and has become a personal aspiration for myself of the vet I want to be. Having her encyclopaedic knowledge condensed into writing will be invaluable to many vets, myself included! (Dr Fergus MacNeill BVMS MRCVS)

> I consider myself incredibly lucky to have been a student of Madonna's. From my earliest days in vet school, her contagious passion for exotic medicine was nothing short of inspiring. The knowledge and skills she shared have given me the confidence to tackle my own exotic cases as a new graduate, even if I still on occasion 'hit my panic button', as Madonna would say! Madonna has been instrumental in shaping my veterinary journey, and for that, I am forever grateful. (Dr Eve Maden BVMS MRCVS)

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This book will stop us 'hitting that panic button' and enable you to approach any species with confidence and excitement, and to enjoy treating these incredible species that we are lucky to have entrusted into our care.

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Thank you also to my incredible team, without them the work we do would not be possible! Veterinary medicine is genuinely a team effort with each member playing an invaluable role.

Lastly but by no means least, thank you to Ken McClean. A boss (he always joked I only let him think he was my boss), a mentor, and a true friend. Taken too soon and sorely missed.

Supporting videos

To access the series of videos 2.1–10.1 demonstrating techniques described in the text please visit: https://5mbooks.com/exotics-made-easy-videos



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Abbreviations

ACE	angiotensin-converting enzyme
ACEI	angiotensin-converting-enzyme inhibitors
AGD	adrenal gland disease
ALKP	alkaline phosphatase
AV	atrioventricular
BAL	bronchoalveolar lavage
BUN	blood urea nitrogen
CAR	cilia associated respiratory
CDRM	chronic degenerative radiculomyelopathy
CNS	central nervous system
COP	chemotherapy protocol with cyclophosphamide, vincristine, and prednisolone
CT	computed tomography
DCM	dilated cardiomyopathy
ECG	electrocardiogram
EDTA	ethylenediaminetetraacetic acid
EMLA	eutectic mixture of local anaesthetics
ESF	external skeletal fixator
ET	endotracheal tube
FNA	fine needle aspirate
FRECV	ferret enteric coronavirus
FRSCV	ferret systemic coronavirus
GA	general anaesthetic
GDV	gastric dilatation and volvulus
GI	gastrointestinal
GnRH	gonadotropin-releasing hormone
GUVS	Glasgow University School of Veterinary
HAC	hyperadrenocorticism
HaPyV	hamster polyomavirus
HCM	hypertrophic cardiomyopathy
IgG	immunoglobulin G
IgM	immunoglobulin M
IM	intramuscular
IO	intraosseous
IOP	intraocular pressure
IP	intraperitoneal
IU	international units
IV	intravenous
IVDD	intervertebral disc disease
LA	long axis
LRD	lower respiratory tract disease
MCT	mast cell tumours
NLD	nasolacrimal duct
NSAID	non-steroidal anti-inflammatory drugs
OA	osteoarthritis
OTM	oral transmucosal
OV	ovariectomy
OVH	ovariohysterectomy
PCR	polymerase chain reaction
PCV	packed cell volume
POC	point of care
PRPSS	pet rabbit pododermatitis scoring system
RD	respiratory disease
RR	respiratory rate
SA	short axis
SC	subcutaneous
SCC	squamous cell carcinomas
SDAV	sialodacryoadenitis virus
TMJ	temporomandibular joint
TPS	transfixation pin splinting
TSH	thyroid-stimulating hormone
TV	tidal volume
UPC	urine protein creatinine
URD	upper respiratory tract disease
URT	upper respiratory tract
USG	urine specific gravity
UV	ultraviolet
VFA	volatile fatty acids
VHS	vertebral heart score
WHS	wobbly hedgehog syndrome

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1 All creatures great and small

I am part of the generation that grew up watching reruns of the original UK 1978 James Herriot TV series *All Creatures Great and Small*. The more recent 2020 remake reminded me of one particular scene that is common to both versions.

The veterinary surgeon leaves his consulting room, enters the waiting room, and sees: a dog, a cat, a ferret, a duck, a rabbit, a shoe box (presumably with a small furry inside), and a budgie in a cage waiting to be seen. Did the veterinary surgeon panic? Did the veterinary surgeon refuse to see any of the patients? Not visibly and definitely not, are the respective answers!

Due to this I started to wonder when it became the 'norm' for small animal veterinary surgeons only to treat dogs and cats. *Why do some species appear to matter more than others, despite the human animal bond not being restricted to any particular species?*

In my experience, veterinary surgeons, students, and some registered veterinary nurses, tend to hit their panic button when presented with an unfamiliar species. Logic often goes out of the window when people panic.

History taking

How to take a history is not really species specific. For example, we ask dog owners: what do you feed your pet? What do the faeces and urine look like? Where is your pet kept? How much exercise do you provide and what forms? What preventative health care is practised? What is worrying you and what are the clinical signs? We then will ask further questions guided by the owners' answers. Yes, with exotics we need to go into much more detail regarding their enclosures, but realistically the vast majority of the history taking is *just the same as more familiar species*.

This is where *species-specific husbandry questionnaires are invaluable*, they can be given to the owners to fill out while they are waiting to be seen. The questionnaires I use are provided in the Appendix for you to utilise in your clinic. The up-to-date husbandry requirements are given per species in the relevant chapters for you to assess the quality of care each owner is providing. Ultimately for many of these species we are trying to recreate Mother Nature in a captive environment. If we know the habitat and diet these species evolved to thrive in, then that is what we aim to recreate in captivity.

All animals feel pain

All vertebrates have the *same anatomical and physiological pain pathways*. We should realise that all the small mammals, reptiles, and birds that we treat, feel pain just as we do. Just because they are good at hiding it (remember that many of them are prey species and hiding illness/pain is an evolutionary adaptation to avoid predation), and that we may not understand how they communicate or their body language does not mean we should not treat the pain.

In an ideal world we would all understand normal behaviour in each species to enable critical evaluation of pain assessment. Also, we need to be aware that the mere presence of an observer can elicit complete immobility in these species and so hide any pain-related behaviours. In the real world this is unrealistic for each of us to understand all the normal behaviours of every species, and my personal approach is if I would find it painful then so will they. The advantage of understanding normal behaviour in these species is that it allows you to evaluate the efficacy of your analgesia. *Acute pain is much easier to see in all species than chronic pain. Failure to alleviate pain is a welfare concern*.

How animals experience pain at an emotional level is still much debated. My personal opinion is how can we doubt that emotional level when we see, for example, how a dog with chronic osteoarthritic pain reacts with such enthusiasm for life after two weeks of analgesia?

Multimodal analgesia is recommended in all species. This is especially relevant in exotics where many of the drugs utilised are unlicensed and may have limited scientific data to back their use. Hawkins (2006, p. 189) wrote: 'Balanced analgesia should be considered in virtually every exotic patient, because the use of multimodal therapy may maximise analgesic efficacy and minimise the potential for individual drug toxicity in these patients'.

The vast majority of drugs used and mentioned in this book are not licensed for use in these species. The doses given are taken from the literature and from my own

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experience. In the UK, we can prescribe them under the cascade system and as such informed consent must be obtained with an off-licence consent form being signed by the client prior to use. Readers outwith (outside) the UK are advised to check the regulations where they are based.

Mostly, exotics are easy – they are just body systems in a different wrapper

Most of what I do on a daily basis (my caseload is 70–80% exotics) is easy; a fact my veterinary surgeon husband vehemently disagrees with. Many years of speaking for various organisations and teaching at Glasgow University School of Veterinary Medicine (GUVS) had me urging colleagues to 'go back to basics' and perform exactly the same clinical exam they would in more familiar species. Recently, it occurred to me that the reason I find it easy is that I do not actually think in terms of species, *I think in body systems*.

Yes, there are species-specific adaptations, but in reality, they are few in number. Each specific body system has the same function, regardless of species. Most body systems have a limited number of responses to disease. *So, if that body system has the same function, and responds to disease in the same or similar way, then surely it stands to reason that it is likely to require the same treatment?*

It is important to know that there are certain medications that cannot be used in certain species. However, when we consult our formularies they will often tell us this! When many of us hit the panic button at the first sight of the patient, we then do not even get to the treatment options stage. We become of no use to person nor beast.

Reinforcing experiences

Asian water dragon

My body systems-based approach was reinforced in 2021 by two experiences. The clinic I work in hosts the selective exotics rotation for GUVS final year students. During one rotation, two of my final year students brought me an Asian water dragon that had its right eye mostly closed. I asked my students what they wanted to do, and nervous giggles ensued. 'Stop hitting your panic button and tell me your plan.' The nervous laughter was followed by blank looks. 'Okay, let's change the species. You are now looking at a 6-year-old male black Labrador with his right eye mostly shut, what do you want to do?' Immediately both students said they wanted to perform an ophthalmic exam and use fluorescein. *All I changed was the species*. Once the fluorescein was applied, the large corneal ulcer was obvious (Figure 1.1). The students correctly identified that analgesia was required as well as topical treatment for the ulcer.

This was a valuable experience as it reinforced the concept of thinking in body systems for all of us. If my students had not panicked at the sight of a lizard, if they had thought in terms of an eye and that the eye has a limited number of responses to disease regardless of species, they would have achieved their diagnosis without my input. Without it, if they had already been qualified, this animal would not have been promptly diagnosed (despite the fact they already had all the skills necessary!), and there would have been an unnecessary delay in treatment, which compromises the welfare of our patient.



Figure 1.1 Corneal ulcer highlighted by the application of fluorescein in an Asian water dragon.

Alpaca

A much more dramatic example further reinforced my method of thinking. I was visiting a good friend's farm and they asked my opinion on one of their cria (baby alpaca). In the UK, alpacas are considered farm animals and so were under the care of a farm animal practice. The cria was 8.5 months old at this point and had a large mass on the medial aspect of the proximal left tibia. The mass was pocked with scabs and fistulas were visible. 'Looks like an abscess to me' (Figure 1.2).

Owner: Yes, it is and I have been squeezing it daily just as I was told.

Me: How long has it been there?

Owner: About eight months.

Me: I would be worried about osteomyelitis and I would also open up that abscess surgically and debride it.

Owner: Oh yes, she does have osteomyelitis, here are the radiographs. (Figure 1.3)

Me: What treatment has she been on?

Her owner produced the medical records. She had been given two injections of analgesia, two 3-day courses of antibiotic injections and had been sedated to remove a small fragment of necrotic bone via a small incision.

I advised I had never examined an alpaca before but in my opinion any abscess needs to be fully opened and may need surgically debriding, a course of analgesia should be given as it is a painful condition and, as osteomyelitis is a deep-seated infection, a long course of antibiotics will be needed, which may be up to 12 weeks. Ideally a culture and sensitivity of the lesion would also be performed.

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Figure 1.2 Swelling on the cria's leg.



Figure 1.3 Radiograph of the cria's leg.



Figure 1.4 The area once fully debrided.

Her owner was seriously considering euthanasia at this point. He asked me to treat her, which I arranged. Having never treated an alpaca before but knowing I am just treating body systems I looked up aetiology and suitable drug doses.

After performing a ring block using lidocaine, I incised the mass, the capsule was over an inch thick and you could hear me cutting through it with the blade. I resected the full capsule and debrided the wound back to more healthy-looking tissue (Figure 1.4). There were pus filled tracts down to the bone. I flushed the wound with sterile saline and dressed the wound with Granuflex hydrocolloid dressing, Elastoplast and pre-stretched Vet Wrap.

She received multiple dressing changes weekly for several weeks. Then as the wound healed the dressing changes were dropped to once weekly (Figures 1.5a–d). *Opening up the abscess allowed the body to push out two foreign bodies and five fragments of dead bone*, all of which were collected in the dressings. Analgesia was given for 6 weeks and antibiotics were injected daily for 12 weeks. *After 16 weeks she was fully healed* (Figure 1.6).

It was highly likely an adult alpaca had kicked her at 2 weeks old resulting in plant material being pushed through the skin and sequestrum of the bone due to the trauma and subsequent infection.

I had never treated an alpaca before but instead of hitting my panic button, I treated it the same way I would treat this condition in any species. We do not squeeze cat bite abscesses – not if we want to live! *Just because an animal does not have sharp carnivore dentition or sharp claws does not mean we should advise different care for the same condition.*

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Figure 1.5 Various stages of healing.



Figure 1.6 Fully healed.

Change of mindset

Life is stressful enough. If we change our mindset from species centric to body systems, then treatment options become more obvious and less intimidating. Regardless of species, I perform the same clinical exam (in reality all a clinical exam really is, is an assessment of body systems!), in the same order, each time. When we have a consistent approach, we are much more likely to find abnormalities. If we are not sure how to treat those abnormalities, that is when we can use a book or ask a colleague for help. Owners really do not expect us to be walking encyclopaedias. They expect us to care.

My aim is to write the book I wish was available when I was a new graduate. I hope you find it useful.

Reference

Hawkins, M. G. (2006). The use of analgesics in birds, reptiles, and small exotic mammals. *Journal of Exotic Pet Medicine*, *15*(3), 177–192. https://doi.org/10.1053/j.jepm.2006.06.004

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2 Rabbits

Rabbits are the third most popular pet in the UK and are rising in popularity globally. Their average life span is 9–11 years of age, and the oldest rabbit I have treated was 18.

Owners bond very tightly with their rabbits; they are frequently kept indoors, and training rabbits has become much more popular. The domestic rabbit (*Oryctolagus cuniculus domesticus*) originated from its wild counterpart the European rabbit (*O. cuniculus*) which evolved to live in the Iberian Peninsula. They evolved to eat grass, leaves, flowers, some roots, and bark, to live in burrows termed warrens, and, as a prey species, to hide disease and pain to prevent predation.

Educating owners on husbandry and how to improve the welfare of their pet (e.g. environmental enrichment) should be part of a standard veterinary visit. New pet owners may be more likely to seek advice but every day is a school day and *we need to be aware that owners may confuse love and dedication to their pet with appropriate welfare*. This is where husbandry registration forms are invaluable in providing us with a non-confrontational overview of how the pet is being kept.

Companionship

As a prey species, they evolved being a food source for many predators. If you have evolved to be 'food', you will feel safer if there are two or more sets of eyes looking out for predators than if you are on your own. If you have evolved to live in a single species colony then you have an innate requirement for conspecific companionship. *Put simply: rabbits need rabbit companions.*

The current recommendation is keeping a neutered male and a neutered female as that combination is most likely to bond. I have seen every combination work and every combination fail and taking the personality of the rabbit into account is also important.

Rabbit rescues will often offer advice on bonding rabbits and some reputable ones even run 'bonding boot-camps'. Bonding rabbits is outwith the scope of this book but is covered in *Behavioural Problems in Rabbits: A Clinical Approach* by Guen Bradbury (ISBN: 9781789180121), which I advise reading.

Guinea pigs do not make suitable companions for rabbits. We all know people who have 'gotten away with it' and claim that their rabbits and guinea pigs live together in harmony and love each other. In reality, guinea pigs and rabbits evolved to live in different niches, have different dietary requirements, different behavioural needs, and different communication methods. Rabbits also harbour *Bordetella bronchiseptica* as a normal nasal commensal, which can cause fatal bronchopneumonia in guinea pigs. The powerful kick of rabbits can result in fractured ribs if aimed at the guinea pig and bullying by either species is possible.

Enclosure

Regardless of whether rabbits are kept indoors or outdoors, the minimum enclosure footprint (for two average sized rabbits) size is: 3 m × 2 m × 1 m high, and they should have permanent access. Enclosures need to be designed to provide an outlet for normal behaviour, 3 m is required for rabbits to be able to run. An enclosed sleeping space (2 m × 0.6 m × 0.6 m) can be provided either inside the enclosure or linked to the outside of the enclosure. Connecting tunnels, raised platforms, and two storey enclosures do not count as part of the footprint.

Environmental enrichment

Rabbits, especially does, have an absolute requirement to dig. This is completely normal, natural behaviour and cannot be 'trained out of them'. Providing acceptable alternatives such as 'dig boxes' will satisfy their requirement to dig and satisfy their owners requirement to redirect the behaviour. Owners will often take their rabbits 'bad behaviour' extremely personally, not realising that *many of the behaviours that they find unacceptable, are in fact, completely normal and natural for a rabbit to perform*.

I regularly recommend that owners also read Bradbury's *Behavioural Problems in Rabbits: A Clinical Approach*. Once owners understand their pet better, they can provide improved environmental enrichment for their pet and the improved understanding strengthens the human animal bond.

Homemade foraging toys (cardboard centres from toilet rolls are favourites), cardboard boxes (with multiple entrance / exit holes – prey species need options), and platforms / sturdy boxes (allowing height to scout for predators)

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are all easy forms of environmental enrichment. Tunnels can also have forage placed in them to allow feeding while feeling safe.

Diet

A diet of 85% forage (hay and grass), 10% dark leafy greens, 4% good quality concentrates (nuggets or pellets – not muesli as this has been shown to contribute to dental disease), and 1% healthy treats should be provided.

Many owners are still unaware that hay should be fed ad lib and that the *minimum consumption should be the rabbit's own body size in hay daily*. Another consideration is type of hay and which cut. It is surprising how many owners really do not know about different types of hay.

While one rabbit may prefer timothy hay, another may prefer meadow hay and yet another may prefer orchard. The quality/taste of hay will vary throughout the year depending on when it was harvested. This is due to the fact plant composition varies throughout the year. Therefore, it is understandable that a rabbit's preference may also vary throughout the year. Alfalfa hay should only be fed to young, growing animals, pregnant or lactating does.

> Educating owners on the available types of hay and potential reputable sources for example, Hop to Forage (www.hoptoforage.co.uk), can help with – 'Oh, my rabbit will not eat hay.'

Wild rabbits spend approximately 80% of their waking time foraging – compare this to captive rabbits. Rabbits will regularly eat while passing faeces. Providing hay beside of, and/or in litter trays encourages further grazing. Using bedding hay and scattering the daily allowance of concentrates through the hay provides environmental enrichment. As humans we mainly eat our food from plates or bowls and we like to see our pets do the same. Unfortunately, the bowls made for rabbit food are massively big when you compare them to the egg cup sized portion advised per 2 kg rabbit. This leads to inadvertent over feeding in many cases as owners will often completely fill the rabbit bowl with concentrates (see Figure 2.1).

Fresh grass should be offered daily. Many owners claim to feed grass daily but on further questioning they may be referring to dried grass. If rabbits belong to owners without a garden, encourage the owners to grow turf in trays, which can be rotated allowing grazing while the other trays regrow.

Rabbits evolved eating a diet that varied throughout the year depending on seasonality. We should try to replicate this in captivity to encourage more natural behaviour. For a list of safe greens they can buy or forage, owners can be directed to websites such as: https://rabbitwelfare.co.uk/rabbit-diet/recommended-vegetables-herbs/.



Figure 2.1 A typical rabbit bowl with the weighed total day's recommended allotment of pellets. It is easy to see how owners can inadvertently over feed pellets.

Water

Wild rabbits frequently ingest grass and plants that are soaked in dew or rain, thereby consuming more water *while eating*. In captivity the main food source is hay, which is a great source of fibre and nutrition but is extremely low in water content. Rabbits that eat a lot of fresh grass or greens will drink less than those who do not. However, neither group is likely to consume as much water as the wild rabbit. The maintenance water requirement is 100–150 ml/kg/day. Water can be provided via a bowl or a bottle. Both methods have pros and cons (see Table 2.1). Many rabbits will find lapping water from a bowl much more natural and in an ideal situation both methods should be utilised.

Rabbits have a unique calcium metabolism. They absorb all calcium from ingested food and excess levels are excreted via the kidneys in the urine. Inadequate water consumption can lead to calcium sand precipitating in the bladder leading to 'sludgy' urine, cystic calculi, renal calculi, and potentially life-threatening renal disease. *I believe that 'sludgy' urine etc. is not due to dietary intake of calcium but rather it is down to the fact that wild rabbits have a much*

Table 2.1 Water Bottle versus water bowl.

Water bottle		Water bowl	
Pro	Con	Pro	Con
Keeps water clean	Nozzle can block	*Rabbits prefer and drink more*	Rabbit may fill with substrate
Prevents spills	Rabbits drink less		May spill

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higher water intake. Many captive rabbits have skeletal pain due to osteoarthritis. They are therefore reluctant to pass urine as frequently and may not be as mobile, leading to an increased risk of urine becoming concentrated and supersaturated, in turn leading to further precipitation.

Encouraging natural diuresis by providing cooled dandelion or nettle teas as drinking water will promote further fluid intake.

Ultraviolet (UV) light

Rabbits are mainly crepuscular, but they can regularly be seen basking in the sun. Indoor only rabbits, or rabbits who rarely are given outdoor access, benefit from a source of UVA/B. The lens of the rabbit eye transmits UVA and the cone cells in the retina are sensitive to these wavelengths. *These facts are highly suggestive that rabbits see into the UV spectrum*.

Many of us associate the sun with 'feeling good'. This is due to the release of beta endorphins triggered by exposure to UV light. Improved skin immune function and conversion of vitamin D to the active form D_3 are also benefits provided by provision of UVB. Dermal conversion of vitamin D to D_3 is self-regulating and it is therefore impossible to overdose! The University of Illinois showed no adverse effects in rabbits exposed to appropriate levels of UVB over a 6-month period.

We know rabbits evolved to live on the Iberian Peninsula and that these areas are exposed to high levels of UV light. Utilising Baines *et al.* (2016) UV Tool we can see that an animal that evolved to live in the Iberian Peninsula would fall under Ferguson zone 3. Providing an artificial sunbeam (UVA/B and heat) will give indoor rabbits physical benefits, mental health benefits, and allow more expression of normal species-specific behaviour. (Figure 2.2).



Figure 2.2 This rabbit is basking under a heat lamp situated beside a UVB lamp. Warm skin absorbs UVB better than cold skin. The owner has made a shelf to which they have attached the lamps. Photo: Dr Krissy Green.

Sexing

Rabbits are relatively easy to sex. Both sexes have something that 'pops' out when the genital region is manipulated. *It is the shape that is important*.

Female

Female rabbits have nipples (which unless they are hormonally active will be covered with dense fur). The shape of the exposed vulva is a slit which extends right to the perineum. (Figure 2.3).



Figure 2.3 Female rabbit. Note the slit shape of the vulva. The brown material on either side is secretion from scent glands and is normal.

Male

The presence of nipples in male rabbits is variable. The shape of the penis varies slightly with the age of the rabbit and the age at which the rabbit was castrated (if it has indeed been castrated). In entire mature males the penis curves down ventrally at the tip and the testicles may be obvious (see Figure 2.4a). In adult males the tip of the penis is circular and has been described as a doughnut shape (see Figure 2.4b). In immature males it can be very

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Figure 2.4a Entire mature male rabbit. The tip of the penis curves ventrally and the testis are obviously in the scrotum in this rabbit.



Figure 2.4b An adult male rabbit who was castrated as a young rabbit. Note the circular tip to the penis.

easy to over evert the penis, resulting in a slit-like shape. The important difference is that this 'slit' is still contained within the doughnut shape – it does not extend to the perineum (see Figure 2.4c). Scrotal sacs are delicate and as the inguinal canals are open, *we cannot rely on the presence of testis to sex as they may have been retracted into the abdomen*.

Preventative health care

Vaccination

Availability and requirement for vaccination will vary depending on the country. The relevant drug company representatives will be able to advise on requirements if the reader is outwith the UK.

Currently I am using the trivalent vaccine Nobivac Myxo-RHD PLUS (MSD), which protects the rabbits against: myxomatosis, rabbit haemorrhagic disease type 1,



Figure 2.4c An immature male. It can be very easy to over evert the penis, resulting in a slit-like shape. Note that the 'slit' is still contained within the doughnut shape – it does not extend to the perineum. The black material is secretion in the scent gland and is normal.

and rabbit haemorrhagic disease type 2. This vaccine is given subcutaneously (SC) from 5 weeks of age and can be given to pregnant does. It is recommended that to provide a full year of immunity, we vaccinate from seven weeks of age or older. If the rabbit has received a different vaccination in the previous 12 months contact MSD for advice on transitioning to the trivalent vaccine as in some cases an additional RHD2 vaccine will need to be administered within 2 weeks.

Classical myxomatosis

Caused by a myxoma virus that is spread by biting insects and direct contact. Early stages of the disease can look like conjunctivitis due to swelling of the eyelids (Figure 2.5). However, it progresses into oedema of the nares, lips, ear base, and genital region. Pyrexia develops and the eyelids become so oedematous they shut. Death often occurs within 14 days, thought to be due to overwhelming bacterial infection. *There is no cure for classical myxomatosis, only prevention by vaccination*.

Atypical myxomatosis

Can occur in rabbits that have been previously vaccinated but whose immunity has waned due to lapsing of booster vaccinations. It presents as a well circumcised hard lesion, full skin thickness, often on the bridge of the nose (Figure 2.6). Reassure the owner that it is non-painful and self-resolving. It is advisable to warn the owner that once the scab falls off it may look alarming.

Rabbit/viral haemorrhagic disease

Caused by a calicivirus, which is spread by fomites including dust on the wind. There are currently two recognised

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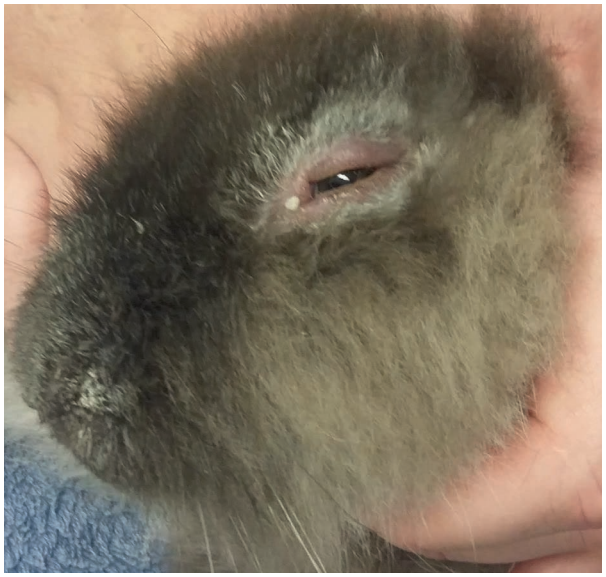


Figure 2.5 A case of myxomatosis with oedema of the eyelids. This may be misdiagnosed as conjunctivitis in the early stages.



Figure 2.6 Atypical myxomatosis.

types. Mortality rates vary depending on type but *any rabbit that has recovered from either type should be considered a reservoir of infection for 2 months*.

RABBIT HAEMORRHAGIC DISEASE TYPE 1

Highly virulent, it causes up to 90% mortality in rabbits over eight 8 weeks of age and does not affect other species such as hares and cottontails. Death may be peracute without any clinical signs. Death is usually due to disseminated intravascular coagulation and bleeding may be seen from the external orifices.

RABBIT HAEMORRHAGIC DISEASE TYPE 2

Less virulent, mortality rates vary ranging 5–70% (20% is average) in rabbits and *will affect other species such as hares and cotton tails*. Unlike type 1, *can affect all ages* of rabbis including kits less than four weeks of age.

Owing to lower mortality rates, chronic cases are more common and can end up jaundiced due to liver involvement. Can recover with supportive care.

Worming

Despite what the data sheet of a well-known rabbit wormer recommends – *rabbits do not require routine worming*. Worms will rarely cause pathology in rabbits and if concerned, we should perform a faecal flotation or send a sample to the lab, allowing us to diagnose and target our treatment.

Encephalitozoon cuniculi (E. cuniculi) prevention

The current information we have indicates that 9 days of fenbendazole administered at 20 mg/kg orally q24 hrs can give some protection against *E. cuniculi*. Therefore, it is recommended that new and existing rabbits should be treated when planning introductions. Prolonged treatment with high doses of fenbendazole has been associated with bone marrow aplasia in multiple species, including rabbits.

Ectoparasitic control

Some owners will ask for year-round ectoparasitic control as part of their myxomatosis management. Owners should be aware that myxomatosis can also be spread by fomites or direct contact between infected animals. Some owners cannot stand the thought of their pet getting fleas especially if they are house rabbits.

Imidacloprid is licensed in the UK to treat fleas.

Ivermectin can be used topically or injected SC. Several options exist for topical preparation, which will vary depending on the country. A dose of 200–400 ug/kg is used every 2 weeks for three treatments and will be effective against fleas and mites.

Selamectin has been shown to be effective at eradicating infections of mites such as *Cheyletiella parasitovorax* and *Psoroptes* cuniculi with a single dose application. Recommended doses vary between 6 mg/kg and 20 mg/kg. *Selamectin may need to be applied weekly to provide adequate protection against fleas according to one study* (Carpenter *et al.*, 2012).

Neutering

We advise neutering rabbits for the same reason we advise neutering any species: *population control, hormonal control for behaviour, and disease prevention*. I recommend neutering most rabbits at 3.5–4 months of age; the giant breeds I will leave until 5.5–6 months of age. The primary fat storage site in does is the mesometrium and mesovarium. Spaying at a young age, before much fat has been laid

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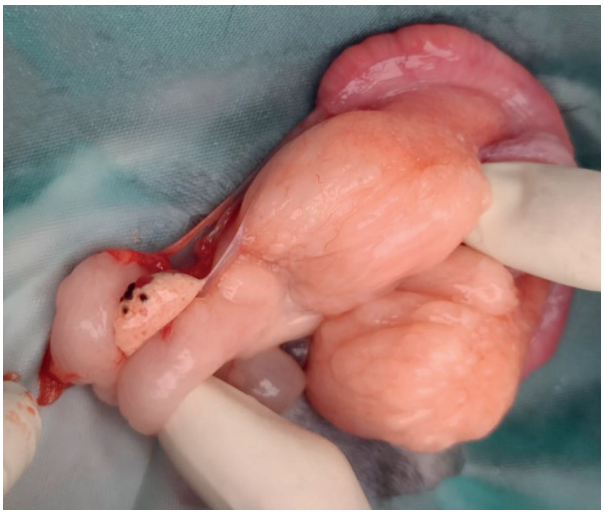


Figure 2.7a The uterus and ovary in a mature doe around 6 months old. Note the high level of fat in the mesovarium and mesometrium.

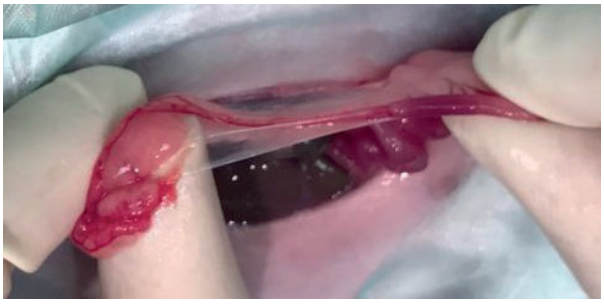


Figure 2.7b The uterus and ovary in a young doe around 4 months old. There has been very little fat laid down.

down makes the procedure easier and therefore safer to perform (Figures 2.7a & b).

Population control

Gestation is 28–32 days long. Rabbits should be kept in pairs (minimum) and the recommended pairing is a neutered male and neutered female for the best chance of successful bonding, *although sometimes the personality of the rabbit is the most important factor when picking a companion*.

Hormonal control for behaviour

In my experience female rabbits show much more territorial aggression than male rabbits. This makes complete sense to me (despite being contradictory to some texts) from an evolutionary point of view.

In the wild the male's role is to mate and chase off other male rabbits. The female's role is to dig the warren, protect the nest, have the kits, and protect the kits. The most dominant doe has the 'safest' nest, that is the deepest nest because predators will eat other does' young before getting to hers. Therefore, she also has to defend the nest from other does – no wonder she displays more territorial aggression.

Neutering at 3.5–4 months of age will in most cases prevent territorial aggression from developing, however if the rabbit is already showing signs of territorial aggression owners can be counselled that we would expect the behaviour to regress with the hormonal regression during the next 6 or so weeks.

Spray marking territory with urine is a common behaviour performed by both sexes although males will perform this more frequently. A rabbit who is tightly bonded to its owner may very well run around the owners' legs while spraying urine upwards – the rabbit is only marking the human owner as important, but the owner is not usually impressed! If this behaviour is fully established then just as with other species, we cannot guarantee this will fully disappear with neutering although the frequency should decrease. If there has been a sudden increase in urine spraying then *we should investigate this animal for stressors in the environment*, why is this rabbit feeling the need to suddenly establish territory boundaries?

Both sexes will perform mounting behaviour, which is not always sexually motivated. Sexually motivated mounting is most commonly performed by the male and can be quite aggressive – my friend's male rabbit burst a football while mounting it. This can become a problem if the object of desire is not interested, the household cat, the pet chicken, or the owner's leg!

After neutering any sexually motivated mounting we would expect to regress with the hormonal regression during the next 6–8 weeks.

Fighting between conspecifics can have hormonal influence. Even with surgical neutering owners may notice their rabbit's mount, 'squabble', or seem more irritable during spring due to the 'spring fever' phenomenon. Bradbury describes this in detail in her book, which is mentioned earlier in this chapter.

Neutering for disease prevention

Does

The main condition I neuter to prevent is *uterine adenocarcinoma, which will occur in up to 80% of unneutered does of 4 years of age or above*. I have diagnosed uterine adenocarcinoma in a 1-year-old doe. The vast majority of these will have metastasised to the lungs by the time of diagnosis. If you are suspicious of uterine adenocarcinoma already being present, radiographing the chest pre-operatively is recommended although the owners should be counselled that no obvious metastasis does not guarantee no spread, as the metastasis may be too small to see on radiograph as of yet.

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Ovariohysterectomy also prevents ovarian neoplasia, mucometra, pyometra, false pregnancy, and mastitis. All of these conditions are reported in the rabbit. Depending on age at neutering it can also help reduce the risk of mammary neoplasia.

OVARIOHYSTERECTOMY (OVH)

My belief with spaying any species is that you make the wound as large as you need to. Wounds heal 'side to side' so a 4 inch (10 cm) wound will heal as fast as a 2 inch (5 cm) one. Most of the vets I see who run into problems perioperatively are trying to operate through a tiny wound. First, learn to spay correctly. Second, learn to perform the surgery faster. Last, learn to perform the surgery through a smaller wound. It is completely unrealistic to expect a vet who is 2 years qualified to spay through a wound the same size as a 20-year qualified vet. These expectations (sometimes it is the vet putting pressure on themselves) lead to high stress levels, demoralisation, and increased morbidity.

The primary fat storage site in does is the mesometrium and mesovarium. Mature does (6 months and older) have significant amounts of fat at these sites even at a normal body condition score. This can make surgery much more difficult as rabbit tissue is very friable compared to more familiar species, when they are overweight, it can result in a stressful procedure with higher risks.

Personally, *I have a policy of not neutering any animal that is badly overweight*. Neutering is an elective procedure, once I have explained the increased risks associated with operating on an obese patient (I tend to describe the fat melting and increased risk of bleeding due to everything being slippery – it is like operating in a chip pan) the vast majority of owners 'get it'. Neutering obese animals is in my opinion not good animal welfare and I advise my owners that I will always put their animal first. If owners cannot correct their husbandry pre-op they will not do it post-op. I do not shame owners, education and empathy is key to getting them on board. Since instituting this policy in the practice 15 years ago, we have only had one complaint about the policy, and our vets do not suffer the stress of spaying fat bitches / cats / rabbits.

I recommend neutering rabbits at 14–16 weeks of age. Giant breeds take longer to mature, and I will generally leave the giant breeds until 6 months of age before recommending neuter.

Apart from anaesthesia considerations, and the friability of rabbit tissue, there are five main differences between spaying rabbits and spaying more familiar species.

- Rabbits have a large caecum in close proximity to the abdominal wall, tenting the linea alba upwards is important on entry to the abdomen to prevent inadvertent piercing of the caecum.
- Rabbits have no uterine body. There are two horns, two cervixes, and the structure that is often mistaken for a uterine body is in fact the flaccid internal vagina. The internal vagina frequently fills with urine when the doe urinates.
- The ovaries are approximately an inch away from the end of the uterine horns. It is important to appreciate this and visualise the ovaries as there have been several cases anecdotally where the ovaries have been left behind.
- Catgut should not be used. Rabbits struggle to break catgut down leading to increased risks of adhesions and granuloma formation, which can cause chronic pain.
- Rabbits form adhesions readily and are used in human adhesion studies because of this. In humans, abdominal adhesions are extremely painful. Gentle tissue handling will help reduce the risk. *If swabs are required in the abdomen, then pre-soaking them with saline will reduce the risk of drying out viscera*. If any of the viscera or fat becomes dry it will increase the risk of adhesions. Flushing the abdomen with warmed sterile saline post-operatively can also help reduce the risk and will hydrate your patient.

SURGICAL PROCEDURE FOR SPAY

The umbilicus can be difficult to appreciate in many rabbits. An easier landmark is the most caudal pair of nipples (Figure 2.8). Holding your scalpel in a pen like grip, make a skin incision midline approximately 2 inches (5 cm) long, between the nipples.

Blunt dissect through the subcutaneous layer to expose the muscle. The thickness of the subcutaneous layer varies between individuals and in some it is almost absent. The linea alba is sometimes not as obvious as we are used to in dogs. There are often three white lines running roughly parallel – *the middle one is the linea alba*.

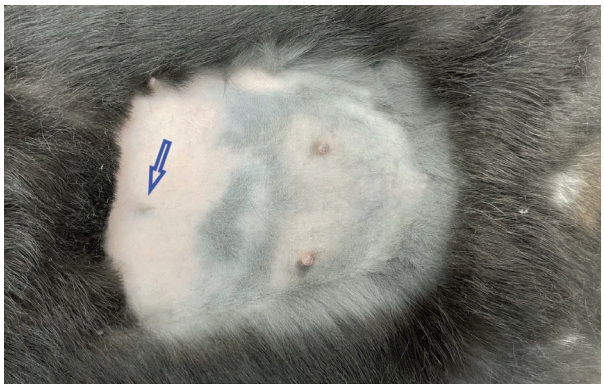


Figure 2.8 The landmarks for spay incision, the umbilicus and caudal pair of nipples. The umbilicus is highlighted by the blue arrow.

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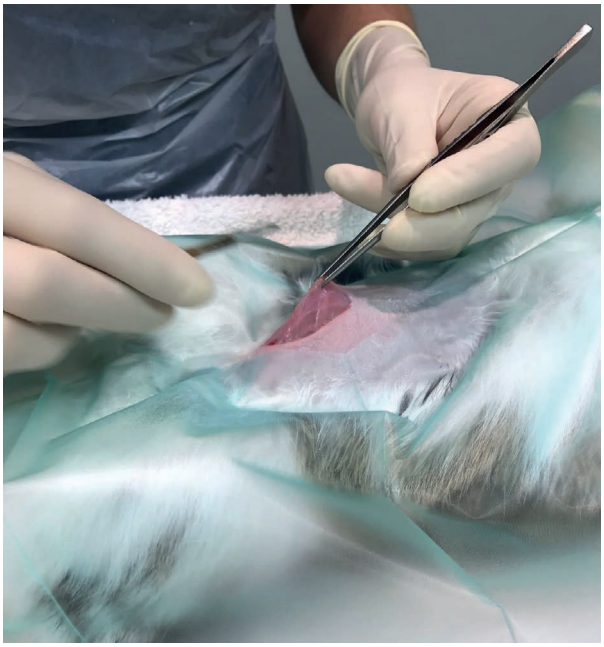


Figure 2.9 It is important to tent the abdominal muscle prior to the reverse stab incision to prevent inadvertently incising into the caecum.

Grasp the linea alba in your rat tooth forceps (ensuring you have only grasped the muscle and not underlying viscera), tent the muscles up as high as you are comfortable with (the muscle layer is not overly friable) and perform a reverse stab incision. We tent the muscle to elevate away from the large caecum (Figure 2.9). Enlarge the opening in the muscles using scissors, ensuring the tips are pointing up (so we know what we are cutting).

The uterus sits between the bladder and the descending colon (it's just a uterus in a different wrapper). Locate the end of the uterine horn, the infundibulum will run cranially from the end of the horn and then circle back around the ovary. If the ovary does not exteriorise easily, massage the fat distal to the ovary between your fingers which will often loosen the tissue. The ovarian artery runs perpendicular to the ovary (Figure 2.10). Visualisation of the artery is not always possible due to fat storage. An avascular window is present cranial and caudal to the ovary (Figure 2.11).

Holding the ovary in your non-dominant hand, break down the avascular windows to allow placement of artery forceps across the ovarian artery (or where you know it will be) (Figure 2.12). Do not pre-crush the ligature site. Using 3 metric (2/0) Vicryl or similar material place two ligatures.

Place another artery forcep directly above the one already placed, so that the two are touching. Keeping the one closest to the ligatures still and steady, rotate and twist the other forcep away (Figure 2.13). This causes the tissues to stretch and tear, any remaining tissue can be cut. Check for any bleeding before letting go of your ovarian stump. I leave approximately 1 cm tails on my ligatures, this allows me to find the site quickly if I am worried about a bleeding stump.

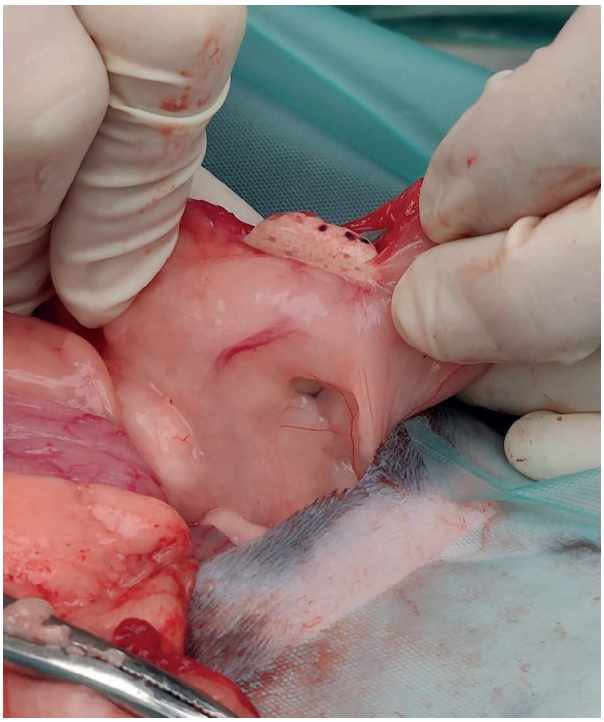


Figure 2.10 The ovarian artery can be seen running roughly perpendicularly from the ovary. The right side of the photo is cranial.

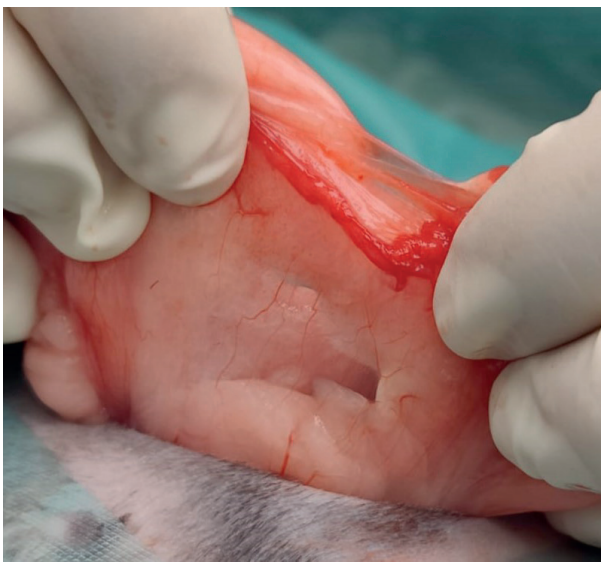


Figure 2.11 The ovary is being held in the hand on the right. There is an avascular window present in the centre of the photo, which is caudal to the ovary. The red, tortuous structure is the infundibulum.

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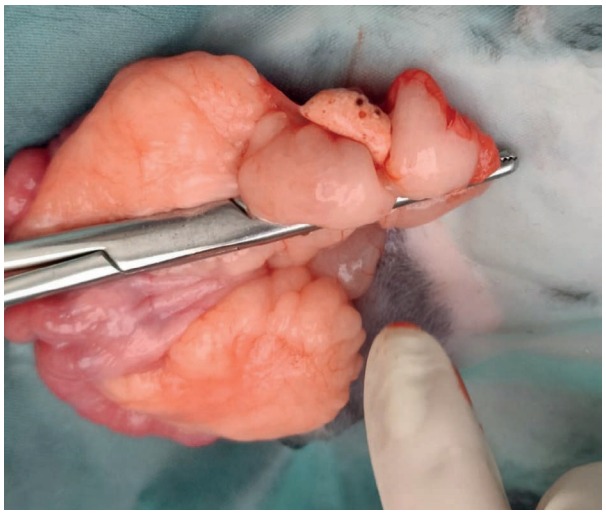


Figure 2.12 Place artery forceps across the ovarian artery (or where you know it will be).

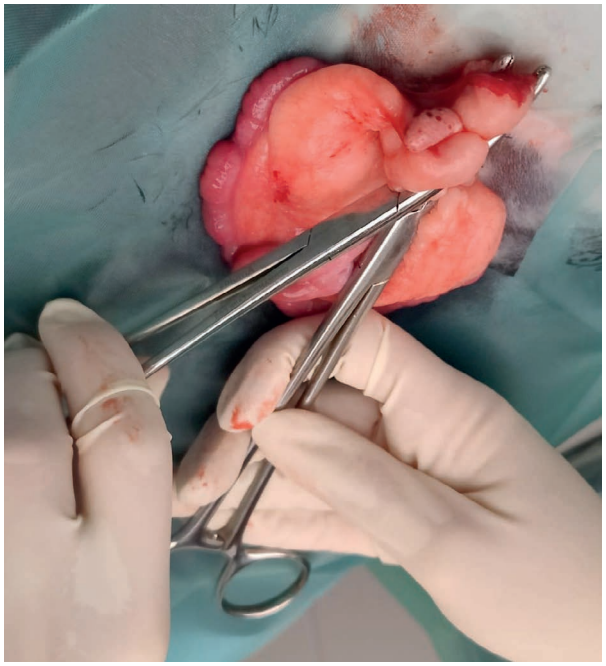


Figure 2.13 Place another artery forcep directly above the one already placed, so that the two are touching. Keeping the most distal set still, rotate the top set away to break the tissue.

See Figure 2.14 for uterine anatomy. The uterine artery runs parallel to the internal vagina and past the cervixes. Approximately one-third of the way along the uterine horn it forms multiple branches. Ligating just before it branches is much easier. *Do not place the ligatures near the internal vagina as there is a risk of incorporating the ureter.* Massage the mesometrium to find the avascular window on either side of the uterine artery. Place your artery forcep through these windows and across the artery. Do not

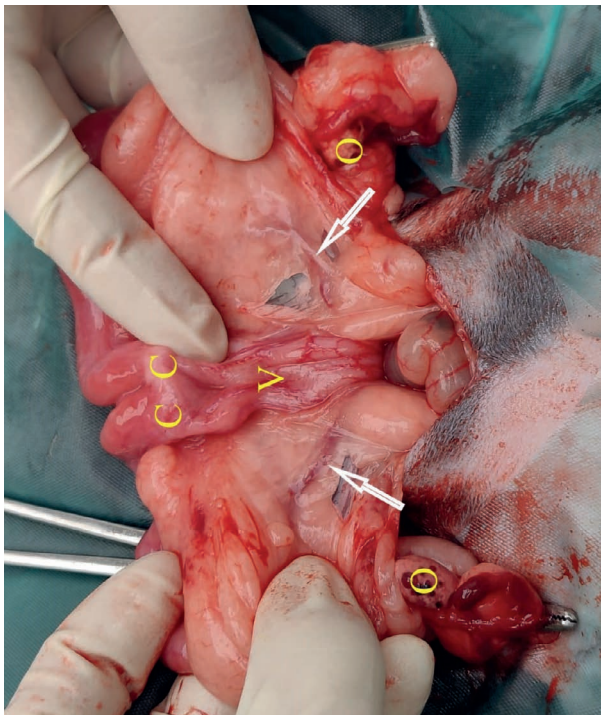


Figure 2.14 Uterine anatomy: the two cervixes are marked by a yellow C, the internal flaccid vagina is marked with a yellow V, both ovaries are marked with a yellow O, and the uterine blood vessels are highlighted by the white arrows.

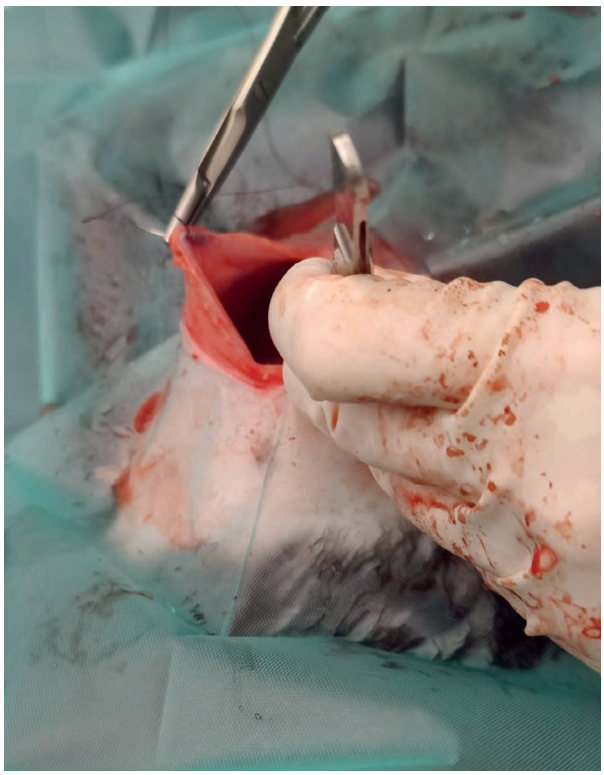


Figure 2.15 Lift the muscle while placing the cruciate sutures to ensure the caecum is not punctured.

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pre-crush the ligature site. Using 3 metric (2/0) Vicryl or similar material place two ligatures. Place another artery forcep directly above the one already placed, so that the two are touching. Keeping the one closest to the ligatures still and steady, rotate and twist the other forcep away. This causes the tissues to stretch and tear, any remaining tissue can be cut. Check for any bleeding before letting go.

How to remove the uterine horns is much debated. Ligating above (uterine side), through and below (internal vaginal side) the cervixes are all recognised techniques with their own pros and cons. *My preference is through or just below the cervix.* Ligating above the cervixes will inevitably leave some uterine tissue behind. In very young does this is unlikely to cause a problem as the ovaries have been removed before the uterus has been exposed to higher levels of sex hormones. In older does there is a risk of uterine adenocarcinomas forming if uterine tissue is left behind. As the internal flaccid vagina will sometimes flood with urine during urination, ligating below the cervixes carries a theoretical risk of uroabdomen if the stump is not oversewn.

Place one artery forcep across both cervixes, using 2 metric (3/0) Polyglactin 910 with a swaged-on needle (Vicryl, Ethicon), place a transfixing ligature between the cervixes. I then place a circumferential ligature distal to the transfixing.

Place another artery forcep directly above the one already placed, so that the two are touching. Keeping the one closest to the ligatures still and steady, rotate and twist the other forcep away. This causes the tissues to stretch and tear, any remaining tissue can be cut. Check for any bleeding before letting go.

Check all stumps for any signs of bleeding. Any bleeding stumps should be religated. Collaclot is an avian collagen sponge (available from IMS wholesalers) which can stop a life-threatening bleed. It is extremely effective and much less expensive than other collagen sponges on the market. They have a long shelf-life and can be used in any species. I then flush the abdomen with 100 ml warmed sterile saline which can help reduce adhesions and rehydrate the rabbit.

Repair the muscle layer with cruciate sutures 2 metric (3/0) Polyglactin 910 with a swaged-on needle (Vicryl, Ethicon), lifting the muscle to see placement of the needle at all times (I have seen students stitch the muscle onto the caecum when operating on cadavers). Rabbits can move unpredictably in multiple directions (think binkie!), *cruciate sutures counteract tension in every direction, they are faster to place and have few knots*.

Further analgesia can be provided by performing local anaesthesia splash blocks on closure. A mixture of l idocaine 2 mg/kg (maximum 4 mg/kg) and bupivacaine 1 mg/kg (maximum 2 mg/kg) can be used. Doses higher than the maximum recommended may lead to toxicity.

If there is a subcutaneous layer, this can be closed (1.5 metric (4/0) Polyglactin 910 with a swaged-on needle (Vicryl, Ethicon)) using a simple continuous pattern then an intradermal pattern is used for skin closure. *Buster collars should not be used on rabbits – they create stress (which leads to anorexia) and prevents eating of caecotrophs*. The majority of rabbits will not interfere with their wounds if they have adequate analgesia on board, which should be continued for a minimum of 10 days. Restrict exercise post-operatively for a week.

BUCKS

Compared to uterine adenocarcinoma in the doe, *testicular neoplasia is slow to metastasise and surgery at diagnosis is often curative*. A firm mass will be palpable within the testis with possible shrinking of the contralateral testis.

Testicular masses may also be cysts or abscessation. Scrotal swelling could indicate herniation of abdominal organs. Ultrasonography and careful palpation of the area can help differentiate.

CASTRATION

Castrating rabbits is essentially the same procedure we do regularly in other species – we remove the testicles and want a live patient at the end of it. Apart from the anaesthetic considerations and avoiding the use of catgut, the only other difference is that rabbits have open inguinal canals, therefore *if we do not close the tunic our patient has a real risk of abdominal organs herniating into the vaginal tunic/scrotum with potentially devastating consequences*. The open inguinal canal can also make castration challenging if we do not stabilise the testicle as the slightest pressure exerted on the testicle will push it back into the abdomen. If this happens, gentle pressure just cranial to the pelvis will usually result in the testis slipping back into the scrotum.

The two techniques I teach are 'open/closed' or 'closed' castrations. When I first started castrating rabbits the open/closed technique gave me the confidence that I had indeed taken all of the testicle. Now I am more confident, the closed technique is faster. *I hold my scalpel in a pen-like grip as this gives me much better control of direction and depth of my incision.*

Basic technique

Hold the base of the testicle in your non-dominant hand, clamped between your middle finger and ring finger (the testicle should now be on the palm side of your hand) (Figure 2.16). Stabilise the testicle between your thumb and middle finger. Use your index/pointer finger to stretch the scrotal skin. Do not let go of the base of the testicle (Figure 2.17). The scrotal skin is fragile in rabbits, so not much pressure is required to incise through it. Excess pressure will result in going through the skin and

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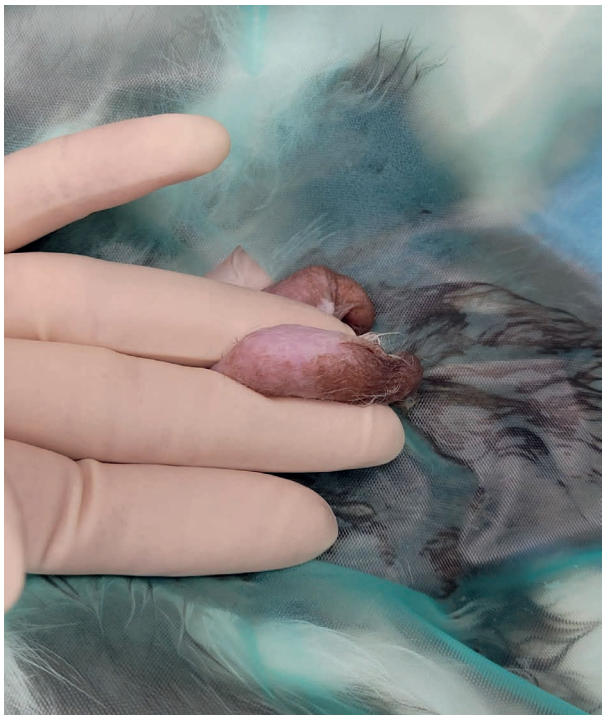


Figure 2.16 Hold the base of the testicle in your non-dominant hand, clamped between your middle finger and ring finger with the testicle on the palm side of your hand. This prevents the testicle slipping back into the abdomen.

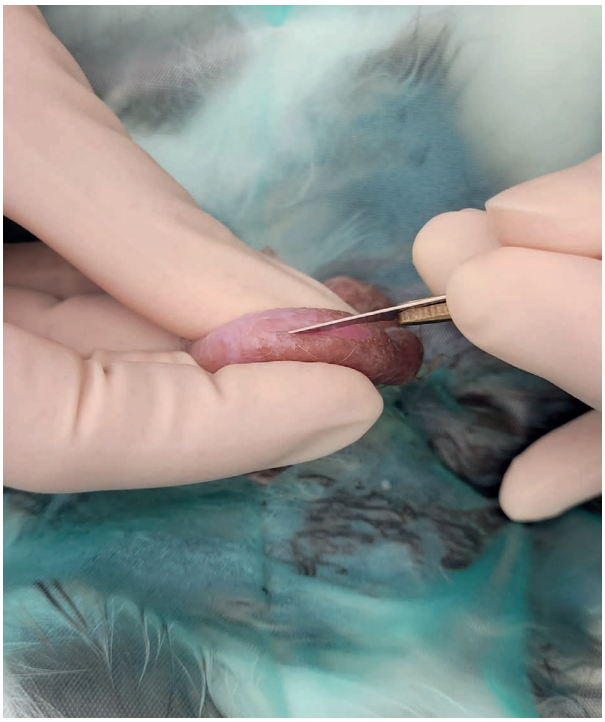


Figure 2.17 With testicle base still clamped between your fingers, use thumb and forefinger to stretch skin for incision.

straight through the vaginal tunic and potentially into the testicle.

Holding the scalpel in your dominant hand, feather light stroke in the centre of the scrotal sac, extending roughly half the length of the testis. Using blunt/blunt small scissors, push between the scrotal skin and vaginal tunic then open the scissors. This utilises blunt dissection to break down the attachments between the tunic and the scrotum (Figure 2.18). Repeat in all four quadrants. Using blunt/blunt scissors ensures we do not accidentally pierce the testicle.

Use your index finger and thumb of your non-dominant hand to gently squeeze the testicle caudally while your index finger and thumb of your dominant squeeze the caudal pole (epididymis) of the testicle cranially. This results in the testicle (which is still within the vaginal tunic) popping up through your incision (Figure 2.19). Once the epididymis is visualised through the tunic, we place an artery forcep across the epididymis (Figure 2.20). Now the testicle is stabilised by the artery forcep, we can let go with our non-dominant hand. Hold the artery forcep in your non-dominant hand (do not pull upward) and use a swab to help strip the rest of the scrotum down off the tunic (Figure 2.21). The tissue can be quite tough in mature bucks.

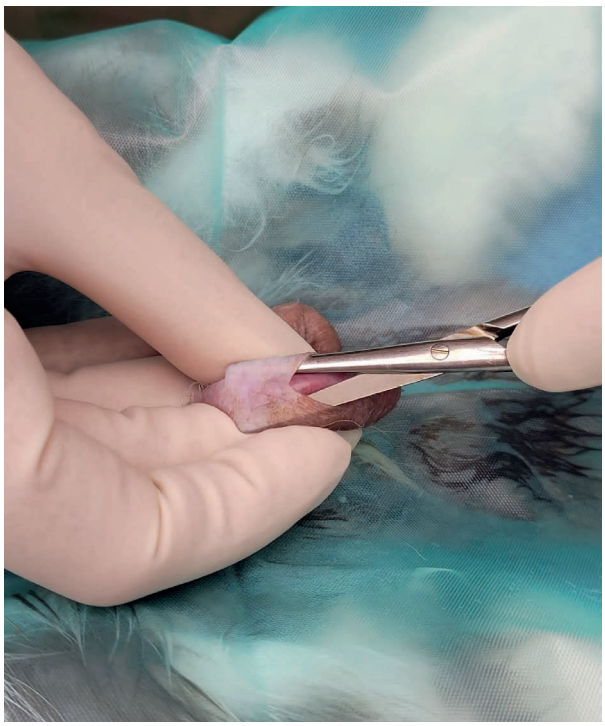


Figure 2.18 Using blunt/blunt small scissors, push between the scrotal skin and vaginal tunic then open the scissors.

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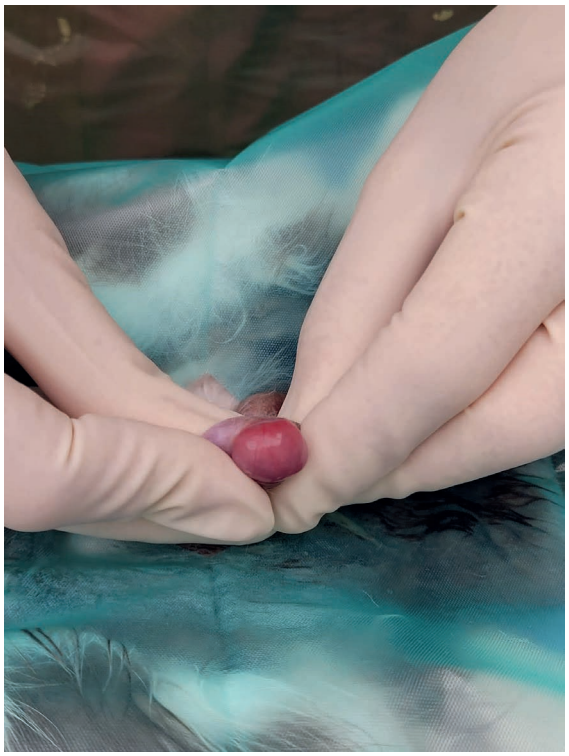


Figure 2.19 Gently squeeze the testicle caudally while your index finger and thumb of your dominant squeeze the caudal pole (epididymis) of the testicle cranially.

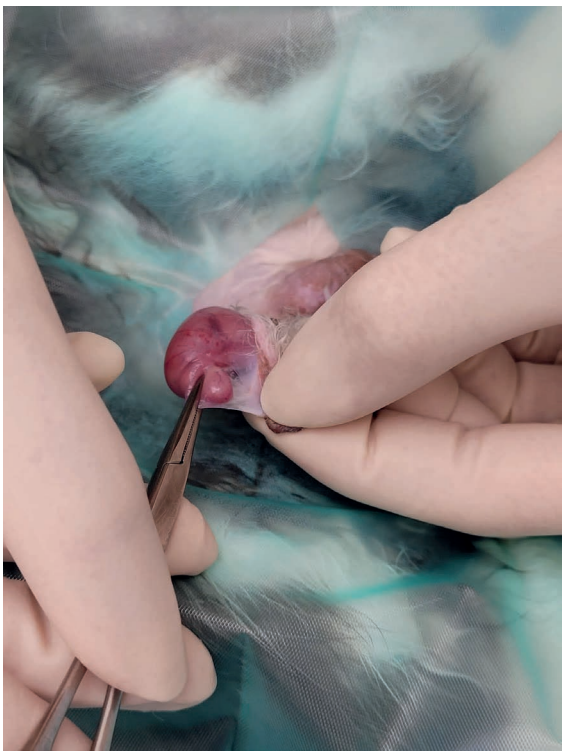


Figure 2.20 Once the epididymis is visualised through the tunic, we place an artery forcep across the epididymis.

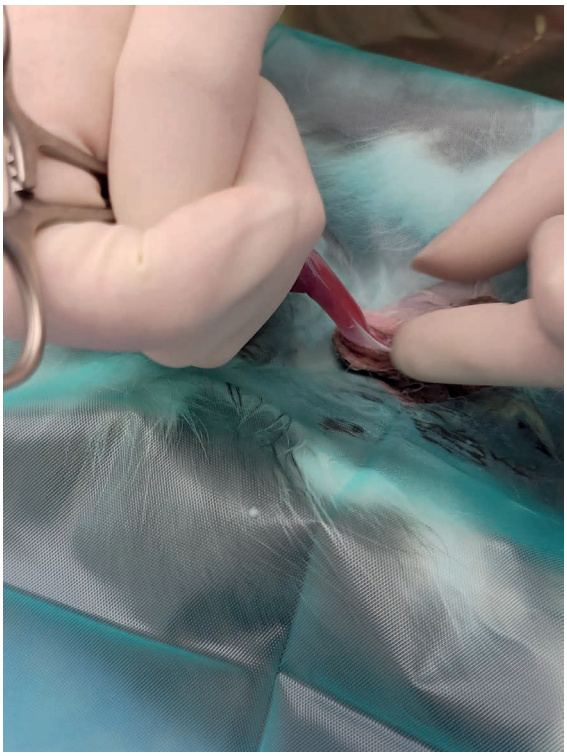


Figure 2.21 Hold the artery forcep in your non-dominant hand (do not pull upward) and use a swab to help strip the rest of the scrotum down off the tunic.

Open/closed technique

Mature bucks often have very thick tunics and the testicle is not always easily visualised. Rabbits also have very elongated testis and have a long fat pad (Figure 2.22). This means it can be difficult for inexperienced rabbit surgeons to be confident they have the entire testicle. Making a small incision into the vaginal tunic directly over the testicle and milking the testicle through the incision allows direct visualisation and confirmation that the whole testicle will be taken. This is the 'open' part of the technique.

Place artery forceps across the tunic 1 cm cranial to the base of the testis. My personal ligature of choice is Vicryl or a similar material. I do not pre-crush ligature sites in rabbits as the tissue can be very friable. Use 2 metric (3/0) Polyglactin 910 with a swaged-on needle (Vicryl, Ethicon) to perform a transfixing ligature which includes the tunic, vascular bundle of the testis and the vas deferens. I also place a circumferential ligature (my preference in all species is to place two ligatures if I have space). This converts the technique back into a 'closed' technique. No viscera can herniate as the tunic is now closed.

Place another artery forcep directly above the one already placed, so that the two are touching. Keeping the one closest to the ligatures still and steady, rotate and twist the other forcep away (Figure 2.23). This causes the tissues to stretch and tear, any remaining tissue can be cut.

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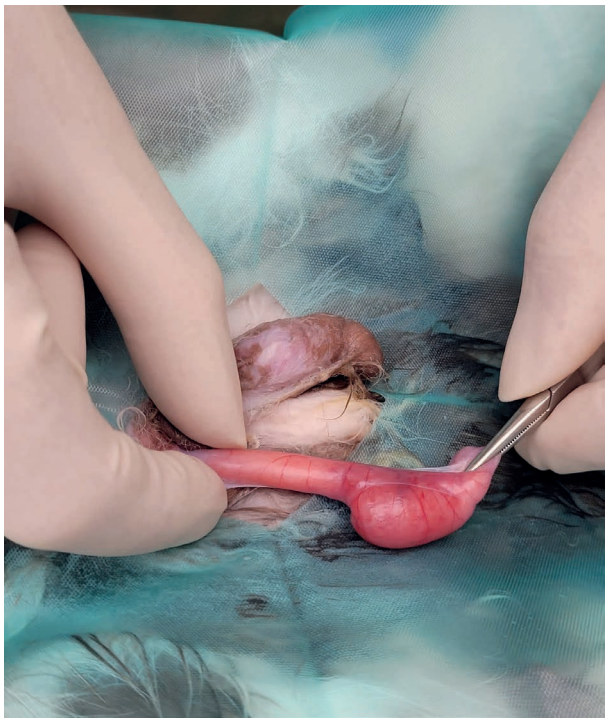


Figure 2.22 Rabbits have an elongated testis and a long fat pad.

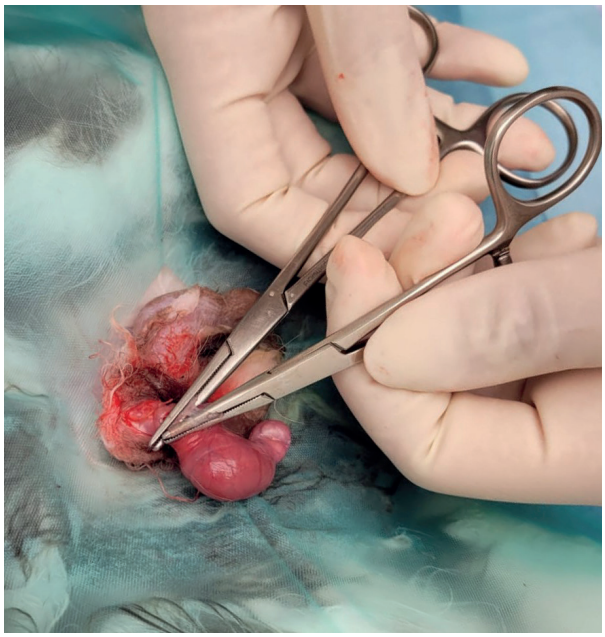


Figure 2.23 Keeping the forcep closest to the ligatures still and steady, rotate and twist the other forcep away.

Check for any bleeding and tuck the stump back into the scrotal sac.

Massage the scrotal sac to bring the edges of the wounds into alignment prior to applying wound glue (Figure 2.24). EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) may then be applied.

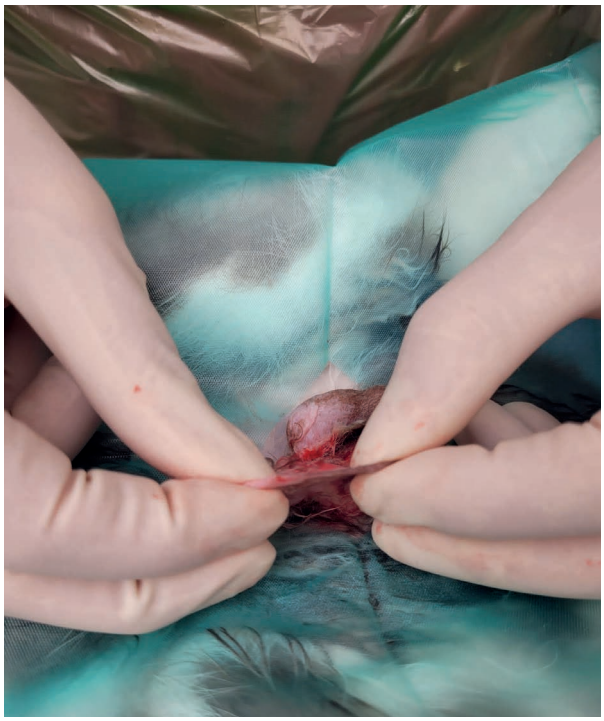


Figure 2.24 Massage the scrotal sac to bring the edges of the wounds into alignment prior to applying wound glue. EMLA may then be applied.

Closed technique

The only difference is that we skip the step of opening into the tunic to check we have the full testicle. It is very common for the penis to protrude during surgery, which is not concerning. Syringe feed on recovery and send home with a week of meloxicam.

Hospitalisation

As a prey species, extremely sensitive to stress caused by the sight, sound, and smell of predators, there are several factors we need to take into consideration. *Stress is immunosuppressive for any species and as such we aim to minimise it.*

In an ideal world we would all have prey species wards. For many practices that is not the reality. Other options include setting up pens/collapsible kennels in a quieter area of the clinic or utilising the cat ward provided the kennels are not facing each other.

The ward should be kept warm, but care should be taken *not to heat above 22°C (71.6°F)* as above this temperature heat stroke becomes a real risk. I aim to keep my ward between 18–20°C (64.4–68°F) as this ensures the animal is not having to expend energy to stay warm. A digital max/min thermometer and hygrometer (purchased inexpensively from a well-known online retailer) allows me to monitor the temperature and the humidity within the room.

Prey species do not like absolute silence. In the wilderness there is always birdsong or other animal noises unless

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there is a predator around. For this reason, playing music at a low level is recommended. Two rabbit specific appeasement pheromones are now available: Rabbit Comfort (Beaphar) and Secure Bunny (Signs Products). I use Pet Remedy (Pet Remedy Ltd) plug-ins in each of my wards to try and help mitigate stress while hospitalised. *Stress (environmental, inappropriate handling or severe pain) has been shown to cause a marked and prolonged reduction in renal blood flow and glomerular filtration rate and oliguria may result. Stress also causes gut stasis and we need to be conscious that acute renal failure can accompany any severe life-threatening disease in rabbits*. Consider a nutraceutical such as zylkene in stressed, hospitalised rabbits or midazolam. Personally, I use the following doses for zylkene: rabbits <1.5 kg 75 mg, rabbits 1.5–3 kg 225 mg, rabbits > 3 kg 450 mg.

Kennel set up

How the kennel/pen is set up is also important. *Slippery surfaces are stressful and can result in injuries*. Lining the floor with paper and providing a deep bed of hay is a good choice. If the rabbit is used to having blankets or towels at home, then they can also be used.

A litter tray with a familiar substrate should be given if the rabbit is litter trained. I have learnt from bitter experience that some rabbits will not pass faeces if they are not given a tray. A hide should be given which can be a cardboard box or even half the front of the enclosure being covered by a blanket.

Providing both water bottles and heavy earthenware water bowls (helps prevent spillages) can encourage water intake. Fresh grass and dandelion leaves should be offered to every inpatient and can act as an appetite stimulant to many rabbits.

For elective procedures vets or front of house staff can request the owners bring their rabbits a 'packed lunch' of favourite and familiar foods. Owners often love this phrase and I have experienced some well packed and personalised lunch boxes! If the rabbit is being admitted as an emergency, then I will ask the owner to bring in some of their normal food if it is not a food I already have. *Familiar food is important even if it is not ideal*, for example, muesli. Once the pet has recovered, that is the time to gradually change the diet to a more advisable one.

Admit the bonded companion

The bond between rabbits is scent based. Failure to admit the bonded cage mate can result in stress-induced anorexia (in either or both rabbits), the breakage of the bond, fighting, injuries, upset owners, and it may be difficult to re-bond the rabbits. The security of their companion may also lessen the perceived effect of pain as has been shown in rodents. *I always advise admitting the bonded cage mate at the same time*. Owners are, in my experience, happy to bring the cage mate in once they understand why.

In cases where there are more than three bonded rabbits (three cage mates is my limit), I will ask the owner to bring in the one it spends the most time with. With larger groups of rabbits, it is common for them to 'buddy' up. Scent swapping (rubbing each group with a clean towel then using the towel to rub the other group transferring the scent) to ensure both groups are marked with the communal scent can help ensure a smooth reintroduction at home.

Hospitalisation sheets should be used, and the animals weighed twice daily.

Venipuncture sites

Wrapping the rabbit in a towel (bunny burrito see Video 2.1) using EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) makes venipuncture much easier.

My vein of choice is the marginal ear vein, which is located on the dorsal aspect of the pinna running parallel to the lateral margin of the pinna, both for intravenous (IV) catheter placement and blood sampling. I regularly obtain 2 ml samples from this site although I often use only a 23 g needle and allow the blood to drip directly into the blood collection tube. I have collected 8 ml of blood from the marginal ear vein, it is perfectly possible with patience.

As with more familiar species one vein is often bigger than the other, therefore I clip fur over the vein in both pinnae (Figure 2.25). I apply approximately 1 mm thick



Figure 2.25 With the rabbit wrapped in a burrito, both pinnae have fur clipped over the later margin to see which vein is biggest.

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layer of EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen), enough so there is a noticeably white patch, then I get the rest of my equipment organised. In my experience, EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) takes around five minutes to cause venodilation, some local anaesthesia and does not require a dressing to work. *Pick your catheter size judged on the size of the vein, not the size of your patient*. For most rabbits I will end up using a 26 g IV (purple in UK) catheter, but for the bigger rabbits I have used up to a 20 g (pink in the UK).

Placing IV catheters in rabbits is extremely easy. The ear vein is fixed in place by the skin (therefore it does not 'bounce') and it is very superficial. Winged catheters are much easier to tape in as you can bend one wing around the edge of the pinna. Dressing tape such as Micropore (3M) is applied proximal and distal to the cannula in exactly the same manner we would in our canine patients (Video 2.2). *I do not use bandaging materials inside the pinnae or over the cannula*. In my experience, the added weight from these materials can irritate the rabbit and increase the likelihood that the patient removes their cannula.

Alternative sites for venipuncture are the cephalic vein, lateral saphenous vein (Figure 2.26), or the jugular. I rarely utilise the jugular as pet rabbits tend to find it stressful, plus obesity and the presence of dewlaps can make the procedure much more difficult.

The cephalic vein is popular with many vets, but I tend to only use it for blood sampling as they are much more likely to chew IV catheters out of this site. *Elizabethan collars should not be used in rabbits*. Elizabethan collars make eating food more difficult (not what you want in an anorexic patient!), the eating of caecotrophs impossible, and are an unnecessary source of severe stress.



Figure 2.26 Lateral saphenous vein. holding the rabbit at the edge of a table with the leg to be sampled being allowed to drop over the side of the table allows easier access. The vein is raised above the stifle.

The maximum volume of blood that can be safely taken is 1% of their body weight. Therefore, in a 2 kg rabbit we could safely take 20 ml, which is more than we would ever need to. Many biochemistry analysers can run a profile on 0.3 ml of blood.

Biochemistry and haematology profiles can be run 'in-house' using our usual mammalian profiles or sent to an external laboratory. Both options have pros and cons. The main pro for running in-house is that you obtain your results quickly whereas the main con is that you have to interpret whereas many labs will provide interpretation upon request.

See Table 2.2 for reference ranges for biochemistry and haematology.

Drugs contraindicated in rabbits

If in doubt, always check the formulary as it will often tell you whether it is safe to use a drug in any given species. My favourite formulary is James Carpenter's *Exotic Animal Formulary* now on its sixth edition (Carpenter & Marion, 2022).

Antibiotics to avoid in rabbits include penicillin, amoxicillin, clavulanated amoxicillin, ampicillin, clindamycin, lincomycin, and erythromycin. These drugs, especially if given orally, will cause a fatal antibiotic induced gut dysbiosis. The last three mentioned antibiotics are not safe to be given by any route.

> Penicillin and amoxicillin can be given safely by subcutaneous injection, although I always warn the owner there is a small risk. *We need to remember that if the drug leaks onto the coat, it needs to be thoroughly washed off as grooming equals oral ingestion.*

Fipronil should not be used in rabbits as it has been associated with several mortalities. Corticosteroids should be used exceptionally sparingly in rabbits. It is rare for me to use steroids in rabbits. They are very steroid sensitive and the resulting immune suppression can cause *Pasteurella* flare ups for example.

Species-specific anatomy/behaviour

Rabbits are lagomorphs with elodont – hypsodont dentition (elodont: continuously growing throughout life and hypsodont: long anatomical crown compared to the root). They have four incisors in the maxilla and two in the mandible. Dental formula: $2 \times (I\ 2/1;\ C\ 0/0;\ P\ 3/2;\ M\ 3/3) = 28$.

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Table 2.2 Rabbit biochemistry and haematology.

BIOCHEMISTRY (Korn et al., 2018; *Carpenter and Marion, 2018)	REFERENCE RANGE JUVENILE (UK units)	REFERENCE RANGE JUVENILE (US units)	REFERENCE RANGE ADULT (UK units)	REFERENCE RANGE ADULT (US units)
ALKP	87.3–217.8 IU/l	87.3–217.8 U/l	21.0–121.0 IU/l	21.0–121.0 U/l
ALT	6.0–24.9 IU/l	6.0–24.9 U/l	6.0–33.0 IU/l	6.0–33.0 U/l
AST*	–	–	14–113 IU/l	14–113 U/l
AMYL*	–	–	200–500 IU/l	200–500 U/l
GLDH	2.2–44.0 IU/l	2.2–44.0 U/l	2.0–14.0 IU/l	2.0–14.0 U/l
GGT	0.7–13.8 IU/l	0.7–13.8 U/l	0.0–14.0 IU/l	0.0–14.0 U/l
LDH*	–	–	34–129 IU/l	34–129 U/l
CK	279.5–1649.3 IU/l	279.5–1649.3 U/l	248.0–2493 IU/l	248.0–2493 U/l
GLU	6.0–11.9 mmol/l	108–214 mg/dl	6.23–11.91mmol/l	112.24–214.57 mg/dl
CHOL	1.0–5.1 mmol/l	38.61–196.91 mg/dl	0.28–2.65 mmol/l	10.81–102.32 mg/dl
TRIGYLCERIDES	0.5–4.2 mmol/l	44.25 –371.68 mg/dl	0.2–2.62 mmol/l	17.7–231.86 mg/dl
BA*	–	–	< 40 µmol/l	< 40 µmol/l
TBIL	0.0–1.3 µmol/l	0–0.08 mg/dl	0.1–2.12 µmol/l	0.01–0.12 mg/dl
UREA	2.6–8.1 mmol/l	15.62–48.65 mg/dl	5.5–11.6 mmol/l	33–69.67 mg/dl
CREA	24.6–69.0 µmol/l	0.28–0.78 mg/dl	35.0–127.0 µmol/l	0.4–1.44 mg/dl
TP	40.1–65.9 g/l	4–6.59g/dl	51.5–67.0 g/l	5.15–6.7 g/dl
ALB	28.4–44.3 g/l	2.84–4.43 g/dl	33.5–44.0 g/l	3.35– 4.4 g/dl
GLOB	11.3–22.3 g/L	1.13–2.23 g/dl	15.7–26.5 g/L	1.57–2.65 g/dl
CA	1.9–5.3 mmol/l	7.62–21.24 mg/dl	1.51–4.26 mmol/l	6.05–17.07 mg/dl
PHOS	1.3–3.2 mmol/l	4.03–9.91 mg/dl	0.69–2.06 mmol/l	2.14–6.34 mg/dl
K	3.7–5.78 mmol/l	3.7–5.78 mEq/l	3.95–5.61 mmol/l	3.95–5.61 mEq/l
Na	111.4–149.2 mmol/l	111.4–149.2 mEq/l	124.0–147.0 mmol/l	124.0–147.0 mEq/l

HAEMATOLOGY	UNITS	REFERENCE RANGE
Red blood cells	10^{12}/l	5.1–7.6
Packed cell volume	%	30–40
Haemoglobin	mmol/l (g/l)	6.2–9.31(100–150)
Mean corpuscular volume	fl	60–69
Mean corpuscular haemoglobin concentration	mmol/l(g/l)	18.62–21.72(300–350)
Mean corpuscular haemoglobin	pg/cell	19–22
Reticulocytes	%	2–4
Platelets	10^{9}/l	250–650
White blood cells	10^{9}/l	5.2–12.5
Differential blood count (relative values)		
Monocytes	%	2–10
Lymphocytes	%	30–50
Heterophils	%	30–60
Eosinophils	%	0–5
Basophils	%	0–8

Changes in total white blood cell counts are unusual in rabbits. Heterophil:lymphocyte ratio is approximately 1:1 in a healthy adult; this alters in disease or stress.

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The *tight cardiac stomach sphincter prohibits vomiting or eructation*. They are hindgut fermenters with a large caecum and have a lower skeletal mass than other comparably sized mammals (7% versus 16%). The lower skeletal mass means their bones are lighter weight. Rabbits are built for speed, with huge muscle mass. They are the walking McDonalds of the wild for predators. This is why it is important we advise owners before we start to handle their pet: it is a recognised risk of handling rabbits that if they kick out awkwardly, they can fracture their own spine.

Stress (environmental, inappropriate handling or severe pain) has been shown to cause a marked and prolonged reduction in renal blood flow and glomerular filtration rate and acute kidney injury and oliguria may result.

Using a towel or a blanket on the table to reduce the slippery nature of the surface makes rabbits feel safer and they are less likely to struggle.

Rabbits have no pads on their feet – the tissues are protected by thick layers of fur.

Male rabbits have open inguinal canals. Failure to appreciate this and take steps to close the canal during castration can lead to inguinal herniation of abdominal organs.

Rabbits should not be given ketamine by intramuscular injection. *Intramuscular (IM) administration of ketamine can cause neuropathic pain leading to self-mutilation (including rabbits who have almost chewed legs off) in this species (it has also been seen in rodents), unsettlingly this can occur as long as two weeks after the injection meaning it may not be linked to the injection* (Figure 2.27). I do not administer IM injections in rabbits for any drugs.

Rabbits hide illness and pain. Again, from an evolutionary point of view this makes complete sense because if you show illness or weakness, you get eaten. So, by the time a rabbit looks overtly ill or painful, they literally no longer care if they live or die. Many owners underestimate this and unfortunately many of us in the veterinary profession also do too.

Osteoarthritis in rabbits

Acute and surgical pain is easier to appreciate but what about chronic pain especially arthritic pain?

Owners can be shocked or initially disbelieving of a diagnosis of osteoarthritis in a rabbit of less than 3 years of age, yet I frequently diagnose osteoarthritis in that age group. How I explain this seemingly strange occurrence is discussed below.

Osteoarthritis is a degenerative condition affecting the cartilage and bones of a joint. It is commonly referred to as the wear and tear arthritis. Wild rabbits commonly die before 1–2 years of age (many die before even reaching reproductive age) due to a combination of pressures such as: predation, disease, or trauma (cars etc.).

In captivity (protected from such pressures) wild rabbits have attained 9 years of age. Therefore, the evolutionary pressures on rabbits have selected for fast moving, light boned individuals that breed at a young age, have a short gestation, and produce many young.

From an evolutionary point of view, by the time a rabbit is 1–2 years of age it is already skeletally old. To me it makes complete sense why 'young' domesticated rabbits develop wear and tear arthritis, as evolutionary pressure did not select for a robust skeleton designed for longevity. *The added pressures that domestic rabbits face such as: enclosures that limit proper movement, lack of UVB light, inappropriate flooring/substrate, and obesity, further compound the damage and pain caused by the wear and tear arthritis*.

Once this is explained to owners, I find they completely understand why this has occurred in their beloved pet and they are much more willing to work with me. Companies such as Oxbow now produce joint supplement treats, which may provide some support to the joints. Reports are contradictory as to whether it is beneficial in naturally occurring osteoarthritis, although evidence does exist that it may be beneficial in reducing cartilage swelling. Ultimately, it should cause no harm, may work, and many owners like to give supplements.

Osteoarthritis of the temporomandibular joints will cause secondary dental disease due to pain caused by chewing. Osteoarthritis of any joints may cause secondary dental disease due to a decrease in appetite, which as it happens slowly, will often not be noted by the owner. In our clinic we have noticed a sharp decline in dental disease cases since a more unified approach to multimodal analgesia was adopted.



Figure 2.27 Self-mutilation in a rabbit 1 week after OVH. The mutilation started at the site of ketamine injection in the lumbar epaxial muscles and required months of gabapentin to allow healing.

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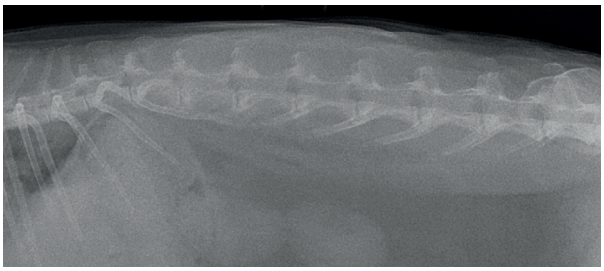


Figure 2.28a Lateral radiograph showing narrowed disc spaces.

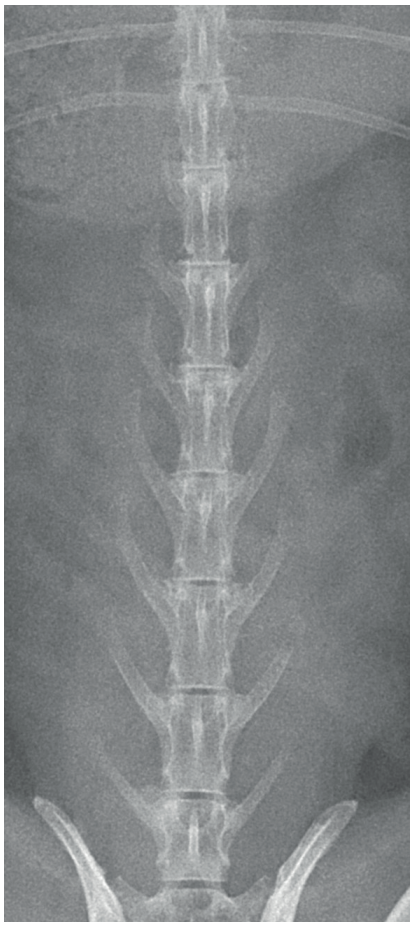


Figure 2.28b Same rabbit as 2.28a but a ventrodorsal radiographic view showing the same narrowed disc spaces.

Radiography can be utilised to investigate osteoarthritis or spinal pain in exactly the same way as more familiar species (Figure 2.28a–d).

Pain in rabbits

Ensuring that during our history taking we target our questions to include mobility can help. We already do that with cats as they tend to show more behavioural adaptations to osteoarthritic pain compared to dogs, but we often forget to do that with rabbits (stop hitting your panic button!)



Figure 2.28c A different rabbit radiograph with osteoarthritic changes to the elbow.

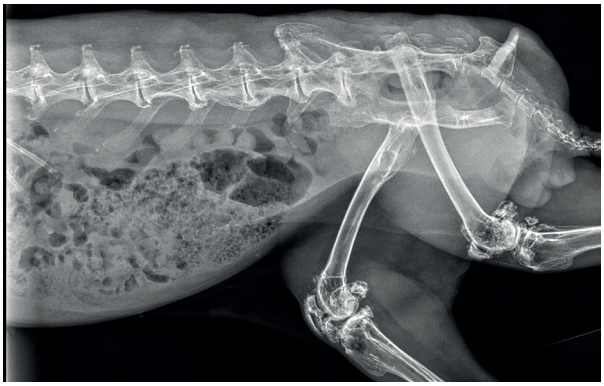


Figure 2.28d A lateral radiograph of a continental giant rabbit showing severe OA changes in the stifles and spondylosis of the spine.

Recognition and alleviation of pain are an essential component of every animal's care.

One of our jobs as veterinary professionals is to *actively look for pain*. In Chapter 1 we discussed that all vertebrates have the same anatomical and physiological pain pathways.

If we accept that animals and people share similar mechanisms of pain and analgesic action then we should realise that the variation of analgesic efficacy in different individual people means that it is highly likely that a similar variation will be seen in animals – further reinforcing the need for regular pain assessments.

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Chronic pain negatively impacts quality of life severely. It causes chronic maladaptive stress, activation of stress hormones, sleep disruption, immunosuppression, and reduces social interactions (which for an innately social species further exacerbates stress levels).

A grimace scale has been developed for rabbits and can be found at: https://www.nc3rs.org.uk/3rs-resources/grimace-scales/grimace-scale-rabbit.

Stress and fear may increase the degree of pain perceived by an animal and steps should be taken to minimise these emotions.

How many veterinary professionals genuinely perform the exact same clinical exam in a rabbit that you do in a dog? How many do a full skeletal exam, spinal palpation, and limb range of motion in every rabbit patient? If you do not do this – why not? Osteoarthritis and spinal pain is massively under diagnosed in rabbits simply because we do not always think to look for it (Video 2.3).

Placed on a towel for security, the neck flexion tests are performed exactly the same as in more familiar species. Extending the forelimbs one at a time allows you to differentiate if more pressure was required to extend one limb therefore identifying pain. View the rabbit from above, does the rabbit lean more to one side (taking pain off the sore side) or is a limb abducted? (Figures 2.29a & b) These signs are subtle and are easily missed if not actively looked for.



Figure 2.29a This rabbit is sitting with the weight shifted mainly to the right to alleviate pain. The left hind is no longer tucked under the rabbit (which in normal rabbits it would be).



Figure 2.29b The same rabbit as Figure in 2.29a, this time I have placed my fingers on either side of the pelvic bones to highlight the tilt. My middle finger is higher than my pointer finger whereas they should be at the same level.

Spinal palpation in rabbits can make vets nervous. Please let me reassure you, you will not fracture a rabbit's spine by performing a good spinal palpation. The exact same level of pressure is needed that you would use on a Jack Russell terrier. Failure to perform correctly will lead to failure to spot pain. Remember to palpate the cervical spine and that the thoracic spine runs between the scapula and needs palpating too.

Rabbits will not vocalise or turn their heads to look at you if they are painful during spinal palpation. *They vibrate when you press on a painful area of their spine. The epaxial muscles will spasm* also (Video 2.4). Back pain can sometimes be confused with abdominal pain and vice versa. There are several pain scoring methods available for rabbits.

My routine for hands off assessment of 'is this rabbit sore', is to look at ear position (more challenging in lops) and eye shape. Rabbits that are very painful tend to hold their ears flat against their backs rather than in a relaxed upright/slightly slanted position. Their eyes take on a squinting shape when the rabbit is painful (Figures 2.30a–d).

Analgesia in rabbits

Consideration of the type of pain we are looking to control will lead to logical choices of the type of analgesics we select. *Multimodal analgesia is recommended* just as in more familiar species. *Rabbits have a much higher metabolic rate than our more familiar species and as such their required doses will be much higher.* I manage owners' expectations by explaining that 'pain winddown' can take 8–12 weeks in chronic pain cases but that I reassess after 2 weeks. Asking owners to focus on three behaviours can also help their perception of whether their pet has improved: e.g. using

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Figure 2.30a Note the wide, open, round shape of the eye which is normal in the rabbit.



Figure 2.30b Note the eyelids have become slightly flattened and now have a 'sleepy' or mild squint. During the clinical exam it is not normal for rabbits to appear sleepy. This rabbit is sore.

a litter tray, lying fully stretched out, jumping up on the sofa.

Oral or parenteral vitamin B has been shown to reduce inflammatory and neuropathic pain in many species and can be used as part of the multimodal analgesic approach.

My preferred non-steroidal anti-inflammatory (NSAID) is meloxicam. I have been using meloxicam at 0.6 mg/kg q 12 hrs for around 20 years now. Recently some studies have shown that certain individuals will require up to 1 mg/kg meloxicam to achieve blood levels consistent



Figure 2.30c Note the eyelids have become flattened and now have a pronounced squint. This rabbit is very sore. I had seen the rabbit for a second opinion after a pinnae resection where electrocautery had been used to resect the pinnae but no attempt to stitch the skin over the cartilage had been made. Rabbits have exquisitely sensitive ears, the wounds were not healing and the rabbit was becoming more miserable. After corrective surgery to cover the cartilage and multimodal analgesia the rabbit became playful within 24 hours and normal eye shape resumed.



Figure 2.30d Note the eyelids are only open a small amount during the clinical exam. This rabbit is in a lot of pain.

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Table 2.3 Rabbit analgesia.

Analgesic	Dose
Meloxicam	0.6–1 mg/kg q12hrs PO, SC
Paracetamol (acetaminophen)	15–25 mg/kg q8–12hrs PO
Tramadol	10–30 mg/kg q6–12hrs PO
Gabapentin	10–30 mg/kg q8–12hrs PO
Maropitant	4 mg/kg SC
Methadone	0.3–0.7 mg/kg q4hrs SC dependant on pain scoring
Amantadine	3–5 mg/kg q24hrs PO
Lidocaine	2 mg/kg (maximum 4mg/kg)
Bupivacaine	1 mg/kg (maximum 2mg/kg)
Acupuncture	

with analgesia. Personally, I evaluate response to analgesia and adjust as required. Just as with more familiar species I utilise meloxicam in many types of pain. *As rabbit stomachs always contain food, I am not concerned about administering with food in this species*.

Paracetamol (acetaminophen) works well if given in conjunction with meloxicam. I order mine from my usual wholesaler and use 15–25 mg/kg q8–12hrs.

In my experience tramadol works extremely well for various types of moderate pain. In humans, around 20% of the population do not get any analgesic effects from tramadol and I am highly suspicious the same will be true in some animals due to the lack of a particular enzyme. Liquid tramadol is often more cost effective when obtained from a compounding company. However, many rabbits will take the contents of 50 mg capsules dissolved in a known quantity of water (therefore providing you with a mg/ml solution), which the owners can make up daily for oral administration. I use 10–30 mg/kg q6–12hrs.

Gabapentin works well for neurogenic pain and is my go-to analgesic if I am suspicious of disc disease or in cases of root disease. The dose may need adjusting up if the patient has been taking the medication for a long time. *This further highlights the importance of regular pain scoring*. I start my gabapentin at 10 mg/kg q 12 hrs and will go up to 30 mg/kg q8 hrs. Chronic pain results in anxiety, gabapentin also has an antianxiety effect.

Maropitant can be useful for visceral and skeletal pain, administered by SC injection at a dose of 4 mg/kg. The exact mechanism is unknown and more work needs to be done on how much analgesia is provided but I have certainly found it to be useful in some cases. Also, just because rabbits cannot vomit does not mean they cannot feel nauseous.

Methadone has a short half-life in rabbits but it is useful in cases of extreme pain. I will start at the lower end of the dose range and titrate up if I am not satisfied with the level of analgesia provided. I will start at 0.3 mg/kg and increase up to a maximum of 0.7 mg/kg, this will last for between 2 to 4 hours and further dosing is dependent on pain levels. *Opioids can cause problems with gut motility but so can pain*.

Amantadine is an antiviral (in humans it is mainly used to treat Parkinson's) which works on the NMDA receptors and provides analgesia as part of a multimodal approach. I have not used it often in rabbits, the dose recommended is 3–5 mg/kg PO q 24 hrs.

Ketamine provides analgesia by blocking the NMDA receptors. I have used it at the same dose used in dogs 0.5mg/kg SC q7 days for four weeks then monthly or as required.

Further analgesia can be provided by performing local anaesthesia splash blocks on closure of surgical sites. A mixture of lidocaine 2 mg/kg (maximum 4 mg/kg) and bupivacaine 1 mg/kg (maximum 2 mg/kg) can be used. Doses higher than the maximum recommended may lead to toxicity.

Acupuncture has shown great success in alleviating chronic pain as part of a multi-modal approach. It has been shown to work by inducing endogenous opiates and reportedly naloxone will reverse analgesic effects of acupuncture. *The acupuncture needles do not cause damage to the muscles therefore are not associated with self-mutilation. This makes sense as the needles are solid and not being used to inject a substance into the muscle, disrupting the fibres*. We use acupuncture regularly in our clinic in rabbits (Figures 2.31a & b).

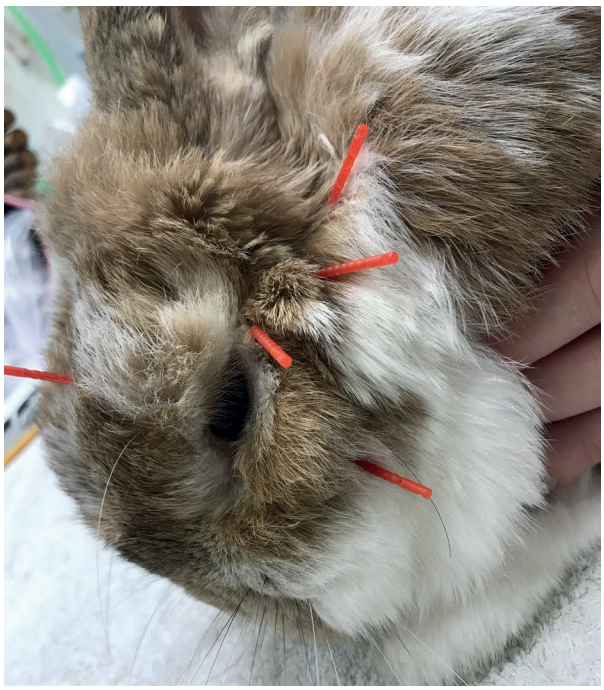


Figure 2.31a This rabbit is receiving acupuncture to help with dental pain. Note the squinting eye position. Photo: Dr Krissy Green.

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Figure 2.31b This rabbit is receiving acupuncture to treat spinal pain. Photo: Dr Krissy Green.

Clinical exam

As previously discussed, we warn owners about the risk of spinal damage and we place a towel or blanket on the table. No animal likes a slippery surface. Prey species have evolved to be prey, therefore have an innate requirement to escape predation at a split-second notice. This is why they find a slippery surface so frightening, if they cannot find purchase, they cannot escape predation. Using the blanket or towel reduces the chance of them injuring themselves or you as their handler.

If possible, take the top off the carrier to enable easier, less stressful removal of the rabbit. I will look at the rabbit face on to check for facial symmetry. Facial nerve paralysis can be as subtle as one upper lip drooping compared to the contralateral lip and is easily missed without this step. Conversely, inner ear disease can result in facial nerve irritation initially leading to a facial grimace, i.e. the lip is held higher on the affected side and may become paralysed leading to a droop in later cases (Figures 2.32a–c).

Personally, I prefer to perform the rest of the examination from behind so that the rabbit is facing its owner. I perform the same clinical exam, in the same order that I do for any other species (except that I perform oral examination of the molars last). If we do this consistently, we will diagnose issues, we may not know how to treat them at this stage but that is when we pick up a book or phone a colleague for advice. *Owners really do not expect us to be walking encyclopaedias – they expect us to care*. See Table 2.4 for biological data.

Rabbits have two blind spots, directly below their nose and the opposite position at the back of their heads. This means if you immediately try to look at the incisors you can startle your patient!

I start by stroking the animal's head, between the eyes and under the eyes as this is an evolutionary safe



Figure 2.32a This rabbit has a left sided facial grimace due to an ear-based abscess and otitis interna/media.



Figure 2.32b This rabbit has a much more subtle right sided facial grimace due to an ear-based abscess and otitis interna/media.

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Figure 2.32c Radiograph of the rabbit pictured in 2.32b, note the destruction of the tympanic bullae. This is unusual in my experience, it is much more common to see increased radiopacity of the affected tympanic bullae.

Table 2.4 Biological data.

Parameter	Reference range
Average longevity	8–12 years
Respiratory rate	30–60 bpm (nose twitches do not correspond with breathing)
Heart rate	250–350 bpm
Body temperature	38.5–40°C (101.3–104°F)
Maintenance fluid therapy	100–150 ml/kg/day
Sexual maturity	4–6 months (varies with breed)
Gestation	28–32 days

place to touch them – i.e. this is where other rabbits will groom them. Once I have done this, I will progress on to stroking the lateral cheeks starting from below the eyes, moving gradually forward with each stroke until

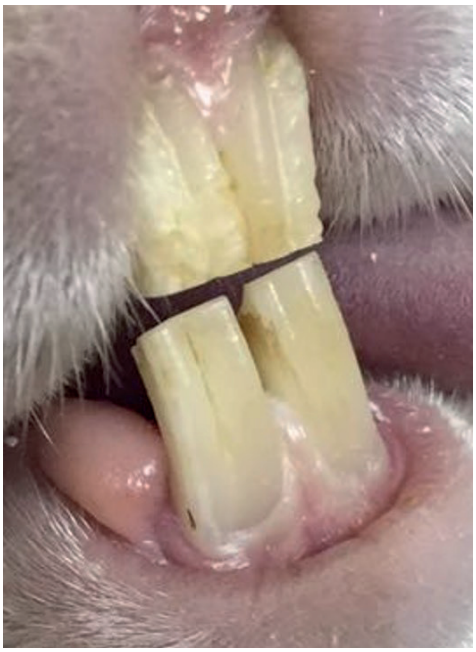


Figure 2.33 Note the horizontal ribbing of the enamel, which is completely abnormal.

I reach the upper lips and I can gently part the lips and visualise the incisors. Check for symmetry, a horizontal occlusal surface, and whether there is horizontal ribbing to the enamel. The presence of any asymmetry, diagonal occlusal surfaces, and/or horizontal ribbing are all indicators that dental disease is present and a careful oral clinical exam of the molars should be performed (Figure 2.33).

Is there any evidence of nasal discharge? As obligate nasal breathers, rabbits fastidiously groom their face and nose, so a minor nasal discharge may not be evident at the nares – check the medial aspect of their front legs by running your thumbs down the inside of the front legs. If there is a nasal discharge, we will often feel crusting on the fur. I will assess the length of the nails on the front feet at the same time. Nails that are too long will alter the weight bearing during movement, which can exacerbate pre-existing joint disease.

Palpate the mandible for any masses, which may or may not indicate dental disease.

I check for ocular symmetry from above the head, is there any evidence of exophthalmos? Are the eyelids normal? Take note of any ocular discharges, massage rostro-distal to the medial canthus to check for discharge which may indicate dacryocystitis. Is the third eyelid protruding? Is there any inflammation? Are any cataracts present? Apply fluorescein to check for corneal ulcers.

Palpate the base of the pinnae where they join the skull. I compare both sides at the same time as I am looking for any asymmetrical swellings at the base which could indicate an ear-based abscess (more prevalent in lop eared rabbits – see

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section on ear disease). Rabbit pinnae are very sensitive and the pain of ear disease can be accompanied by anorexia – sometimes anorexia is the only clinical sign the owner will notice. Is there any sign of: crusting, wax (normal wax is very pale in rabbits and can vary from light brown to almost white), ear mites (*Psoroptes cuniculi*), inflammation, foreign bodies (grass awns, etc), masses, pus (may require cytology as can be hard to differentiate from wax but other clinical signs will help). Remember – they are just ears!

A skeletal examination performed the same way as more familiar species and as previously discussed in the Pain section is my next step. I also check for thoracic compliance by compressing the thorax laterally mid thorax. *Lack of thoracic compliance indicates that there is pathology in the thorax such as: thymoma, abscessation or dilated cardiomyopathy to name a few*. Sometimes this subtle finding is the only clinical sign indicating radiography. If the mass is big enough to cause decreased venous return, then bilateral exophthalmos will be present due to engorgement of the large venous sinus behind the globe. *I will ask the owners if the eyes have been like this at home as some rabbits' eyes bulge mimicking exophthalmos if they are very fearful at the clinic or have been stressed badly by the journey*.

Thoracic auscultation is performed on both sides and listening to several areas on each side. Take note of any adventitious noises but also of any areas of dullness. If you are finding it difficult to differentiate between upper respiratory sounds and lower respiratory sounds – listen to the ventral neck. We do this all the time in brachycephalic dog breeds. I do not count heart rates in rabbits as I find it impossible with heart rates between 250–350 beats per minute. If it is beating, I cannot count it (if I can count it then the rabbit may be bradycardic), and the rhythm sounds normal, I move on.

Check through the coat and examine the skin. We are looking for the same issues we would see in any examination of fur and skin: lumps, scabs, alopecia, dandruff (check for *Cheyletiella parasitovorax*), pustules, sores, wounds (including flystrike), poor grooming, and fleas (plus any other ectoparasites) (Figures 2.34a & b). Examine the genital region. Is there any evidence of urinary scald or faecal build-up? If the patient is an entire male palpate the testis. Examine the anus. Examine the palmar and plantar aspects of the feet for any sores or fur loss.

Rabbits, unlike dogs and cats, do not have pads to protect their feet, so loss of the thick fur can have devastating consequences. Ulcerative pododermatitis (hock sores),



Figure 2.34a The coat looks rough due to poor grooming by the rabbit. *In a wild animal, lack of coat care may be life threatening as they require their fur to be in good condition to help with thermoregulation*. Note also the rabbit is holding its left hind leg abducted due to pain.



Figure 2.34b For comparison, this is the same rabbit Figured in 2.34 a after a course of multimodal analgesia. The coat is smoother and the left hind is no longer abducted.

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Figure 2.35 Faeces from the same rabbit. The smaller, darker, more uneven faecal pellets are from when the rabbit started to eat less and just before the rabbit became completely anorexic. The bigger, more even, lighter coloured faecal pellets are when the rabbits started eating.

can quickly become life threatening for rabbits as the skin is thin, the tendons and bone lay in close proximity and can become secondarily infected. Overgrown nails alter how rabbits walk and can exacerbate this condition.

Gently palpate the abdomen. I start my palpation as a massage and gradually increase pressure until I can feel organs. I perform cranial to caudal and then ventral to dorsal. *Fatty liver disease occurs quickly in the ill rabbit (especially if obese), the enlarged and friable liver can rupture and cause abdominal bleeds*. Take note of any areas producing repeatable flinches (pain) and any abnormal masses. Check for tympany or gas build-up. Are there faecal pellets palpable? It really is just a gentler version of the same examination we do in other species. I often use abdominal palpation to assess hydration status as discussed below.

Personally, I do not listen for gut sounds, which is controversial. Many colleagues who I respect and admire do perform this part of the clinical exam. For me, if I have taken a thorough history (including faeces numbers, shape, size, and colour) (Figure 2.35) and performed a thorough clinical exam I know whether I have to administer gut prokinetics or not. *Vast majority of ill or painful rabbits will benefit from prokinetics (once a foreign body has been ruled out then realistically you will not cause harm)*. Gut sounds may also be misleading as in cases of foreign body the gut sounds may initially increase as the intestines try to move the blockage.

I also do not routinely take rectal temperatures in rabbits. I made this decision because the rectal wall is actually very thin and during laparotomies, I can see the colour and consistency of the faecal pellets easily through the colon. For me the risk of a rabbit being injured outweighs the potential benefit of the knowledge of the rectal temperature. *As rectal temperature can be a prognostic indicator, I do take the rectal temperature in dull or moribund rabbits and I also monitor the rectal temperature during anaesthesia*.

I perform the conscious oral dental examination last as most rabbits do find this stressful. Many owners are unaware that rabbits have molars and that all their teeth grow continuously. I advise owners that we can miss up to 50% of dental disease cases during conscious oral exam as rabbits chew the scope and stick their tongues down the end of it!

Performing conscious oral clinical exam

After wrapping the rabbit in a bunny burrito, I will ask an assistant (which may be the owner) to hold the rabbit vertically with its back nestled against their breastbone supporting the thorax and its hindquarters. I use the medium metal otoscopic cone.

Restraint of the head is important. I use my non-dominant hand (for me that is my left hand) to restrain the head, standing on the right side of the rabbit I will hook my left pinkie finger behind and around the base of the left pinnae, I place the palm of my hand flat onto the skull and use my index (pointer) finger and thumb to hold onto the point of the zygomatic arch just distal to the eyes. I then pull gently caudally with my index and thumb. This achieves three things: gently lifts the rabbits head slightly, opens the nares, and prevents your fingers from passively slipping forward and occluding the nares during the oral examination. It is difficult to passively slip forwards when you are actively pulling back (Figure 2.36).



Figure 2.36 Restraining a rabbit's head for oral clinical exam when conscious using an otoscope.

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Rabbits will not normally have food (unless they have literally been eating immediately before the examination), excess of saliva or fur in their mouth. Presence of these increases the clinical index of suspicion for dental disease.

I place the otoscope cone into the right diastema just caudal to the upper incisors. I move over the tongue and use the scope to move the tongue out of the way. Examine the soft tissues for any wounds, ulceration, or bleeding. Estimate the length of the lower left arcade. Are there any spikes growing medially towards or into the tongue? Move the scope up to visualise and estimate the length of the left upper arcade and compare the symmetry of both upper arcades. Move around to the front of the rabbit but do not change the position of your non-dominant hand. Use the scope to move the cheek away from the right upper arcade, again examine the soft tissues for any wounds, ulceration, or bleeding. Is there any evidence of spikes growing buccally? Are the occlusal surfaces of the teeth almost horizontal (normal) or are they diagonal? Move around the rabbit, without taking the scope out, and perform the rest of the clinical exam to include the left upper and right lower arcades.

Many vets (including myself) used to focus on looking for spikes when performing this exam. Spikes are painful due to the soft tissue damage they cause, and many rabbits resist the soft tissue being pulled off the spike to allow visualisation. Now I advise looking for asymmetry (seeing spikes is a bonus). *Asymmetry is often easier to appreciate even in the chewiest of rabbits*. If they have pain on one side, they will preferentially chew on the other side which leads to asymmetrical wear of the continuously growing molars (Figure 2.37). If in doubt I advise full oral exam under sedation or general anaesthesia (once stabilised if anorexic) including skull radiographs. This is discussed fully in the section on dentistry.

Anorexia and gut stasis (ileus)

Anorexia is a true emergency in rabbits which can lead to gut stasis, a negative energy balance and fatty liver syndrome developing within 24–48 hours. It is important to remember that *anorexia is a symptom* not a condition in rabbits.

Unfortunately for us, anorexia is a nonspecific reaction to stress which can be: physical (pain anywhere and illness) or psychological (separation from cage mate, change in environment). A good clinical history and thorough clinical exam will often help direct your diagnostic tests. *I assume any anorexic rabbit is in pain somewhere.*

For example, if the rabbit has a painful or bloated abdomen without an obvious discrete mass, I will recommend a full general health profile and a conscious dorsoventral radiograph to check for gas in the gastrointestinal tract.



Figure 2.37 If they have pain on one side, they will preferentially chew on the other side which leads to asymmetrical wear of the continuously growing molars. Although this rabbit is anaesthetised, if I see this during an oral exam of a conscious rabbit, I know the rabbit has dental disease which requires investigation and treatment.

Measuring blood glucose levels

As an absolute minimum I would recommend using a point of care (POC) glucometer to estimate the blood glucose in an anorexic rabbit with a painful abdomen. Rabbits run on volatile fatty acids (VFA) rather than glucose (diabetes mellitus is extremely rare in rabbits apart from one strain of New Zealand whites), this means that we can utilise blood glucose to differentiate cases of anorexia which may require immediate surgical intervention from those that should be stabilised before any anaesthetic or surgical procedure is considered. I do not worry that POC are not as accurate as biochemistry machines as I am interested in trends rather than absolutes. See Table 2.5.

Table 2.5 Glucose measurement interpretation.

Glucose reading (mmol/l)	Interpretation
<15	Likely stress
15–20	Stress or pain (retest 15 minutes)
>25	Severe pain (possible abdominal catastrophe), analgise, IV fluids, full biochemistry and haematology, diagnostic imaging (retest 15 minutes)

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Glucometer readings of less than 15 mmol/l are consistent with stress, I will retest in 30 minutes (sooner if the rabbit is deteriorating) to ensure it is not increasing but otherwise I stabilise the rabbit pre-investigation as below (see Stabilisation).

Glucometer readings of between 15–20 mmol/l are more difficult to interpret, and I will begin stabilising the rabbit and retest in 15 minutes to ensure the reading is going down. If the glucose reads higher, I will add in more analgesia and retest after another fifteen minutes. If the glucose is coming down, I will continue stabilisation and retest every thirty minutes until readings of less than 15 mmol/l are achieved, then test hourly until the rabbit is eating on its own.

Glucometer readings of over 25 mmol/l can indicate abdominal catastrophe such as a gastrointestinal foreign body or a liver lobe torsion. These are the cases that may require prompt intervention with very little time available to stabilise first. Simply put they may be dead before they are stable. For these cases I advise full biochemistry, haematology, multimodal analgesia, intravenous fluids, warmth, radiography/ultrasonography and *no prokinetics until you have ruled out a foreign body*.

Normal or low blood glucose levels in dull or obviously painful rabbits should raise concerns of sepsis, or liver disease.

If the rabbit is dull, I recommend taking a rectal temperature. The normal rectal temperature of a rabbit is: 38.5–40°C (101.3–104°F). Hypothermia in rabbits can be a prognostic indicator with one study showing that rabbits with a body temperature at presentation of 37.22°C (99°F) or lower had a three times higher death rate than individuals with a normal body temperature (Di Girolamo *et al.*, 2016).

Liver lobe torsion

Sheen *et al.* (2022) found that lop breeds (in particular dwarf lops) are over represented, most rabbits were over two years of age (mean 4.6 years old), but there was no sex predilection. The exact pathophysiology is unknown, and I suspect this condition is underdiagnosed as many sudden death rabbits are not presented for investigation.

Clinical signs

Clinical signs include sudden onset anorexia, reduced or absent faecal output, lethargy, hiding, or unusual postures (due to abdominal pain). Liver lobe torsion can quickly progress to hypovolaemic shock, systemic inflammatory response, and if left untreated will progress to disseminated intravascular coagulation and death.

A painful mass may be palpable in the cranial abdomen. The stomach should not be massively distended. There may be pale mucous membranes, tachypnea, dull mentation, dehydration, and hypothermia.

Diagnosis

Liver lobe torsion will show raised liver enzymes and anaemia on blood profiles *(run in-house – there is not time to send these samples to an external lab). If the abdominal bleed is peracute then it should be noted that the haematocrit may be normal as there has not been time for a fluid shift into the vascular bed to correct the hypovolaemia*. Thrombocytopenia may also be seen. Ultrasonography may show free fluid (blood) in the abdomen and an enlarged liver lobe, with rounded lobar margins and mixed parenchymal echogenicity. If colour flow Doppler is available, it will show a lack of/reduced blood flow to the area. In the vast majority of cases (94.1% in a retrospective study, Sheen *et al.*, 2022), it is the caudate liver lobe that is affected.

Treatment

Hypothermic rabbits should be actively warmed, opiate analgesia (alongside multimodal) should be given, prokinetics, and assist feeding (provided the rabbits mentation allows). Once liver lobe torsion has been confirmed, broad spectrum antibiotics should be administered to minimise the risk of bacterial translocation. Sheen *et al.* (2022) recommended the combination of 30 mg/kg procaine penicillin SC q 24 hrs and 20 mg/kg metronidazole PO q 12 hrs for two weeks.

Prompt surgical intervention to perform a liver lobectomy is required. A circumferential ligature will achieve occlusion of the lobe and transection of the lobe is performed distal to the ligatures. In severely anaemic cases (< 20% PCV) a blood transfusion should be given perioperatively. Postoperatively monitor vitals, and ensure there is a stable PCV and stable plasma total protein (therefore no active blood loss).

Owners should be counselled that even with resection of the affected lobe, the outlook may be poor. That being said, *several studies have shown good clinical outcomes with swift diagnosis and intervention*. Supportive care should continue until the rabbit is eating and defecating. Analgesics should be continued for ten days post-operatively.

Gastrointestinal foreign body

The cardiac sphincter of the stomach prevents vomiting or burping in the rabbit. Any gastrointestinal foreign body will quickly result in obstructive stasis with dire consequences without prompt diagnosis and intervention. *Rabbits continuously produce saliva so fluid and gas will build up proximal to the blockage, resulting in gastric and/or intestinal distention and severe pain*. The stomach will be easily palpable in the left cranial abdomen. Severe hyperglycaemia and hyponatraemia will frequently be present on the blood work.

Severe gastric distention is not only extremely painful, it will impinge on the diaphragm making respiration more difficult,

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Figure 2.38a Note the severe distention of the stomach. As rabbits are diaphragmatic breathers, the pressure from the stomach was severely restricting breathing. The owner could feel the stomach and thought it was a tumour.

reduces cardiac efficiency and causes compression of intra-abdominal blood vessels leading to compromised perfusion of organs (Figures 2.38a & b) The rabbit Figured presented 30 minutes previously and was an immediate surgical case due to the rabbit struggling to breath (surgery went well, the rabbit received shock IV fluids, and went on to make a full recovery). *If a rabbit does not have restricted breathing, then I will usually try to medically manage the case as described below.*

Foreign bodies frequently consist of trichobezoars (compacted hair and food) although ingestion of carpets or rubber materials is also recognised. Repeated bouts of trichobezoars may indicate an underlying motility issue. In my experience, the most common sites of obstruction are the proximal duodenum (Figure 2. 39) and the pylorus. Stress related gastric ulceration is commonly seen and avascular necrosis of the gastric or intestinal wall can occur.

Radiography

Radiographs will show fluid and gas filled viscera. A dorsoventral view can be taken in a conscious rabbit by placing the rabbit in a plastic tub or a lateral radiograph may be taken if the rabbit is moribund or sedation can be used.

On a lateral radiograph the *signs of gastric distention are the stomach projecting past the costal arch and the stomach*

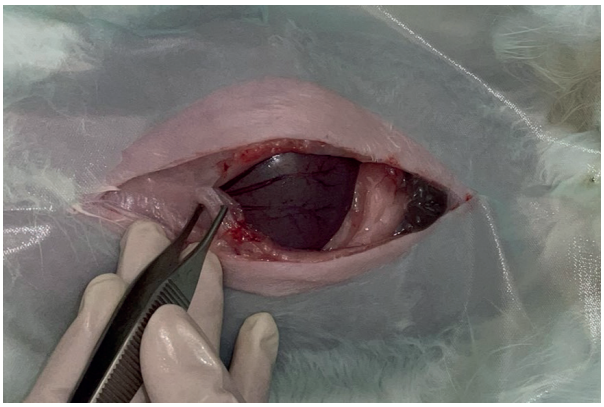


Figure 2.38b The sheer size of the distended stomach leads to compromised soft tissue perfusion. Note the lack of bleeding and the very pale muscles of the abdominal musculature.

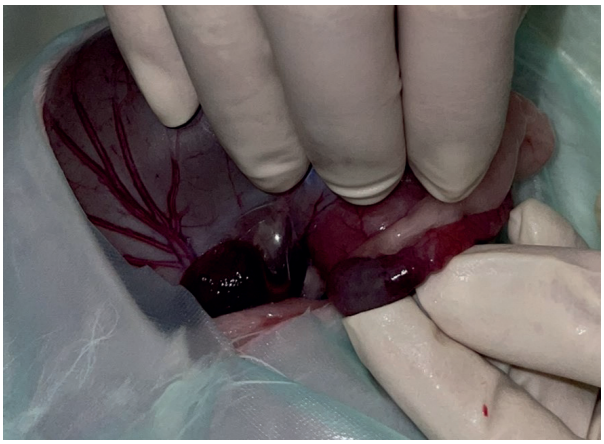


Figure 2.39 The most common site of obstruction is the proximal duodenum. This picture is an intraoperative photo of the rabbit in Figure 2.38.

contacting the ventral abdominal wall. Sometimes it may be difficult to decide if the stomach is distended due to ileus or obstruction. Steinagel and Oglesbee (2023) showed that rabbits that had gastric distension at or beyond the caudal level of L2 all had gastric obstruction, hyperglycaemia, hyponatraemia, and also had signs of shock. In their paper they described using orogastric decompression to relieve the pressure caused by the stomach to alleviate clinical signs and give medical management time to work (Figures 2.40a–c).

Orogastric decompression

Orogastric decompression of the stomach will immediately relieve: the abdominal pain due to gastric distention, the compression of blood vessels, the pressure on the diaphragm affecting the breathing of the rabbit. This may give the rabbit time to respond to medical management, but it is not without its own risks.

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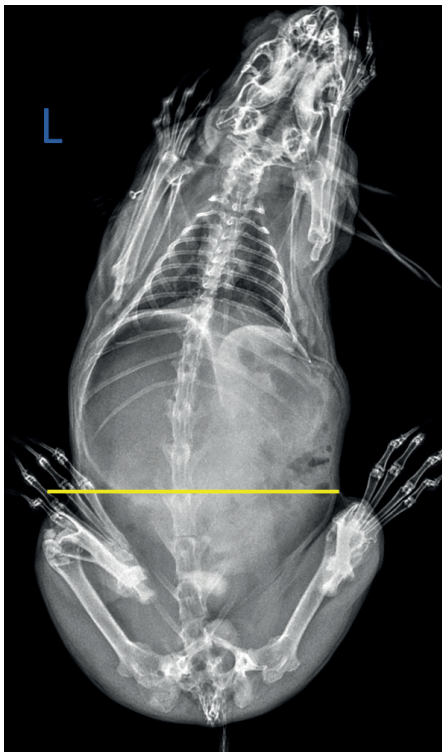


Figure 2.40a A conscious radiograph of the rabbit pictured in 2.38. The yellow line is drawn at the caudal border of L2, note the distension of the stomach beyond it.

Risks of orogastric decompression are anaesthetic risks (although these would also be same with surgery), risk of aspiration of gastric contents, and risk of accidental tissue damage during tube placement (which includes the risk of perforating an area of gastric ulceration).

Orogastric decompression can be performed under sedation with midazolam (either as a single agent or as part of a combination) unless the rabbit is severely depressed.

Equipment required: oral speculum to prevent the rabbit biting through the tube (can use medium metal cone from an otoscope), 14 Fr (4 mm diameter) nasogastric tube, permanent marker, lubricating jelly, syringe and collecting dish.

Using a 14 Fr (4 mm diameter) nasogastric tube (I usually cut the end off to make the opening larger), pre-measure the tube from the rabbits' mouth to the 10–11th intercostal space, mark the tube. The rabbit should be placed in ventral recumbency with the head ventroflexed to cause the tube to be passed into the oesophagus. Place the oral speculum into the rabbit's oral cavity, lubricate the tube and pass it through the speculum and into the stomach up to the premeasured mark. In my experience this is not as easy as texts make it sound due to the tightness of the cardiac sphincter. Gravity may result in gastric fluid flowing out of the tube but if this does not happen, positioning within the stomach may be confirmed by attaching a

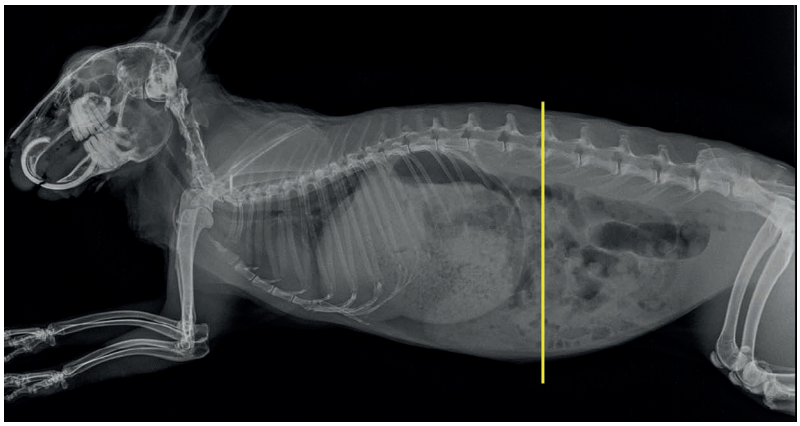


Figure 2.40b This rabbit has mild gastric distention due to stasis but as the yellow line shows, the stomach is nowhere near the caudal border of L2 and is not in contact with the ventral abdominal wall.

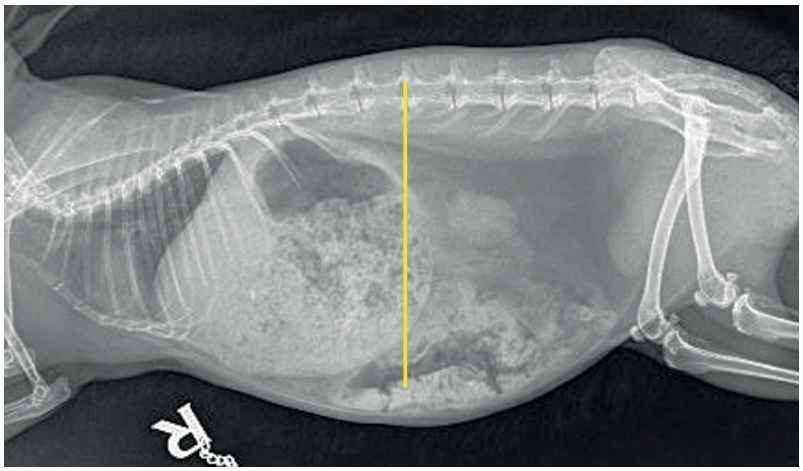


Figure 2.40c Note the stomach is in contact with the ventral abdominal wall and as the yellow line on this radiograph clearly shows, the stomach extends beyond the caudal border of L2. The contents of the caecum are dehydrated. This rabbit had a proximal duodenal foreign body.

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syringe and aspiration of gastric contents. Gentle massage of the stomach via palpation will aid removal of the gastric contents via the tube. Radiographs may be taken to confirm return to normal gastric size, although this may be achieved by palpation also.

Medical management of foreign body

Analgesics such as methadone, maropitant and paracetamol should be administered parenterally, immediately. I do not administer a NSAID at this stage due to the inherent risks associated with NSAID use in shocked animals. Warmed IV fluids are administered and I will recheck the blood glucose every fifteen minutes. Nothing should be administered orally at this stage. Thermal support should be given.

Owners should be counselled that if the rabbit does not respond to medical treatment within the first hour, then an exploratory laparotomy will be the next stage if the owner wishes to proceed with treatment. Many cases of foreign bodies in rabbits will respond to medical treatment provided adequate analgesia and IV fluids are administered. It is not uncommon for the second blood glucose reading to be higher than the first, if the rabbit is not deteriorating clinically, adjust analgesia as necessary and retest the blood glucose in another 10 to 15 minutes.

Good response to treatment will result in the blood glucose levels decreasing and the rabbit choosing to start eating. Offer these rabbits freshly picked grass and dandelion leaves as standard although flat leaf parsley is also a favourite of many rabbits. Once the rabbit is eating well, *I encourage exercise to help with bowel motility*. Close monitoring of urine and faecal output should be continued until normal appetite has returned.

If, despite good quality multimodal analgesia and IV fluids, the blood glucose is not falling or the rabbit is clinically deteriorating then progression to surgery or euthanasia should be discussed.

Surgical intervention for foreign body

Gastrointestinal surgery is more challenging in rabbits, the stomach is never empty of food/contents which increases the risk of contamination, the small intestinal wall is delicate and the caecum contains a large volume of ingesta and bacterial soup. However, it is not impossible and using the same skills as we do in more familiar species then it is a realistic option. In some cases, the blockage can very gently be 'milked' through the digestive tract into the large intestine although I have had the experience of the small intestine rupturing with minimal force. In that specific case I euthanised the rabbit on the table as the entire small intestine was friable so resection and anastomosis was not an option. Alternatively, the foreign body may be milked back into the stomach and either broken down by manipulation through the stomach wall or removed by gastrotomy. The rabbit pictured in Figures 2.38 and 2.39 had the foreign body milked back into the stomach, I then milked the fluid from the stomach into the small intestines by massaging the stomach. Once the pressure was released from the stomach, I could palpate the fur pellet and managed to tease it apart without entry into the stomach. A year has passed since that surgery and at time of writing the rabbit is still doing well (Figures 2.41a & b).

Pancreatitis

Pancreatitis is underdiagnosed in my opinion. This seems to be spontaneous in rabbits and although the literature reports that obesity predisposes, all bar one of the cases

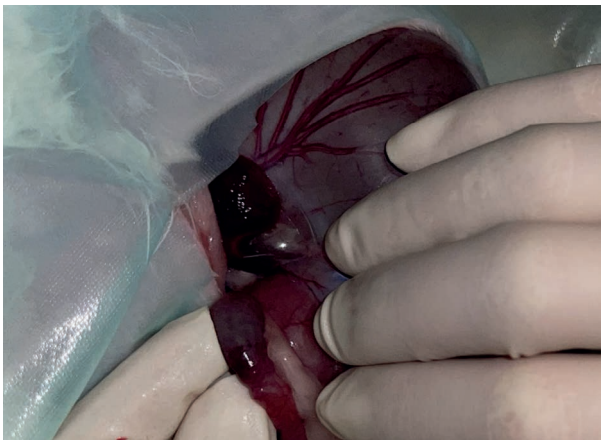


Figure 2.41a The stomach prior to milking the foreign body from the proximal duodenum back into the stomach. Note the distended blood vessels and the colour of the stomach wall.

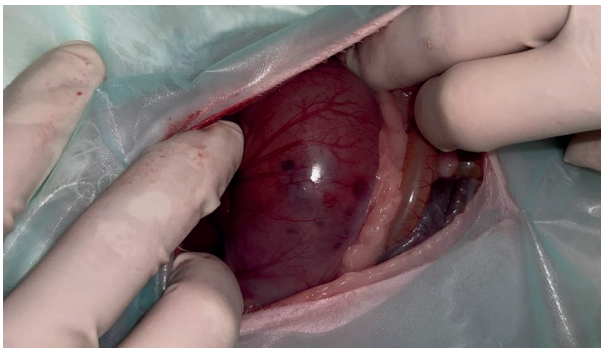


Figure 2.41b After the foreign body was milked into the stomach and the stomach contents flowed into the small intestinal tract. Note the colour of the wall of the stomach, areas of haemorrhage have become apparent from the stretching of the wall, and the return of the gastric blood vessels to a more normal size. The duodenum is visibly full of fluid.

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I have diagnosed have been in rabbits of a normal body condition score.

Clinical signs are nonspecific and are often attributed to gut stasis for another reason: anorexia, tooth grinding (due to pain), abdominal pain (sometimes localised to cranial abdomen), peritonitis, fat necrosis.

Biochemistry will often show an elevated amylase, frequently several times the upper reference range limit (200–500 IU/l). *Unlike more familiar species, amylase in rabbits is highly organ specific to the pancreas*. Blood glucose is also usually elevated due to the severity of the pain and can be monitored using a POC glucometer to gauge response to analgesia.

As with any case of pancreatitis in any species, sudden death can occur. However, I have had good clinical success with hospitalisation, IV fluids, multimodal analgesia (meloxicam, paracetamol, maropitant, and occasionally methadone), famotidine, metoclopramide and assisted feedings (10–20 ml/kg/feed). If I have used methadone, I will usually tailor the frequency of doses to pain scoring and glucometer readings, and many cases will only require the methadone for 24–48 hours. Multimodal analgesia and famotidine should be continued for two weeks to ensure resolution of any secondary gastric/duodenal ulcers or peritonitis. In my experience, rabbits have not had repeated bouts of pancreatitis, but it would be advisable to advise it could be possible.

Stabilisation of the anorexic rabbit

The mainstays of stabilisation are analgesia (previously discussed), IV fluids, assisted feeding, thermal support, and prokinetics.

Dehydration

Hydration status in rabbits can be challenging to ascertain. Skin tent, eye position and consistency of caecal contents (normal will feel like a 'bag of runny porridge') can all be utilised but in my experience are only obvious in severely dehydrated patients, albeit the caecal contents is often the easiest to appreciate. *It should be remembered that the abdominal viscera are thin walled, any anorexic rabbit may have fatty liver syndrome and organs can be ruptured if palpation is too rough*. I advise using fingertip pressure, gradually massaging the abdomen cranial to caudal with a very gradual increase in pressure as needed.

Gut stasis in rabbits is always associated with dehydration. Even though there may not be an obvious loss via diarrhoea, there is often a fluid shift into the gut lumen, resulting in a clinically significant fluid deficit within the tissues without a change in body weight. It is recommended to *assume that any anorexic rabbit is at least 5% dehydrated*.

Oral rehydration with assisted feeding should not be underestimated but given that maintenance is believed to be 100–150 ml/kg/day intravenous fluids should be used in any ill rabbit. *I do not agree with subcutaneous fluid administration*. It is incredibly painful and dehydrated patients will not absorb fluids via this route as efficiently. *Rabbits hide pain so relying on the fact your patient 'allows' you to perform subcutaneous administration is not rationale for it not being painful*. IV access is easy in rabbits (see Venipuncture). Peritoneal injection of fluids is risky due to the risk of inadvertently puncturing the large caecum. If IV access cannot be achieved, then sedation (if not moribund), local anaesthesia and intraosseous (IO) administration of fluids should be considered as in more familiar species.

> Stress (environmental, inappropriate handling or severe pain) has been shown to cause a marked and prolonged reduction in renal blood flow and glomerular filtration rate and oliguria may result. Stress also causes gut stasis and we need to be conscious that acute renal failure can accompany any severe life-threatening disease in rabbits.

Conscious rabbits should not be placed on their backs as this will further stress and immunocompromise an already ill patient. It can be challenging to get some owners to accept that their rabbit does not 'enjoy' being held like a baby. They freeze due to an evolutionary advantage which was meant to confuse predators (there is no evolutionary safe reason you are tipped onto your back) long enough that the rabbit may get that split second to escape.

IV catheters can be connected to giving sets or boluses can be administered every 30–60 minutes. Personally, I often tape giving sets to rabbits' backs (with enough line loose that the rabbit may freely move its ear), which keeps the giving set out of the way (Figure 2.42). I include a loop of the giving set taped to the back which means should the rabbit tug on the giving set, rather than causing the IV line to be dislodged, the loop is pulled straight. If they chew through their second giving set, I will move to boluses and make a note in the patient's file for future reference that this individual should get boluses. *Hydration status should be assessed throughout the day*, which enables you to alter the rate and volume of fluids therapy as required. My intravenous fluid of choice in most cases is saline.

Assisted feeding

The term assisted feeding is preferable to force feeding when discussing with owners. There are several high-quality, purpose made foods available such as: Oxbow Critical Care, Supreme Recovery, Burgess Excel Dual Care or EmerAid Herbivore. All four are high in fibre to promote gut motility, are palatable (although individuals may prefer one over another), and easy to prepare. Feeding syringes with widened nozzles are available from wholesalers made by Supreme Pet Foods and Oxbow.

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Figure 2.42 I often tape giving sets to rabbits' backs with enough line loose that the rabbit may freely move its ear. The rabbit was then transferred back to its kennel.

Some rabbits will require wrapping in a towel (bunny burrito technique) for assisted feeding, with *recommended volumes of 10–20 ml/kg/feed three to four times daily to support gut motility*. Some individuals will tolerate smaller, more frequent feeds better.

Freshly picked grass, hay and the rabbits' normal diet should always be readily available to encourage the rabbit to eat on its own.

Prokinetics/gastric protectants

Once a foreign body has been ruled out, then drugs to help promote normal gastrointestinal motility can be administered. Metoclopramide (1 mg/kg q8–12hrs, may be less effective in young rabbits, increases gastric emptying and small intestinal motility) and cisapride (0.5–1 mg/kg q8–24hrs, increases gastric emptying, small intestinal motility, and large intestinal motility) are the two most commonly used drugs. Metoclopramide can reduce renal blood flow, which may exacerbate any underlying renal issues although in practice I have not experienced an issue.

Cisapride is available from compounding companies such as Summit or Bova (UK) or Doctors Foster and Smith (USA). Domperidone is also used at a dose of 0.5 mg/kg q12hrs. I will use both cisapride and metoclopramide until the rabbit is eating and passing normal faeces.

Stress-induced ulcers seem to be common in rabbits and are frequently seen during post mortem examinations. It can be difficult to know if administration of NSAIDS contributed to the gastric ulcers, however it does not stop me administering NSAIDS if clinically appropriate. I will add in a gut protectant if I am concerned about the presence of ulceration.

Ranitidine (2–5 mg/kg q12hrs) was my drug of choice due to its dual effect of being a prokinetic as well as lower gastric acid production. At time of writing this drug is only available from compounding companies. Other viable options are cimetidine (5–10 mg/kg q6–12hrs) or famotidine (0.5–1 mg/kg q12–24hrs).

Sucralfate 25–50 mg/kg q8–12hrs can be used in suspected cases of gastric or duodenal ulceration. Sucralfate can be sourced from compounding companies such as Summit or Bova (UK) or Doctors Foster and Smith (USA). *Sucralfate requires an acidic environment to work and should be given at least 30 minutes before a H2 blocker*.

Some owners prefer grinding the tablets and mixing with water for administration. Alternatively, the pill can be administered via a pill giver or a 1 ml syringe with the nozzle and top cut off.

Once stable (eating and passing faeces) some owners will decide against further investigation. Depending on your findings during the clinical exam this could be an acceptable approach. For example, if you found spinal or hip pain then continuing on with long term analgesia without radiographs is absolutely fine if the rabbit is clinically stable.

However, *if dental disease is suspected then owners should be counselled this approach would be completely inappropriate*, full investigation is required for prognosis, treatment, and management decisions to be taken.

General anaesthesia

Reducing risks associated with general anaesthesia starts before admission. *With only a few exceptions (foreign body, bloat, or a liver lobe torsion), ill rabbits should be fully stabilised* (rehydrated, passing faeces and starting to eat) before an anaesthetic should be administered.

Rabbits cannot vomit and need a near constant supply of fibre to maintain gut motility therefore they should not be starved before an anaesthetic. Owners are used to starving themselves or their dogs and cats prior to surgery, so we need to be clear when we book rabbits in for surgery that they should not be starved. Advise owners to bring a 'packed lunch' of favourite and familiar foods.

Pre-anaesthetic blood tests

The need for these is debated among vets. Personally, I advise they are an option but acknowledge in the young

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healthy individual the vast majority will be within reference ranges. The benefit is that it will give you a personalised reference range for this individual patient and as rabbits are so good at hiding disease, occasionally we get unexpected results which may alter our plans. Blood can be taken from the marginal ear vein, cephalic vein, or the lateral saphenous vein. The jugular is not commonly used in rabbits due to the short neck and presence of a dewlap in many. There is nothing wrong with aseptically dripping the blood from a needle into the collection tube. Biochemistry can be run 'in-house' using the same pre-op profiles used for dogs and cats (they are mammal profiles) (see Table 2.2 for reference ranges).

Pre-emptive analgesia/prokinetics

Once admitted (and if not already on medications) administer meloxicam 0.6–1 mg/kg and metoclopramide 1 mg/kg. These can either be given orally or by SC injection.

Induction

There are several anaesthetic protocols available for use in rabbits. There is an argument to be made that the safest anaesthetic is the one you are used to. That being said, it has been recognised for at least 20 years, mask induction of rabbits is not recommended and will increase risk of death. This is due to breath holding, severe stress, and sensitisation of the myocardium to catecholamines.

I have used a triple combination of medetomidine, butorphanol and ketamine as my anaesthesia cocktail of choice for twenty years. All I have changed over the years is the dose and route of administration.

I mix 0.1 ml/kg medetomidine (1 mg/ml), 0.05 ml/kg butorphanol (10 mg/ml), and 0.05 ml/kg ketamine (100 mg/ml) in a syringe. I give slowly IV to effect which usually is a third to half of the volume (Table 2.6 for mg/kg dosing). *The triple combination also provides multimodal analgesia*. I do not use methadone as although it is a more potent analgesic than butorphanol, it will only last for 2–4 hours. Buprenorphine will only last up to 6 hours. Butorphanol will last up to 12 hours and therefore may also have an effect on anxiety/stress.

Alfaxan (alfaxalone, Jurox) is another induction agent, is licensed for use in rabbits above 16 weeks of age and for use as an induction agent prior to inhalational anaesthesia. Pre-oxygenation is advised as life-threatening hypoxaemia can occur secondary to apnoea. Intubation should be performed as soon as possible to provide oxygen and intermittent positive pressure ventilation if required. *The analgesic properties of alfaxalone are limited, therefore other means of analgesia will be required*.

The large cornea should be protected by applying an ocular lubricant.

Table 2.6 General anaesthesia dose.

Drug	Dose mixed in syringe IV to effect	Reversal
Medetomidine	0.1 mg/kg	Atipamezole 0.5–1.5 mg/kg
Butorphanol	0.5 mg/kg	
Ketamine	5 mg/kg	

Intubation

Whether I intubate the rabbit is dependent on why I am anaesthetising the rabbit. Personally, I will only intubate for head surgery (not dentals), abdominal or thoracic surgery, or a procedure which will last longer than 15 minutes. I do not routinely intubate for routine castrations.

Although many clinicians perform blind intubation in rabbits, this makes me uncomfortable due to the risks of foreign bodies being pushed into the trachea (Figure 2.43). I prefer to intubate using direct visualisation of the glottis.

With the rabbit in ventral recumbency, an assistant extends the head vertically so that the nose of the rabbit is aimed toward the ceiling and lifts the head. The front legs of the rabbit should be dangling just off the table (Figure 2.44a–c) A long sterile otoscope cone is introduced into the diastema and directed down the throat to the glottis. The amount of pressure required to do this (you need to direct the cone down the oral cavity vertically and tilt the handle so that the cone moves towards you while inside) and how deep the glottis is can be intimidating. The tongue will sometimes go blue due to venous congestion caused by the scope.

The epiglottis will be seen sitting over the glottis and can be disarticulated by touching it with the cone. A local anaesthetic spray such as Intubeaze (20 mg/ml lidocaine hydrochloride monohydrate, Dechra) is applied to the glottis.



Figure 2.43 Blind intubation in rabbits makes me uncomfortable due to the risks of foreign bodies being pushed into the trachea. Note the hay seed situated at the entrance to the trachea of this rabbit, visualised via an otoscope.

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Figure 2.44a The rabbit is held with its head vertically so that the nose of the rabbit is aimed toward the ceiling.



Figure 2.44b A long sterile otoscope cone is introduced into the diastema and directed down the throat to the glottis.



Figure 2.44c An uncuffed ET tube with the connector removed is guided down the cone, into the trachea.

Most rabbits require a size 2.5 uncuffed endotracheal (ET) tube. I will have three sizes available: 2, 2.5, 3. Judge the size of the tube required on the size of the glottis just as we do in dogs and cats.

An uncuffed ET tube (remove the connector first) is guided down the cone, into the trachea. Alternatively, a dog urinary catheter can be used as a guide wire. Place the urinary catheter into the trachea, remove the otoscope and feed the ET tube over the urinary catheter and into the trachea. Reattach the connector. Check for air movement from the tube to ensure correct placement has been achieved. Fix in place with a tie (Video 2.5).

I have a three tries rule. If I cannot get the ET tube placed on the third attempt I stop and use a facial mask, ensuring the nares are not occluded. *Repeated attempts can result in trauma, oedema, and laryngospasm.*

Some practices now have rabbit V gels. These are specially designed tubes which when placed, sit on top of the glottis. It is recommended to use them with capnographs to ensure they have not moved. I do not use them and have heard multiple anecdotal reports of them moving out of place during surgery, however they can be placed quickly which is of benefit in an emergency.

Maintenance

I use a T-piece circuit, with an oxygen flow of 1.5–2 l/min. Some rabbits will not require isoflurane (my gaseous

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anaesthetic of choice) as the induction cocktail may be enough for the procedure. More of the induction triple combo can be administered to deepen the anaesthesia or isoflurane 2–4% as required.

Monitoring

Monitoring the anaesthetic is the same as for more familiar species, except there may still be a faint pedal in the front leg even with surgical depth anaesthesia. *Many texts mention the palpebral reflex remains, however when ketamine is used there will be no blink.*

Multiparameter monitoring is possible. MAI Animal Health make the Vetcorder Portable Monitor, which is suitable for all species, including dogs and cats, for under £1,000. It can record an electrocardiogram (ECG), heart rate, pulse oximetry (placed on tongue or over the central auricular artery of the pinnae, Figures 2.45a & b), and rectal temperature. If recording an ECG, rather than use Alligator clips (which can traumatise the skin), insert small gauge needles through the skin (25 g) and attach the clips to the needles.

If that is outside the practice's budget, a soft, flexible tipped rectal thermometer can be used to monitor rectal temperature. Hypothermia is a major risk factor in anaesthetic deaths. A human foetal Doppler can be purchased online (I used eBay) for around £50, ensure you request the 8 mHz probe rather than the standard 2 mHz one. Respiratory rates can be counted and pulse oximeters can be used from other species but they may struggle to get an accurate reading due to the high heart rate.



Figure 2.45a Placement of the pulse oximeter on the central auricular artery of the pinnae.

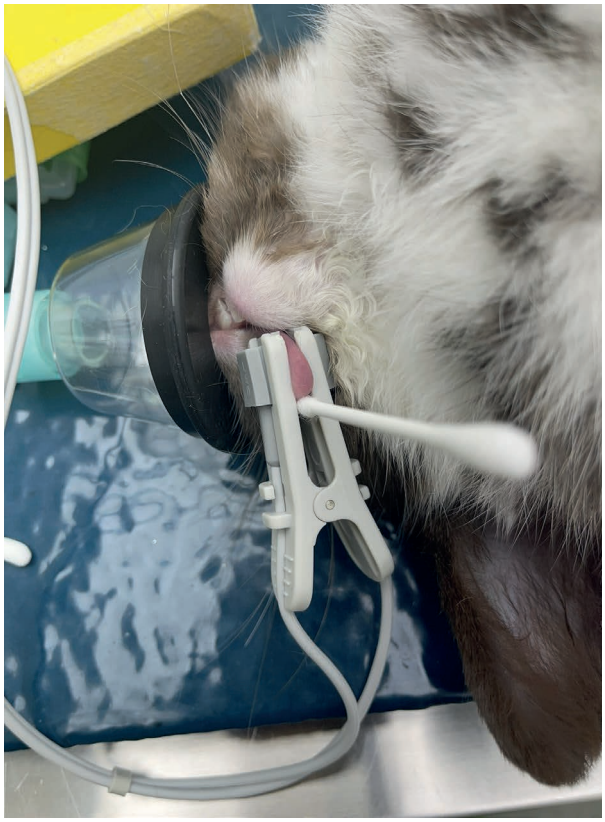


Figure 2.45b Placement of the pulse oximeter on the tongue. The small size of this particular rabbit meant that the spring of the pulse oximeter was strong enough to cause occlusion of the blood vessels which prevented a reading. Simply placing a cotton bud beside the tongue relieved some of the pressure on the tongue allowing a good reading to be achieved.

If using a pulse oximeter please remember that medetomidine can cause peripheral vasoconstriction leading to inaccurate results in some patients, although in my experience if placed on the tongue I get good readings.

Theatre

Rabbits have a high surface area to volume ratio that means they lose heat rapidly. The resulting hypothermia can cause fatalities. *Keeping the ambient temperature in theatre high, wrapping the extremities (including the ears) in bubble wrap, placing a towel on the operating table, and using a plastic drape (also allows visualisation of your patient) are all easy ways to help mitigate heat loss*. Minimise the number of times the door is opened as each time it happens the ambient temperature in the room will drop.

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Positioning

Rabbits are diaphragmatic breathers. Their small thoracic cavity in comparison to their large abdominal cavity can make breathing more difficult due to the weight of the abdominal organs sitting against the diaphragm. *Placing a folded towel under the thorax elevates the thorax and allows the abdominal organs to move away from the diaphragm*.

Surgical preparation

Rabbit skin is delicate and can be traumatised by inappropriate (and sometimes even appropriate) clipper technique. To conserve body temperature, minimise the surgical clip. Only one agent is used for surgical scrub, either chlorhexidine or an iodine-based skin disinfectant depending on the surgeon's preference. We do not use a surgical spirit-based spray as during evaporation this causes loss of heat.

Transparent, plastic surgical drapes are ideal as they allow visualisation of the rabbit while conserving body heat. The required surgical window can be cut into the drape and a surgical spray adhesive is used in lieu of towel clips.

Recovery

Medetomidine can be reversed with 0.1–0.5 ml/kg atipamezole (5 mg/ml) SC, reversing the medetomidine will also reverse the analgesia provided by medetomidine but that is not reason enough for not reversing.

Maintain the rabbit on oxygen, and *if intubated, extubate when the jaw tone increases*. Shivering thermogenesis is a sign of recovery from anaesthesia. Continue to monitor while in an incubator or a warm room (ideally around 26°C (78.8°F)) until up and moving then the temperature of the room can be reduced to 20°C (68°F). Brodbelt *et al.* (2008) found that 64% of the perioperative deaths occurred during the post-operative recovery so close monitoring until the rabbit is ambulatory is ideal.

Syringe feed (10–20 ml/kg Oxbow Recovery, Science Recovery, or EmerAid Herbivore) once the rabbit is fully conscious and monitor faecal output. Advise the owner to monitor food intake and faecal output. If the owner is unable to syringe feed at home, then they must bring the rabbit back to the practice as soon as possible for admission if they will not eat at home. In the vast majority of cases with good analgesia this will not be an issue (Table 2.3).

Dental disease

Our current understanding of dental disease is that it is a *common, multifactorial condition*. Genetics, diet (by inappropriate wear of the teeth, reduced lateral movement and a wider vertical movement leading to abnormal pressures, spikes form in the mouth and over time abnormal pressures result in retrograde unerupted crown growth), and husbandry (lack of UVB exposure leading to osteomalacia and loosening of the teeth) are all implicated in the development of dental disease. Not all owners are aware that rabbits' teeth grow continuously throughout life.



Figure 2.46 The owners presented this rabbit as an emergency thinking it had something stuck in its mouth.

Time taken to educate owners on correct diet and husbandry at their first vaccination visit can help reduce the risk of dental disease. Since I implemented this 15 years ago, I have seen a dramatic decrease in the numbers of dental disease cases I am seeing as a first opinion vet. A diet sheet is given to every new rabbit client.

Careful examination of the incisors to assess: length, shape, occlusal surface, and enamel quality is important. Subtle signs such as horizontal ribbing of the enamel (see Figure 2.33) should increase our index of suspicion for dental issues. *When there is diagonal wear of the incisors, the painful side of the mouth is the side with the longest lower incisor*. Malocclusion can be dramatic and obvious to us but missed by the owners. The rabbit in Figure 2.46 was presented as an emergency because the owner thought something was stuck in the animal's mouth.

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An honest, open, and frank conversation is needed. *Not all owners will be emotionally, financially or time invested enough to proceed with treatment*. Owners need to be prepared to expect repeated dental procedures approximately every 3–6 months, with the second dental procedure occurring in a shorter time frame while the rabbit becomes used to chewing in a more normal pattern. In some cases, euthanasia (which was the outcome for the rabbit in Figure 2.46) is the best option for that individual. *Euthanasia is not a welfare concern (a difficult concept for me to understand as a young vet), however existing in chronic pain and substandard husbandry is a massive welfare issue*.

In the vast majority of cases there is no cure, management of this lifelong condition with repeated dental surgeries, long term analgesia, and husbandry reviews will be required. This requires dedication and financial input from the owner.

Some early cases of dental disease without root changes may be curable after the first dental procedure, with dietary corrections so that they are mainly fed on fresh growing grass, hay, and dried grass. This type of diet encourages prolonged chewing in a natural chewing pattern. The high silicate phytoliths in the grass promotes normal wear of the teeth. Treatment of any underlying health concerns such as osteoarthritis is important, as in adult or aged rabbits presenting with dental disease for the first time, the dental disease is likely secondary to uncontrolled chronic pain (causing a prolonged subtle reduction in appetite).

In my experience most owners are keen to do anything that is required – apart from feed their rabbit's hay! There is a subset of dedicated rabbit owners who are utterly convinced their rabbits are the exception to the rule and do not like hay or grass. Explaining that their rabbit may just have been too painful to eat hay and offer dried grass instead (which is softer) may help. Full dietary review and guiding owners to trusted websites like the ones discussed previously in the diet section can help.

Once the rabbit is stable enough (passing normal faeces but not necessarily eating on its own) I advise general anaesthesia, radiographs of the skull, full exploration of the oral cavity and dental surgery. Radiographing the skull is compulsory if the owner wishes me to perform a dental. Radiographs are required to stage the disease and provide a prognosis.

I radiograph the first time I perform a dental and how frequently I repeat the radiographs depends entirely on the stage of the disease, mild 'root' changes I will repeat the radiograph in 6 months (then in a year if still requiring dental procedures but minimal 'root' changes), severe 'root' changes I recommend repeating the radiographs at 3 months then at 6 months. Ongoing frequency is dependent on if there is progression of 'root' changes and clinical judgement is utilised.

Without knowing the condition, length, and shape of the maxillary and mandibular 'roots' (technically they are actually unerupted crowns) we cannot give information on prognosis or know if long term analgesia is required. *Without this information, rabbits with moderate to severe 'root' disease will be in chronic pain, will not chew properly and will require much more frequent dentals*. With appropriate analgesia, rabbits cope much better with severe 'root' disease than guinea pigs.

Dental radiographs

Once anaesthetised (see section on General anaesthesia) I will take a right lateral and a dorsoventral skull radiograph as routine. *Good positioning is required for the radiographs to be of diagnostic use*. Radiographs should be performed without an endotracheal tube (ET) to ensure we do not miss anything due to superimposition. Rostro caudal skull radiographs are useful for assessing nasal sinus, temporomandibular joint, and the angle of the occlusal plane.

Personally, I do not often take oblique views unless I am worried about dental abscessation and need further assessment. Severe root disease is easy to see (see Figure 2.47) but mild or subtle changes can be harder to appreciate if you do not spend all day looking at rabbit skull radiographs. I utilise three sets of lines drawn onto the radiographs to help with assessing dental disease (Crossley & Böhmer 2009) (Figures 2.48a–c).

Lateral skull radiograph (Figure 2.48a)

Correct positioning is vital, in a true lateral skull radiograph both mandibles are overlying each other as are the tympanic bullae. Rotation of the skull, meaning the bodies of both mandibles are visible is not a lateral view, it is an oblique and the lines cannot be drawn on the radiograph.



Figure 2.47 Severe root disease is readily appreciated. Note the penetration of the palatine bone by the upper incisor root and the mandibular cheek teeth roots that have grown through the mandibular cortex. The increased radiolucency around mandibular roots are suggestive of abscessation.

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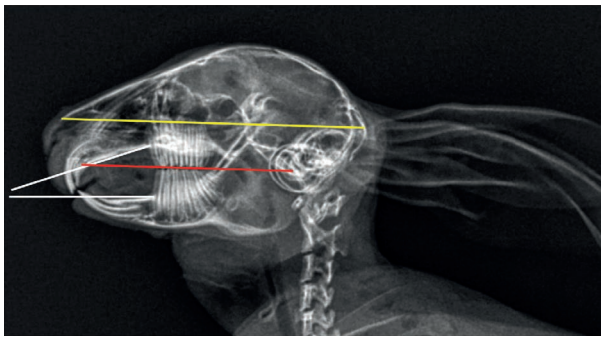


Figure 2.48a Right lateral radiograph of a normal rabbit skull with the objective dental lines drawn.



Figure 2.48b Dorsoventral radiograph of a normal rabbit skull with the objective dental lines drawn.

Two lines (drawn in white) are drawn along the palatine and mandibular bones rostral to the dental arcades. These lines should converge and if the lines are carried forward, we would expect them to cross approximately 2.5–5 cm (1–2 inches) away from the nose. This can vary slightly depending on breed (skull shape does vary) but it is a nice visual indicator of the degree of overgrowth of clinically erupted crowns and is usually the first set of lines I will draw.

A line (in yellow) drawn from the tip of the nasal bone to the occipital protuberance. There should be *minimal to no sign of 'roots' above this line in the normal rabbit*.

Another line (this time in red is drawn) from the alveolar crest at the proximal tip of the hard palate to the height of the ventral third of the tympanic bullae. This should run parallel to the yellow line and run through the occlusal plane in the normal rabbit.



Figure 2.48c Rostro caudal radiograph of a normal rabbit skull.

Dorsoventral skull radiograph (Figure 2.48b)

The first line drawn, connects the lateral margin of the maxillary first incisor's tip with the medial edge of the mandibular ramus on the same side caudally. Another line, which diverges slightly from the previous one, runs from the lateral border of the tympanic bulla to the lateral rim of the contralateral maxillary incisor. *No part of any tooth should be visible outside of these two lines apart from the tips of the apices of the significantly curved maxillary second and third cheek teeth*.

Rostro caudal skull radiograph (Figure 2.48c)

This position gives information on the nasal sinus/turbinates, temporomandibular joint surface (check for dislocation or evidence of degenerative joint disease) and the angle of the teeth occlusal table.

Figures 2.49a–d show the same lines drawn on radiographs of rabbits with dental disease.

Oral examination

Once I have radiographed, I will perform a full oral examination. In the normal rabbit:

- Cheek teeth grow at a rate of 2–4 mm per month compared to the incisors which grow at a rate of 2–4 mm per week.
- Incisors are 3–5 mm long and it can be normal for the lower incisors to be marginally longer. There should be a horizontal occlusal surface. Vertical ridges are normal.
- Each mandibular tooth occludes with two maxillary teeth and works as a functional unit. The mandible is narrower than the maxilla and at rest the cheek teeth

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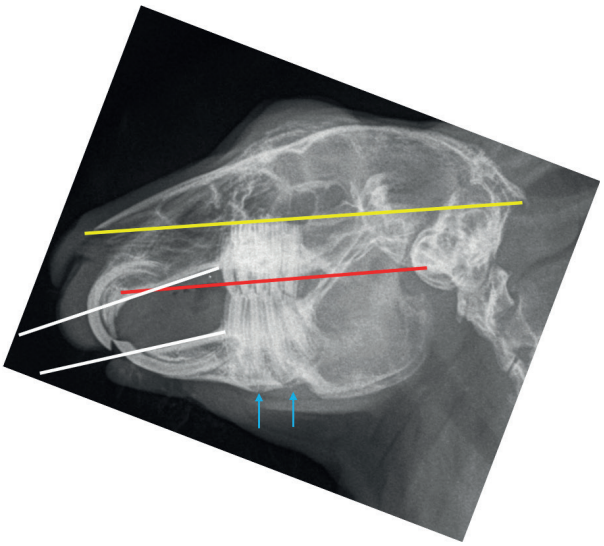


Figure 2.49a Right lateral radiograph of a rabbit skull with dental disease with the objective dental lines drawn. The blue arrows are highlighting the penetration of the mandibular cortex by the 'roots'.

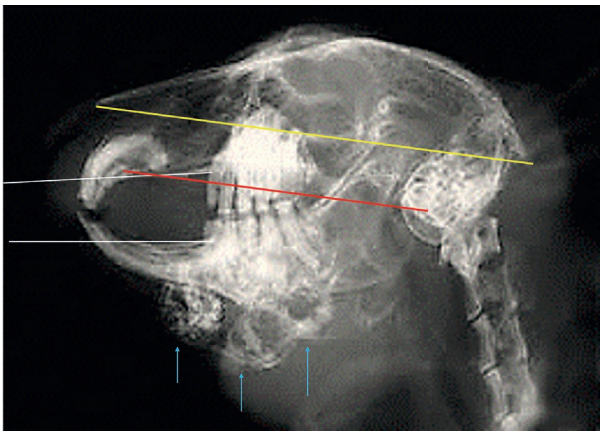


Figure 2.49b Right lateral radiograph of a rabbit skull with severe dental disease with the objective dental lines drawn. The blue arrows are highlighting areas of abscessation that are being walled off.

are not in occlusion. Chewing is performed using lateral movement.

- Both upper arcades (1–3 mm above gingival margin) are shorter than the lower arcades (3–5 mm above gingival margin), the occlusal plane is almost horizontal, the mouth is symmetrical and the teeth are ridged and not smooth. On the lingual aspect of the lower arcades, it is normal to see higher ridges which can be mistaken for spikes. If these are vertical and not flaring lingually I do not worry as long as the rest of the mouth is symmetrical and there is no coronal overgrowth.

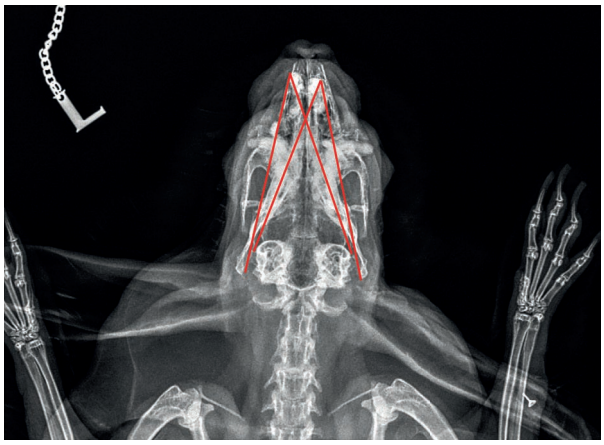


Figure 2.49c Dorsoventral radiograph of a rabbit skull with dental disease with the objective dental lines drawn.

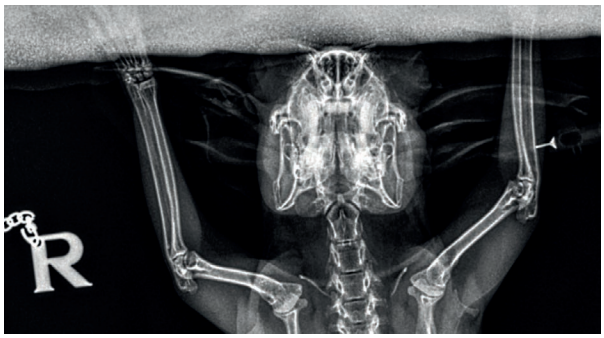


Figure 2.49d Rostro caudal (skyline) radiograph of a rabbit skull with dental disease.

Rabbits with dental disease are likely to have asymmetrical dental arcades. Generally speaking, the upper arcades develop spikes/flare out buccally and damage the buccal mucosa while the lower arcades develop spikes/flare out lingually and damage the tongue (Figures 2.50a–c) Both scenarios are very painful. Imagine if every time you ate, your tongue was scraped to the extent it caused a large wound.

The aim of dental surgery is to restore the correct shape, length, and symmetry of all the dental arcades therefore allowing normal functioning of the teeth.

Incisor malocclusion can be managed with repeated trimming of the teeth (this can be performed conscious in many patients) using a diamond cutting disc (use of guard advised) or a dental drill bit with the water turned off, as rabbits become distressed by the water being sprayed into their face (Figures 2.51a–c).

The normal incisor eruption rate is approximately 3 mm a week but this can increase to 7 mm a week if they are unopposed in malocclusion. Some rabbits will find such

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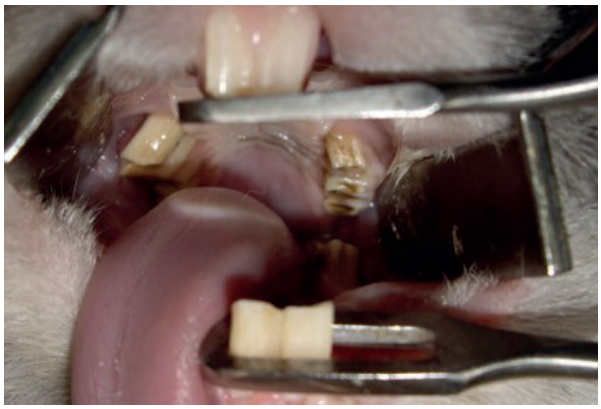


Figure 2.50a Sometimes rather than a spike, the whole tooth overgrows and flares buccally. Note the asymmetry of the upper arcades.



Figure 2.50b Spikes affecting the upper arcades flare buccally and damage the cheek.

frequent trimming stressful, therefore if the incisors are non-functional, extraction is recommended.

> Incisors should never be clipped with molar cutters or nail cutters. This practice is outdated and barbaric. Due to the conformation of the enamel, clipping causes the enamel to shatter leading to vertical fractures, pulpitis and potentially apical abscessation. There is no excuse for clipping incisors.

Dental surgery

Extraction of the incisors

Owners worry that their rabbit cannot eat without their incisors. Counselling owners to consider incisors as scissors, that rabbits have prehensile lips and longer



Figure 2.50c Spikes affecting the lower arcades flare lingually and damage the tongue. Can you imagine how painful this would be if it were your tongue?



Figure 2.51a Incisor malocclusion being managed with repeated trimming of the teeth using a diamond cutting disc.

term cutting of grass and fresh foods into bite sized portions is all that is required. Incisors are also used during the grooming process and so owners will need to brush their rabbits more especially during moult. *If incisor extraction is performed, all the incisors should be extracted.*

RISKS ASSOCIATED

Fracture of the incisor; if this happens continue to remove the rest of the incisors and reschedule removal of the

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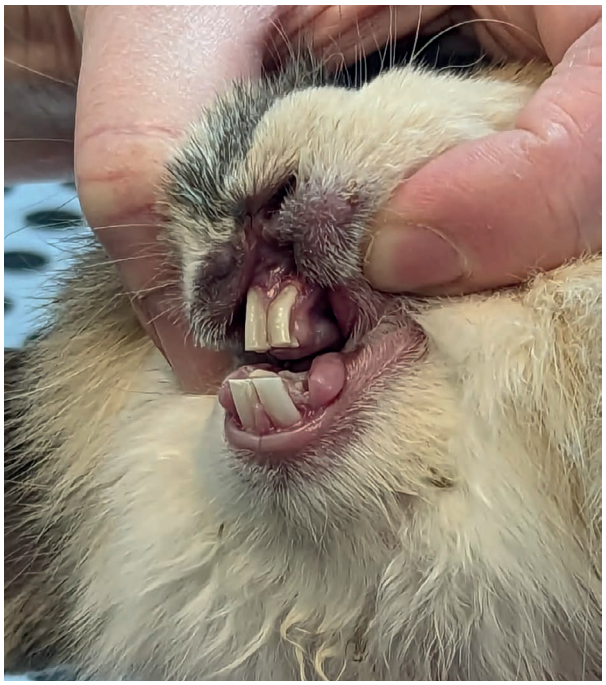


Figure 2.51b The rabbit pictured in 2.51a after the teeth were trimmed. Note the trauma to the soft tissue behind the lower incisors caused by the upper incisors.



Figure 2.51c A different rabbit showing the use of a dental drill to trim incisors with a syringe as a soft tissue guard.

remaining incisor once it has regrown. Post-operative infection in the socket can occur.

EQUIPMENT NEEDED

Crossley Incisor Luxator, size 15 scalpel blade, sterile saline flush, dilute chlorhexidine swab, curved root forceps or curved artery forceps, 1.5 m (4/0) Polyglactin 910 with a swaged-on needle (Vicryl, Ethicon), needle holder.

I prefer to position rabbits in lateral recumbency for this procedure. If the coronal length of the incisors is excessively long, it is advisable to reduce the coronal length (via diamond cutting disc) prior to starting the extraction. Restrain the head in the same grip used for conscious oral exam taking care not to occlude the nares (Figure 2.52). Wipe the gingiva over the incisors with your chlorhexidine swab.

The Crossley Incisor Luxator has two ends: a shorter tighter curved blade and a longer gentler curved blade. These blades match the curve of the incisors: upper incisors = shorter tighter curved blade, lower incisors = longer gentler curved blade (Figures 2.53a–i and Video 2.6 Incisor Removal) Using the wrong blade makes the procedure much more difficult.

The 'root' of the incisor is often three times the length of the clinical crown. It is not 'pretty' surgery, there will be bleeding and occasionally a nose bleed. There will be cracking noises and sensations.

I remove the upper incisors first. Using the 15 blade, incise the periodontal ligament mesial, medial, and lateral to the main upper incisors (Figure 2.53a).

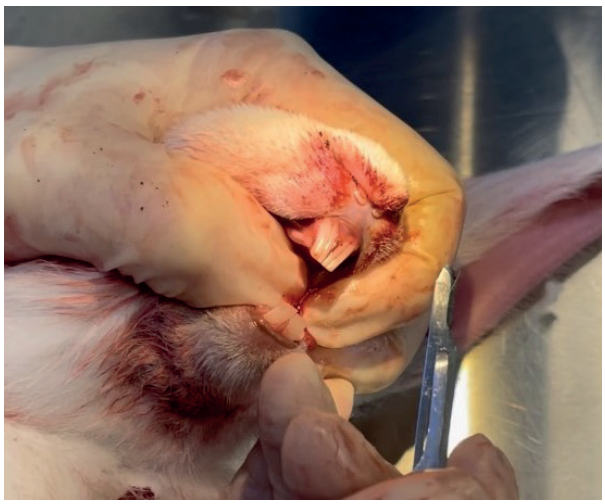


Figure 2.52 Hold the head with your pinkie finger behind the furthest away pinnae. Place your palm flat on the top of the skull with your thumb and forefinger wrapped around the bridge of the nose (taking care not to occlude the nares) and insert them into the mouth to ensure a firm grip. The rabbit is a cadaver which is why there is blood already.

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Figure 2.53a Using a size 15 blade, incise the periodontal ligament mesial, medial, and lateral to the main upper incisors.



Figure 2.53b Holding the luxator in a pen-like grip and using the shorter blade more tightly curved blade, place between the medial aspect of the right upper incisor and the soft tissue which should result in you entering the periodontal space between the tooth and the bone.

Initially hold the luxator in a pen-like grip and using the shorter blade more tightly curved blade, place between the medial aspect of the right upper incisor and the soft tissue which should result in you entering the periodontal space between the tooth and the bone. With the luxator blade touching the tooth on one side, break down the periodontal ligament by pushing forwards and simultaneously using a 'see-saw' motion (Figure 2.53b).

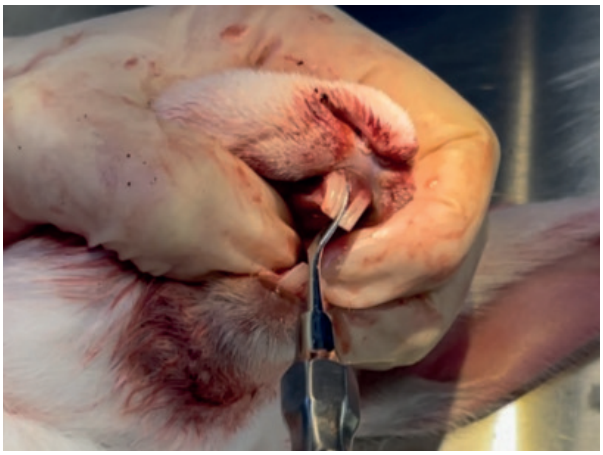


Figure 2.53c Once the luxator blade has slipped into the socket to this amount you can change your grip to over the top.



Figure 2.53d Continue pushing forwards and simultaneously using a 'see-saw' forward motion, and the entire luxator blade will follow the curve of the incisor and 'disappear' into the socket.

Once the luxator blade has slipped into the socket (Figure 2.53c), change your grip to an over the top grip. Continue pushing forwards and simultaneously using a 'see-saw' forward motion, and the entire luxator blade will follow the curve of the incisor and 'disappear' into the socket which is scary the first time we perform this surgery (Figure 2.53d). Gently move the luxator using a laterally side to side motion to help loosen the tooth.

Remove the luxator blade and repeat the procedure on the other side of the same incisor. The lateral side many surgeons find more difficult as the bone is thin and the periodontal space may be hard to enter. Personally, *I find it easier to gain access to the periodontal space by holding the luxator with the blade slightly angled towards the tooth so I*

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Figure 2.53e I find it easier to gain access to the lateral periodontal space by holding the luxator with the blade slightly angled towards the tooth so I am scraping up the side of the tooth into the periodontal space.



Figure 2.53f As the periodontal ligament is present all around the tooth, perform the same procedure over the top of the tooth using the vertical groove in the incisor as a guide.



Figure 2.53g Use the size 15 blade under the tooth and rotate the blade to help loosen the tooth.



Figure 2.53h Repeat steps a to g until the incisor is able to be wiggled back and forth with ease by at least 1–2 cm.



Figure 2.53i Pulling along the curve (if you pull straight out the incisor is liable to fracture) to remove the tooth.

am scraping up the side of the tooth into the periodontal space (Figure 2.53e). As the periodontal ligament is present all around the tooth, perform the same procedure over the top of the tooth using the vertical groove in the incisor as a guide (Figure 2.53f). Use the size 15 blade under the tooth and rotate the blade to help loosen the tooth (Figure 2.53g). Repeat all of the above steps until the incisor is able to be wiggled back and forth with ease by at least 1–2 cm (Figure 2.53h).

Using curved forceps grasp the tooth and push it back and forth into the socket several times. This aims to destroy the apical bud the tooth grows from. Pulling along the curve (if you pull straight out the incisor is liable to fracture) to remove the tooth (Figure 2.53i). Flush the socket with sterile saline to remove any bone fragments or debris. Repeat the procedure on the left upper incisor. Unless the tooth has fractured, close the gingiva over the socket (prevents food or caecotrophs entering) using 1.5 m

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Figure 2.54a Restrain the head for lower incisor extraction by holding the mandible between your forefinger and thumb with the rest of your fingers on top of the head.



Figure 2.54b Using a size 15 blade, incise the periodontal ligament mesial, medial, and lateral to the lower incisors. This rabbit is a cadaver.

(4/0) Polyglactin 910 with a swaged-on needle (Vicryl, Ethicon).

Occasionally the 'peg' teeth are too small to use the Crossley Luxator. In these cases, I will use a hypodermic needle (usually a 23 g) to cut the periodontal ligaments. Bending the needle into a curve will help.

The lower incisors are removed in the same manner except the long blade of the luxator is used (Figures 2.54a–i).

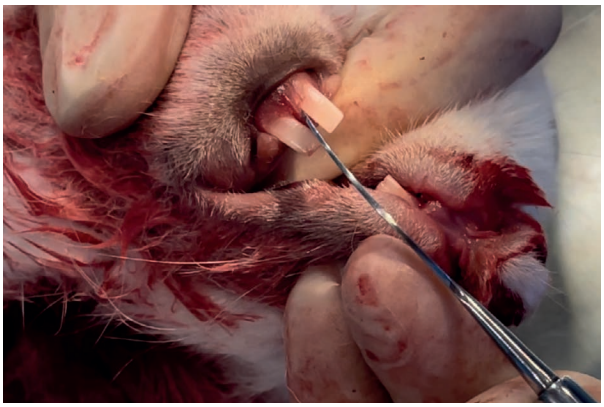


Figure 2.54c Holding the luxator in a pen-like grip and using the longer more gentle curved blade, place between the medial aspect of the lower incisor and the soft tissue which should result in you entering the periodontal space between the tooth and the bone.



Figure 2.54d Once the luxator blade has slipped into the socket to this amount you can change your grip to over the top.

Cheek teeth

RISKS ASSOCIATED

Branches of the facial artery are located in the buccal mucosa, lateral to the last maxillary molars. Trauma to the overlying soft tissue by hand held instruments can lead to death if bleeding is not quickly stopped. In cases of advanced dental disease, elongated maxillary teeth that are loose can damage the palatine artery during luxation. Owners should be counselled that while these complications are rare, a fatal bleed can quickly occur. Collaclot is an avian collagen sponge which will quickly lead to haemostasis. It is inexpensive, has a long shelf and in these situations can be lifesaving.

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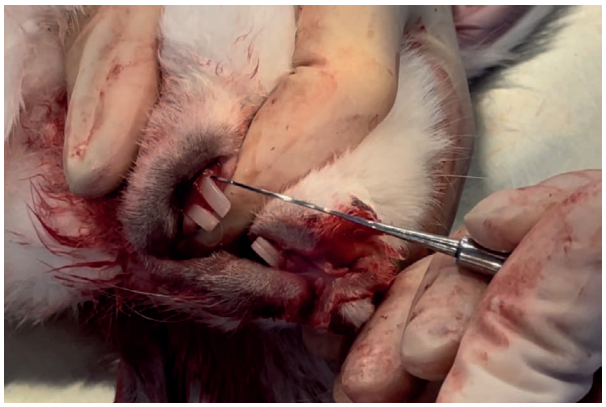


Figure 2.54e I find it easier to gain access to the lateral periodontal space by holding the luxator with the blade slightly angled towards the tooth so I am scraping up the side of the tooth into the periodontal space.



Figure 2.54f As the periodontal ligament is present all around the tooth, perform the same procedure over the top of the tooth.



Figure 2.54g Use the size 15 blade under the tooth and rotate the blade to help loosen the tooth.



Figure 2.54h Repeat steps a to g until the incisor is able to be wiggled back and forth with ease by at least 1–2 cm.



Figure 2.54i Pulling along the curve (if you pull straight out the incisor is liable to fracture) to remove the tooth.

EQUIPMENT NEEDED

Straight dental handpiece with rodent drill bit, diagnostic pen torch with speculum, mouth gag, cheek pouch dilator (or Thudicum nasal speculum), moistened cotton buds.

Owing to the small amount of available space in the oral cavity, I do not intubate for dental procedures. I prefer to position rabbits in dorsal recumbency for this procedure. Positioning like this allows me better access and visualisation of all four arcades. There are many different types of cheek pouch dilators on the market and in my experience, the springs of many of them are too strong, resulting in the instrument 'pinging' out of the mouth or overstretching the cheeks which will be painful. It is important when placing the oral gag over the incisors that it is opened slowly and stop opening as soon as there is any tension felt. *Over opening the mouth will cause damage to the temporomandibular joint.*

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Figure 2.55 The speculum of the pen torch allows illumination right to the tip enabling directed illumination.

I use a diagnostic pen torch with Perspex speculum (Eickemeyer) as both my light source and my speculum/soft tissue guard. The tapered tip of the speculum provides good illumination wherever it is placed, even to the caudal teeth (Figure 2.55).

Use the dental drill with a rodent drill bit and soft tissue guard, held in a pen grip (allows much greater control over the drill), to remove any spurs and to reduce the coronal length (if required) in all arcades. Any crowns that are very loose should be gently pulled out. Remove tooth dust with moistened cotton buds, *ensure both upper arcades are symmetrical and that both lower arcades are symmetrical*.

Hand rasping kits are still available. Only the smoother, diamond rasp should ever be used. Hand rasping takes much longer to perform and there is a higher risk of perforating the arteries. Caution should be exercised as any kind of rasping can potentially lead to damage of the periodontal ligament, loosen the molars, and exacerbate dental disease.

Post-dental care

Assist feed on recovery from anaesthesia. Assisted feeding may be required for a few days, allowing the jaw muscles to readapt to closing the mouth and to allow the occlusal surfaces of the cheek teeth to wear back into function. Monitor faecal output.

Continue analgesia for at least a week after the procedure although cases with 'root' disease will require long term analgesia.

In some cases, the teeth will stop growing. This does not require euthanasia of the rabbit; however, the rabbit will not be able to eat hay. Supplementary feeding with recovery diets or providing softened nuggets longer term is advised. Fresh grass and cucumber 'smoothies' can also help provide fibre (Figure 2.56).



Figure 2.56 Other than her incisors (which are also abnormal looking), this rabbit only has cheek teeth in two arcades and they are very slow growing. She has a good quality of life, receives multimodal analgesia for OA, and is very active and playful. Her owner gives her softened pellets and chopped grass to eat. Her droppings look normal.

Dental abscesses

Periapical infections and dental related abscesses are a common complication of dental disease, which can be frustrating and difficult to treat.

> Rabbits lack the lysosomal enzyme required to digest dead cells which results in their pus being very thick. The abscess itself will often have a dense fibrous capsule with poor blood supply, meaning antibiotics struggle to penetrate the capsule. This is why medical management alone is usually unsuccessful.

Not every facial abscess is related to dental disease, good quality radiographs of the skull and in an ideal world computed tomography (CT) assessment would be utilised to gain the most information regarding the structures involved. Not every owner will be in the position to opt for CT scanning therefore I mainly work on radiographs.

> 'Gold standard' is a term I never use. It can cause shame in owners if they cannot afford to give their beloved pet the 'gold standard' treatment. I use the term 'textbook standard' as it removes the monetary perception. For many people their go-to emotion when they are shamed or fearful (many owners fear our negative perception) is anger. This will increase our stress levels, the owners stress levels and ultimately everyone loses. Small changes in terminology can make a big difference to the entire outcome in many cases.

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The site of the abscess will ultimately determine all the views taken but multiple views and oblique radiograph should be performed. Prognosis is variable depending on location and number of arcades involved. The rabbit should be fully stabilised before surgery.

> To plan a surgical approach, we need to realise that there are three components to the abscess: a capsule, osteomyelitis, and the diseased tissue (dental, bony and soft). All of this can act as a sequestrum and lead to recurrence if any is left behind.

The entire area needs to be opened up, remove all the capsule, use rongeurs to nibble away reactive/infected bone and extract any fragments of teeth. Send some of the capsule for culture and sensitivity as the frank pus is often sterile. Any and all diseased tissue should be removed. Marsupialisation (see section on abscesses) of the area to the skin will allow continued debridement/flushing while it heals. Owners can find this distressing but advising it will increase the chances of success and reassuring them that rabbits heal extremely fast (sometimes too quickly) and usually the large wound will have completely healed within four weeks, will assuage many of their fears.

Medical management post-operatively consists of: multimodal analgesia, twice daily flushing of the wound with saline, using a cotton bud to remove any discharge in the wound, thick scabbing should be removed via soaking and gentle rubbing, topical agents such as: manuka honey, F10 barrier ointment, or Flamazine cream can be packed into the wound, antibiotics should be chosen on basis of culture and sensitivity (but sensible choices include injections of penicillin or a combination of either trimethoprim sulfamethoxazole and metronidazole or enrofloxacin and metronidazole).

Some abscesses will be so extensive they involve an entire arcade and if the mandible is involved, a hemi mandibulectomy may be considered but that is outside the scope of this particular book (and may raise ethical challenges for some owners and vets).

Pasteurellosis

Pasteurella multocida infection is ubiquitous in the pet rabbit trade. Owing to the ubiquitous nature of this bacteria, some authors call into question whether *P. multocida* should be considered a commensal bacterium rather than a primary pathogen.

Regardless whether we consider it a primary pathogen or opportunistic pathogen, *Pasteurella multocida* is implicated in a variety of clinical presentations both acute and chronic in nature.

Many texts historically quote *Pasteurella* as the most common cause of respiratory disease. Although still considered a common cause of respiratory disease it is now acknowledged a wider range of bacterial pathogens can be involved.

Conditions attributed to Pasteurellosis include rhinitis (often atrophic in nature), dacryocystitis, conjunctivitis, otitis media/interna, lower respiratory tract disease (fibrinopurulent pneumonia, pleuritis, pericarditis, pulmonary abscessation), abscessation of any soft tissue, osteomyelitis, reproductive tract disease, or septicaemia.

Diagnosis

Presentation and clinical signs can be suggestive of Pasteurellosis. White blood cell count is often within normal limits (unusual to see results out with normal limits in rabbits), but may display an inflammatory leukogram.

Cytology can be performed in-house, the presence of Gram negative rods and degenerative heterophils further supporting the diagnosis of *Pasteurella* infection. *Owners should be advised that Gram negative bacteria can have unpredictable antibiotic susceptibilities*.

Bacterial culture and sensitivity should be sent before starting any antibiotics although *Pasteurella* can be challenging to grow in the lab, so false negatives may be obtained. Contact your favoured external laboratory for their requirement to perform a *Pasteurella* PCR, however it should be acknowledged that due to the ubiquitous nature of *Pasteurella* in rabbits, *a positive PCR means exposure, not that Pasteurella is the causative agent* of the current condition.

Treatment can be challenging and long. Poor owner compliance can be an issue with owners stopping treatment when the clinical signs resolve rather than continuing the treatment until a negative PCR result is obtained. Hanan *et al.*, 2000 used ciprofloxacin 20 mg/kg orally q24hrs to successfully eliminate *P. multocida* in a group of rabbits with disease associated with *Pasteurella*.

Respiratory disease

Respiratory disease is an extremely common, frequently frustrating collection of conditions to treat. Investigation of respiratory disease in rabbits is very similar to techniques used in more familiar species.

Upper respiratory disease is commonly referred to as 'snuffles' in rabbits regardless of underlying cause. *As rabbits are obligate nasal breathers, upper respiratory tract disease tends to be more stressful/severe than in other species*. It is important to advise owners that in cases of Pasteurellosis we may be looking at control rather than cure as flare ups are common. The normal respiratory rate for rabbits is 30–60 breaths per minute (nose twitches do not correspond with breathing).

Upper respiratory tract disease

The differential diagnosis list for upper respiratory tract disease (URD) is allergy, bacterial (e.g. *P. multocida, B. bronchiseptica*, *Staphylococcus aureus, Moraxella catarrhalis,*

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Pseudomonas spp., among others), dental disease ('root' overgrowth), foreign body, or neoplasia (uncommon).

Common clinical signs include nasal discharge (unilateral or bilateral), sneezing, watery eye/s if nasolacrimal duct (NLD) is involved, reduced appetite, and lethargy in severe cases (rabbit has to choose breathing over eating or play).

Less common clinical signs include pain on facial palpation, pawing at face or epistaxis. *Rabbits are frequently very bright even with moderate discharge and URD may be much more advanced/chronic/serious than the owners appreciate due to the rabbits' frustrating tendency to hide how ill they are*.

During a clinical exam it is important to check if there is any evidence of nasal discharge? As obligate nasal breathers, rabbits fastidiously groom their face and nose, so a minor nasal discharge may not be evident at the nares – check the medial aspect of their front legs by running your thumbs down the inside of the front legs. If there is a nasal discharge, we will often feel crusting on the fur. If there is discharge, what colour is it and is it bilateral? An air flow test can be performed using a wisp of cotton wool just as in more familiar species (see Video 2.7 Air flow test).

Are the incisors normal? Is there a clinical index of suspicion for dental disease?

Thoracic auscultation is performed on both sides and listening to several areas on each side. Take note of any adventitious noises but also of any areas of dullness. *If you are finding it difficult to differentiate between upper respiratory sounds and lower respiratory sounds – listen to the ventral neck.* We do this all the time in brachycephalic dog breeds. Unless there is concurrent lower respiratory disease, the thoracic auscultation is often normal. Check the thoracic compliance is normal. Lack of thoracic compliance indicates that there is pathology in the thorax such as thymoma, abscessation, or dilated cardiomyopathy to name a few and is an indication that diagnostic imaging is required.

Bacterial URD infections can progress into erosive rhinitis and although initially may present unilaterally will often progress onto bilateral involvement. Allergic rhinitis (shavings, hay dust, scented candles, air freshener, perfume are a few common allergens I have come across) may initially present as a clear discharge but progress onto a white pus like discharge with secondary bacterial involvement. Skyline (rostro caudal) view of the skull (I perform by placing the sedated/anaesthetised rabbit in dorsal recumbency and using a sandbag to place behind the skull once the neck is flexed causing the nose to point directly to the ceiling) provides a good view of the nasal turbinates (Figure 2.49c). Good positioning is vital.

Investigation of URD

Pasteurella multocida is ubiquitous within the pet rabbit trade, different strains have differing levels of pathogenicity and can cause a variety of conditions (see above). *B. bronchiseptica* (can cause fatal disease in guinea pigs) and *S. aureus* are both considered normal commensal bacteria in rabbits, but both have also been implicated in cases of respiratory disease which can make bacterial culture a challenge to interpret. It is believed that *B. bronchiseptica* and *S. aureus* can act as opportunistic pathogens in immunocompromised individuals, especially if *P. multocida* has already invaded the tissue. Pure growths of one or two of these bacteria in a culture should be viewed as significant (i.e. they are contributing/causing the disease process) whereas their presence in a very mixed species culture is more suggestive of normal flora.

Radiographs of the skull are strongly advised to assess for any underlying dental disease and assess the nasal cavity of the rabbit. I advise a dorsoventral, lateral, rostrocaudal, and ventrodorsal view to be taken. Check for symmetry, bony destruction, radiopacities (pus is thick), septal deviation – all the same changes we would look for in more familiar species. The only difference is that we also need to check for retrograde 'root' growth into the nasal cavity.

URD may result in otitis media/interna (with or without overt clinical signs) due to migration of bacteria via the eustachian tubes therefore it is important to assess the tympanic bullae. The tympanic bullae should be thin walled, symmetrical, and gas filled (see Figure 2.48b for normal appearance of tympanic bullae). Thickening of the walls of the tympanic bullae and/or increased radiopacity of the bullae are suggestive of an infection (Figure 2.57).

Secondary pneumonia can result due to bacteria translocation via the trachea. While the rabbit is sedated for the radiographs, a deep nasal swab should be taken to send for bacterial culture and sensitivity. Use a small bacterial swab and insert up the nostril (try not to touch the swab to the nares), aiming medially and slightly ventrally. Insert the swab 1 cm (small/brachycephalic rabbits) – 4 cm and gently rotate. See above for interpretation of culture results.

Presence of a foreign body or a large area of radiopacity in the nasal cavity would benefit from flushing the nasal cavity. The rabbit would require intubation to protect the airway before this is performed. Position the rabbit with its head lower than the body (can put the rabbit up on foam blocks), and place a sterile towel below the mouth (to catch any discharge/tissue/object). Using a 5 ml syringe, introduce the syringe nozzle into one nares and flush warmed saline into the nasal cavity. Having the head lower than the body utilises gravity, the fluid should come out the mouth. The trachea is protected by the ET tube. Continue flushing until clear saline only is being obtained. Repeat on the contralateral side. Removing the discharge in this way can be therapeutic in its own right – rabbits eat by sense of smell, therefore flushing the nasal cavity can allow the rabbit to breathe easier and also improve appetite by allowing the sense of smell to improve. If a piece of soft tissue comes out during the flush – send it for histopathology.

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Figure 2.57 On this radiograph there is a subtle but definite generalised increased radiopacity in the left tympanic bullae, the wall is marginally thickened, and there is dental disease with root growth into the nasal sinus. This rabbit was sneezing and had a left sided head tilt suggestive of otitis interna.

Failure to achieve a flush does not necessarily mean a tumour is present, it could indicate that there is inspissated pus that needs surgically removing. *These are the cases that should be using advanced imaging to assess fully with a view to surgical intervention* via a rhinostomy. Unless you are confident with this type of surgery, I would recommend referral.

Treatment and antibiotic therapy for URD

Regular cleaning of their litter tray or latrine corner to stop ammonia build-up is important as ammonia causes paralysis of the cilia in the respiratory tract, preventing mucociliary clearance.

If allergies are suspected then any suitable changes should be made to the home environment, e.g. changing from shavings (especially if scented!) to paper-based bedding, removal of air fresheners. Trial therapy with antihistamines: chlorphenamine maleate 0.2–0.6 mg/kg q12–24hrs, diphenhydramine 2 mg/kg q8–12hrs, or hydroxyzine 2 mg/kg q12hrs, has proved beneficial in many cases.

A recent study (Pellet *et al.*, 2016) looking at the common isolate sensitivities from deep nasal swabs taken in rabbits demonstrated that enrofloxacin had the broadest spectrum with the highest sensitivities to the pathogens isolated in that lab (compared to oxytetracycline, penicillin, trimethoprim sulfamethoxazole).

Rougier *et al.* (2006) found that a high percentage of strains of *Pasteurella* were sensitive to trimethoprim sulfamethoxazole (although marbofloxacin then enrofloxacin had higher levels of efficacy respectively. They also found that many rabbits had concurrent *Bordetella* infections and trimethoprim sulfamethoxazole was more effective than enrofloxacin against *Bordetella* (although marbofloxacin was more effective than either of them).

Owing to fluoroquinolones being important antibiotics both in human and veterinary medicine, an argument can be made for starting mild cases on trimethoprim sulfamethoxazole 20–30 mg/kg q12hrs and using a fluoroquinolone in non-responsive cases.

> We can make an informed choice in severe cases to start the rabbit on either marbofloxacin 5 mg/kg q24 hours or enrofloxacin 10 – 20 mg/kg q12 hours while waiting on our bacterial culture sensitivity results.

Antibiotic courses can vary in length from 4 weeks for mild cases up to 8 weeks or longer for severe or chronic cases. Some owners will find that stressful but with suitable antibiotic choices the risk of dysbiosis is minimal. If an owner is worried, I advise the use of a pre/pro biotic such as Bio Lapis which is rabbit specific. The evidence on efficacy is low but it definitely will not do any harm and may indeed be beneficial.



Figure 2.58a The puncta entrance to the NLD is present in the lower conjunctival fornix. Apply local anaesthetic then use the fur on the lower eyelid to pull the eyelid out which will open the puncta. This rabbit has had fluorescein applied to check for corneal ulceration. The third eyelid is visible and the medial canthus is on the right side of the picture.

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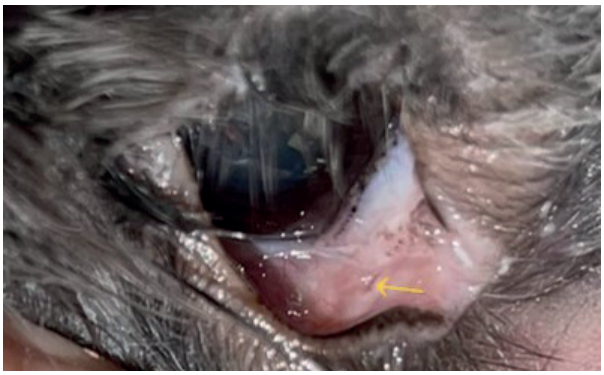


Figure 2.58b In some rabbits, the opening of the puncta will look more like a grey slit than an obvious hole. The yellow arrow highlights the entrance to the puncta in this rabbit.



Figure 2.58c The same rabbit pictured in 2.58b with an IV catheter with the stylet removed being used to cannulate the NLD.

Ciprofloxacin eye drops can be used in cases with conjunctivitis and dacryocystitis. Flushing the NLD can both provide material for culture and sensitivity (before any antibiotics are administered) and is therapeutic allowing entry of the antibiotics (Figures 2.58a–c).

Owners can nebulise their rabbits at home with saline which can make the rabbit feel better by rehydrating the airways. Allowing the owner to nebulise antibiotics at home is a decision for each veterinary surgeon to make. *I am uneasy about potential risks due to owners inadvertently inhaling the nebulised antibiotic*. In the clinic, antibiotics can also be nebulised twice daily for 20–30 minutes. Nebulisation doses have not been evaluated in rabbits, but commonly rodent or bird doses have been used in clinics, see Table 2.7 for doses.

Mucosal linings of the nasal cavity will be swollen, therefore even in cases without erosion they will benefit from meloxicam twice daily.

A mucolytic such as bromhexine 0.5 mg/kg q 12 hrs can help reduce the viscosity of the nasal discharge enabling easier clearance by the rabbit.

Table 2.7 Nebulisation doses.

Drug	Dose (most nebulisers will hold 10 ml)
Saline	10 ml good for hydration of respiratory tract
Acetylcysteine (mucolytic)	10–20 mg per 1 ml saline (higher doses may be associated with bronchospasm)
Bromhexine (mucolytic)	0.15 mg per 1 ml saline
F10	1:250 or ready to use (already prediluted)
Aminophylline	3 mg per 1 ml saline
Amikacin	5 mg per 1 ml saline
Doxycycline	13 mg per 1 ml saline
Enrofloxacin	10 mg per 1 ml saline
Gentamicin	5 mg per 1 ml saline

Ensure that good husbandry is maintained as high levels of ammonia may result in recurrence as opportunistic commensal bacteria just need an immunocompromised host. Advise paper-based bedding and pre-'dunk' hay in water before feeding to reduce any dust.

Reducing stress in the home environment is advisable – check if there are enough hides, separate from predators, add in a Pet Remedy diffuser or a rabbit appeasement pheromone diffuser such as Rabbit Comfort (Beaphar), or a nutraceutical such as zylkene. Personally, I use the following doses for zylkene in rabbits: <1.5 kg, 75 mg; 1.5–3 kg, 225 mg; >3 kg, 450 mg.

Lower respiratory tract disease

Rabbits may seem clinically normal even with advanced disease. One rabbit that was presented for a vaccination, I noted his third eyelids were partially across bilaterally (which was not normal for him even when he was stressed), I questioned his owner who confirmed they had been like that at home for a few days but she had not thought anything of it. On the clinical exam, apart from reduced thoracic compliance and obesity, the rest of the examination was unremarkable. See Figure 2.59 for the lateral radiograph of that rabbit showing the severity of the disease the rabbit was masking.

Owners should be advised that stress could cause a rapid decompensation and even death. Providing oxygen prior to or during clinical exam may be required and for really stressed-out rabbits midazolam should be considered.

The differential diagnosis list for lower respiratory tract disease (LRD) (*which includes diseases affecting the thoracic cavity as they may present as respiratory disease*) is pneumonia, pleuropneumonia, abscessation, pericarditis, pleural

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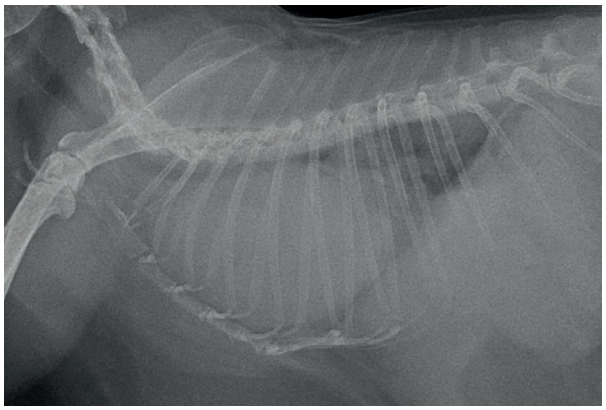


Figure 2.59 This rabbit was clinically well and was presented for a vaccination. The third eyelids were partially across and the rabbit had reduced thoracic compliance but otherwise the rabbit appeared clinically well.

effusion, cardiomyopathy, thymoma, lymphoma, or pulmonary metastatic disease. Common clinical signs include increased expiratory effort, weight loss, depression, exercise intolerance (which may be missed in sedentary rabbits), fatigue.

Auscultation of the lungs should be performed as per more familiar species although using an infant diaphragm will make it much easier. The list of adventitious noises that may be heard are exactly the same as in more familiar species – it's just lungs after all. Of equal importance is lack of sound, i.e. no air movement where air movement would be expected. This would indicate areas of consolidation, e.g. abscessation, tumour or pleural effusion.

A reduction in thoracic compliance immediately alerts us to the presence of a space occupying lesion which could be tumour, abscess, or an enlarged heart (Figure 2.59).

Investigation of LRD

If the rabbit is still passing faeces, then progress to sedation and radiographs. Many rabbits will find it easier to breath once sedated as they are no longer stressed. Careful monitoring is required as breathing may be more difficult in certain positions. *To reduce the risk of artefact from hypostatic congestion we always perform the dorsoventral thoracic radiograph first* before progressing onto right and possibly left lateral radiographs.

Ultrasonography can be utilised to differentiate between soft tissue opacities and fluid. Owners can find the strangest things distressing, so it is important to tell owners that their rabbit will be shaved for ultrasound as many owners will not make the connection between ultrasound and the need for clipping.

Ultrasound guided fine needle aspirates can be useful for differentiating between abscessation and thymoma (see section on thymoma). Drainage of a pleural effusion will immediately provide relief while hopefully allowing a cytological diagnosis.

> Rabbits do not have septa dividing their lungs therefore pneumonia will often have a much more diffuse pattern.

Bronchoalveolar lavage (BAL) can be performed, with 1–2 ml/kg of warm sterile saline being the recommended volume. Intubate with a sterile ET tube, pass a suitably sized (depends on size of ET tube) dog urinary catheter that has been prefilled with sterile saline and already has the syringe attached. This prevents the saline for the BAL being lost in the dead space of the catheter. Instil the saline and then aspirate. Scant saline may be recovered but keeping the syringe attached to the catheter as it is withdrawn, allows the saline that has been aspirated into the catheter to be collected into a sterile tube for submission.

Treatment of LRD

Ultimately the treatment required will be dictated by the actual diagnosis. Pulmonary oedema can be treated using furosemide 2–4 mg/kg q8–12hrs (there are liquid versions available, e.g. Frusol).

Antibiotic choice should be guided by culture and sensitivity. Suitable choices to initiate therapy while waiting for the results include: trimethoprim sulfamethoxazole, doxycycline, enrofloxacin, or ciprofloxacin. I continue antibiotics for 2 weeks after resolution of clinical signs to try and reduce the risk of relapse. Owners should be prepared that antibiotic courses can be three months long. See Table 2.8 for antibiotics commonly used in rabbits.

Meloxicam twice daily is recommended, and nebulisation can benefit by delivering drugs locally, rehydrating the respiratory tract and may produce a 'feel good' sensation as a result.

> Reduction of stress in the home environment will be beneficial.

Nebulisation therapy

Mesh nebulisers are much quieter, produce less vibration, and so are less stressful for the rabbit. Thankfully they can now be bought inexpensively online. Droplet size produced should be less than 0.5 μm. Mucolytics and antibiotics can be delivered using nebulisation. F10 disinfectant (bactericidal, virucidal, fungicidal) can safely be nebulised using a 1:250 dilution of the concentrated product, for 30 minutes twice daily. Mild cases of respiratory disease may be successfully managed with the use of F10 alone.

Videos can be found on YouTube to direct owners on how to set up their own nebulisation cage at home.

Mucolytics can be nebulised enabling the rabbits to clear any discharge more efficiently, which can result in

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Table 2.8 Commonly used antibiotics in rabbits.

Drug	Dose
Amoxicillin	*Do not give orally*
	7 mg/kg SC q24hrs
Azithromycin	15–30 mg/kg PO q24hrs
Ciprofloxacin	20 mg/kg PO q12–24hrs
	1 drop topical q8–12hrs (ocular application good for nasal pasteurellosis)
Chloramphenicol	30–50 mg/kg SC, IM, IV, PO q8–24hrs
Doxycycline	2.5 mg/kg PO q12hrs
Enrofloxacin	10–30mg/kg PO q12hrs
	(IM painful and may result in sterile abscess/muscle necrosis)
Marbofloxacin	5–10 mg/kg PO q12–24hrs
Metronidazole	20–30 mg/kg PO q12–24hrs
Oxytetracycline	15 mg/kg SC q24hrs, 30 mg/kg SC q72hrs
Penicillin G Procaine form	*Do not give orally*
	40,000 U/kg SC q7days for 3 treatments for syphilis
	42,000–84,000 U/kg SC, IM q24hrs
Silver sulfadiazine cream	Topical q12–24hrs
Trimethoprim sulfamethoxazole	20–30 mg/kg PO q12hrs

an increase of sneezing or coughing in the short term. Counselling owners that the rabbits may develop a productive sneeze or cough when given mucolytics will manage their expectations and stress levels.

Ophthalmology

The approach to the ocular exam is the same as with more familiar species. The notable differences in rabbits are more laterally situated eyes, a low blink rate (average one blink in ten minutes), no sclera should be visible (if you can see the sclera that indicates exophthalmos), the insertion of the dorsal rectus muscle is clearly visible through the conjunctiva of the dorsal globe, normal tears can have a slightly milk appearance due to increased lipid (Figure 2.60), dacryocystitis is much more common, ocular pain may be hard to appreciate (increased blink rate, epiphora, squinting are all indicators), there is no menace response.

Fluorescein application should show at the nose within 2–5 minutes (Figure 2.61). Failure to appear or if the dye runs down the side of the face should increase suspicion of a blocked NLD.



Figure 2.60 Rabbit tears can have a high lipid content. These are normal tears in this rabbit.



Figure 2.61 Fluorescein application should show at the nose within 2–5 minutes.

Conjunctivitis

Primary causes include poor husbandry (ammonia, irritants) or allergies such as wood shavings. Secondary causes include pasteurellosis, dacryocystitis, entropion, and conjunctival foreign body.

Once bacterial conjunctivitis is established; a profuse white, thick ocular discharge is produced. Inflammation

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and discharge can mask the underlying cause. Washing of the eyes (especially if it is a lone rabbit) twice daily and topical treatment such as fusidic acid (Isathal, Dechra is licenced in the UK for rabbits).

Dacryocystitis

Dacryocystitis is extremely common. Narrowing, partial or complete blockage of the NLD results in pooling of tears in the lacrimal sac. Failure to drain down to the nose results in spillage of tears and ocular discharge down the side of the face. Very quickly this can result in hardening of the secretions onto the fur leading to clumping, concretion, facial maceration, and facial pain.

Clinical signs are epiphora, conjunctivitis, variable purulent discharge, purulent material expressed when massage lacrimal sac (distal to medial canthus), scabs/hard dried discharge on facial fur which is painful to the touch. Fluorescein application will run down the side of the face. Check for corneal ulceration.

Acute dacryocystitis can occur due to a foreign body or blockage of the NLD by ocular discharge. Acute problems carry a good prognosis following flushing of the NLD and treatment of the underlying condition.

Chronic dacryocystitis is due to dental disease (retrograde growth of the first upper premolar or upper incisor impinges on the NLD), or chronic infection of the NLD leading to scarring and narrowing of the NLD. Stenosis of the lacrimal puncta can occur in severe cases (Figure 2.62).

Computed tomography is the best way to assess the NLD, skull and dental anatomy. However, in many cases this may not be a viable option for owners.

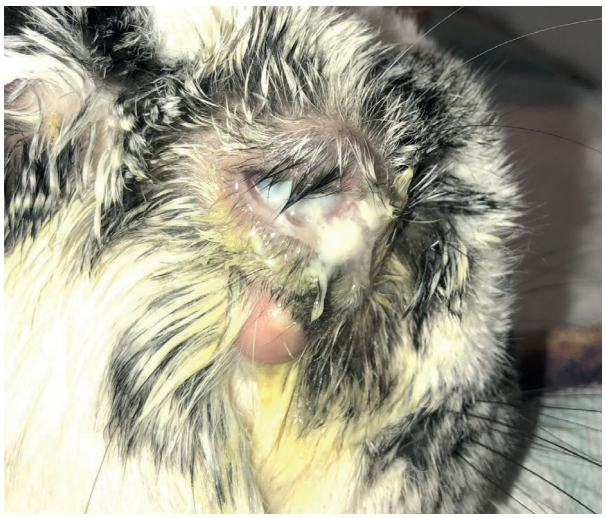


Figure 2.62 Chronic dacryocystitis and abscessation of the NLD. Note the squinting of the eye due to pain. This rabbit had surgical debridement of the abscess and the condition fully resolved with treatment.

Positive contrast radiography can be used to highlight the NLD. Flush the NLD with sterile saline, instil 0.5–1 ml of iodine-based contrast medium. *There is a natural stenosis of the NLD at the base of the upper incisors, dilatation of the duct proximal to the stenosis is not always associated with dental disease*. Assessment of the dentition should also be performed as discussed in the dentistry section.

Personally, I rarely do these contrast studies. Repeated or severe cases should have dental radiography.

Flushing the NLD

In the vast majority of rabbits this can easily be performed when conscious. Equipment required: proxymetacaine, 2 ml syringe, 22 g IV catheter (with the stylet removed), cotton buds, sterile saline, head torch, towel.

Burrito the rabbit and apply proxymetacaine to the affected eyes. Once the proxymetacaine has taken effect, wash the eye with sterile saline to remove as much discharge as possible. I find sitting down while performing this procedure easier. The head torch allows illumination of the lacrimal puncta. The lacrimal puncta are seen as a slightly darker slit/oval in the lower conjunctival fornix near the medial canthus (Figure 2.58b).

Although lacrimal catheters are available, I find them too pliable which makes entry to the puncta more challenging. Using your non-dominant hand, pull the lower eyelid out by the fur and visualise the entrance to the NLD, use cotton buds to wipe away any discharge and slip the 22 g IV catheter into the puncta (Figure 2.58c).

Using a 2 ml syringe (larger volumes increase risk of rupturing the NLD) flush the duct. Initially this may be difficult and discharge may first be flushed back into the eye. Repeated attempts may be required before clots of pus and saline will be seen at the nose. I continue flushing until I get clear saline running through.

Ideally a swab would be submitted for culture and sensitivity. Ciprofloxacin eye drops (Ciloxan, Novartis Pharmaceuticals UK Ltd) are my treatment of choice as they have good activity against *Pasteurella* unless otherwise indicated by sensitivity study. *The facial pain caused by the burning of the skin in reaction to the dried discharge should not be underestimated. Multimodal analgesia is an important part of the therapy*. Syringing saline onto the hardened areas of fur will help soften the areas allowing gentle cleaning of the face. Some cases will require sedation to perform cleaning due to the severe pain associated.

Repeat flushing of the NLD weekly until healed. Owners should be counselled that in chronic cases it is control rather than cure. *Lone rabbits (with chronic dacryocystitis) should have their lacrimal sacs massaged daily and eyes washed daily even when without clinical signs*. This is due to the fact that bonded rabbits spend considerable time grooming each other's eyes. This must help maintain healthy NLD as it is very common, when one of the bonded pair dies,

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for the remaining rabbit to present with ocular discharge. The bond had prevented the owner ever being aware of the eye issue due to mutual grooming of eyes.

Ulcers

Corneal ulcers are most likely under diagnosed in rabbits. Unlike dogs, they are unlikely to rub at a painful eye. A slight squinting of the eye, increased tear production and an increased blink rate may be the only clinical signs associated with an ulcer. It is understandable why this is easily missed by both the owners and the veterinary team.

Corneal oedema associated with ulceration is variable and, in my experience, it can be subtle or absent in many cases. Neovascularisation is also highly variable between individuals. Fluorescein application will readily stain the ulcer (Figure 2.63).

Regardless of species, I advise owners that corneal ulcers do one of three things: heal within a week, take months to heal or lead to the loss of the eye. Managing the owners' expectations right at the start reduces stress for all involved.

Topical treatment as per more familiar species (chlortetracycline hydrochloride, chloramphenicol or ciprofloxacin plus an ocular lubricant) and analgesia should be given.

Non-healing/Indolent ulcers

Underrunning of the cornea can be appreciated by a brighter green halo around the central staining area of the ulcer after fluorescein application. It can take five minutes or more for the stain to spread under the non-adherent cornea (Figure 2.64a).

Proxymetacaine should be applied to the eye and cotton bud debridement can be performed, using the same level of pressure we use to perform debridement in a dog's eye,



Figure 2.63 Fluorescein application stained the ulcer, note the fur loss on the side of the face due to a blocked NLD causing the epiphora to flow down the side of the face. This had hardened and caused the fur to fall out when cleaned off.



Figure 2.64a The brighter green halo around the central staining area of the ulcer after fluorescein application indicates underrunning of the corneal ulcer.



Figure 2.64b The same ulcer post-debridement showing the true extent of the ulcer.

5–10 minutes later (Figure 2.64b for the same ulcer post-debridement). Multiple debridement's may be required and the eye should be checked for presence of a foreign body or ectopic cilia which could be continuing to damage the cornea. Occasionally a grid keratotomy will be required but it should be remembered that rabbits have a thinner cornea than dogs so care should be taken. Topical treatment, regular lubricant, and analgesia (multimodal analgesia may be required) should be continued. Serum eye drops can be made using the rabbit's own blood in the same way we prepare them for dogs. Schedule weekly rechecks, or more frequently if required.

Overgrowth of conjunctiva

Circumferential overgrowth of the conjunctiva is occasionally seen (Figure 2.65). Most cases do not require treatment but surgical resection can be performed if it causes an issue. Owners should be warned that the conjunctiva may regrow over the eye. The aetiology is not understood.

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Figure 2.65 Circumferential overgrowth of the conjunctiva is occasionally seen.

Exophthalmos or enlarged globe?

> The most common question I get asked is: is it retrobulbar or glaucoma? Easiest way to differentiate is to look at the eye. If the third eyelid is across the eye (that is, prolapsed) then the problem must be behind the eye, not in the eye.

Prolapse of the third eyelid

Prolapse of the third eyelid is associated with increased retrobulbar volume.

Many rabbits become very stressed when in the practice leading to vascular engorgement of the large venous sinus behind the eye causing bilateral exophthalmos. *Ask the owner if the rabbit looks like this at home*, if when relaxed at home the rabbit has the same appearance of the third eyelid, then check for a lack of thoracic compliance and advise radiographs. Increased intrathoracic pressure from the thymoma or cardiomegaly will also result in vascular engorgement of the large venous sinus behind the eye causing exophthalmos, *therefore prolapse of the third eyelid may be the first clinical sign of a very serious life-threatening problem*. If the exophthalmos is due to venous engorgement retropulsion of the globe should be possible and non-painful.

Other differentials for exophthalmos include retrobulbar abscess or tumour. Most of these will present unilaterally (there is a potential connection between the two orbital spaces in rabbits therefore it is possible conditions can become bilateral, in practice it is not common). *As abscessation happens gradually it is common for the retropulsion attempt to be non-painful, however this cannot be used to differentiate between abscess or tumour.*

Owing to the presence of a space occupying lesion the globe will not easily retropulse, if at all. The presence of dental disease increases the index of suspicion for abscessation. The sclera will become visible which can be used to locate the area of the lesion (*sclera is visible in same area the space occupying lesion is situated*).

A condition known as idiopathic retrobulbar adenitis is occasionally seen. It causes a non-painful, bilateral exophthalmos due to inflammation of retrobulbar glandular tissue of unknown aetiology. Ultrasonography will show multilobulated retrobulbar masses and should respond to meloxicam and broad-spectrum antibiotics for a seven-day course.

Glaucoma

Glaucoma can occur as a primary or secondary (to uveitis or trauma) condition. Globe enlargement (buphthalmos) occurs readily in the rabbit and circumferential scleral show only occurs in severe cases (Figure 2.66a). *The third eyelid will be in a normal position, i.e. it will not be prolapsed.*

Clinical signs are often subtle in early stages and include signs of ocular pain (epiphora, increased blink rate), buphthalmia, corneal edema, mydriasis, scleral injection, optic nerve head cupping, and vision loss.

Vision assessment

Vision assessment can be tricky in rabbits. Menace response is absent in rabbits therefore of no use in assessing if the eye is visual. Direct pupillary light response is present but is much slower to occur and with a weaker response when compared to the dog (Warnefors *et al.*, 2019). Consensual pupillary light response is rarely observed in rabbits which is unsurprising given that only 10% of the optic fibres actually cross over in rabbits (Giolli & Guthrie, 1969).

Intraocular pressure (IOP)

Reference ranges for IOP in rabbits have been reported, a mean IOP of 9.51 ± 2.62 mm Hg using a rebound tonometer and 15.44 ± 2.16 mm Hg using an applanation tonometer (Pereira *et al.*, 2011). The same paper also reported that IOP was higher when measured early in the day which may suggest monitoring of cases should be performed at the same time of day to ensure accurate conclusions are drawn.

Using the Tonovet Icare rebound tonometer, measurement should be taken from the centre of the cornea 4–8 mm from the surface as per the manufacturer's guidelines as even within the 4–8 mm significant differences in IOP measurement will occur (Rodrigues *et al.*, 2021) (Figure 2.66b). IOP can return to normal as the globe enlarges, making measurement of the IOP as a method of diagnosis more challenging. A retrospective study showed a median IOP at diagnosis was 39 mmHg (26–55 mmHg) (Yuschenkoff *et al.*, 2020).

Secondary lens luxation is common even in eyes that remain visual. Severe corneal oedema and episclerotic congestion is less common in the rabbit (Figure 2.66c). Corneal ulceration may occur secondary to the buphthalmos.

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Figure 2.66a Glaucoma in a rabbit that is prepared for enucleation. Note the normal position of the third eyelid. The sclera was only visible on the dorsal aspect of the globe (the photo was taken from the dorsal aspect which gives the impression the eye is rotated ventrally) and was less obvious when the rabbit was conscious and in ventral recumbency.



Figure 2.66b Measurement of the IOP should be taken from the centre of the cornea. I find it easier to steady the top arm of the Tonometer against my finger as seen here.

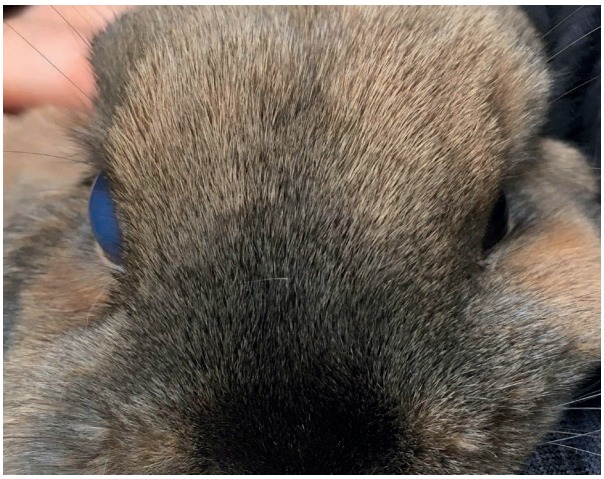


Figure 2.66c This rabbit had glaucoma, note the enlarged globe, the third eyelid is not visible, and this rabbit had diffuse corneal oedema which is uncommon in rabbits with glaucoma.

Treatment

Medical treatment may control the condition for years: timolol, dorzolamide or a combination of both (combination may be synergistic). A combination drop can be administered q8hrs, monitor for side effects such as bradycardia or ileus (due to electrolyte imbalances) and adjust dose as needed.

Some rabbits will never respond to medical management. They are so good at hiding pain that the owners may not realise the eye is blind and painful. End stage glaucoma will necessitate enucleation.

Cataracts

Cataracts are commonly seen in rabbits and can be a result of: congenital, age, lens trauma, or *E. cuniculi*. Referral for removal of the lens by phacoemulsification is a viable option to save vision, but in my experience many rabbits seem unbothered by them. Gradual loss of sight affects them less than sudden loss of sight (Figure 2.67a).

Acute uveitis

Clinically uveitis is the same as in more familiar species: aqueous flare, hypopyon, hyphema, iridial hyperaemia and miosis. Pasteurellosis can be the underlying cause, but any systemic bacterial, septicaemic, or thromboembolic disease should be considered.

Treatment

Systemic antibiotics and topical steroids will help control symptoms while the underlying cause is identified and treated. Atropine may be required to maintain a dilated pupil. Efficacy of atropine will vary between rabbits: in albino rabbits atropine lasts 96 hours, coloured iris rabbits atropine lasts 8–10 days, however some individual rabbits

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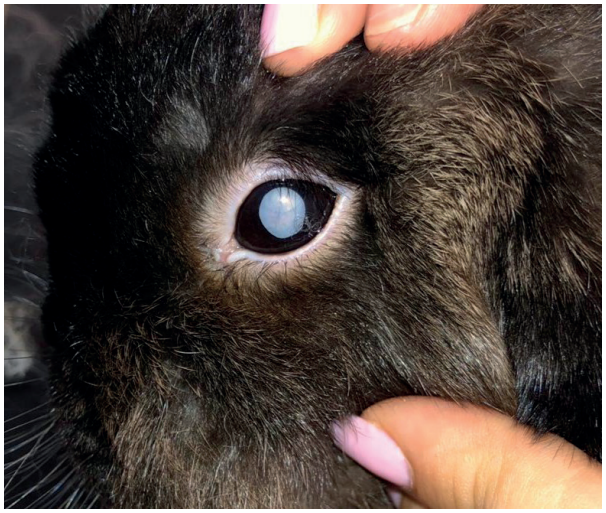


Figure 2.67a Cataract in a rabbit, this rabbit was not stressed or seemingly affected by the cataract. Gradual loss of sight affects them less than sudden loss of sight.

have the enzyme atropine esterase which reduces the activity of atropine to 12 hours. Atropine application may be required up to q6hrs to maintain a dilated pupil *but at this frequency paralytic ileus as a side effect becomes a very real risk*. Personally, I have used atropine q12hrs. Topical steroids will be systemically absorbed and may have an immunosuppressive effect, monitoring of the haematology is advisable.

Phacoclastic uveitis

Lens rupture in the rabbits causes dramatic formation of white iris abscesses (Figure 2.67b). These abscesses can grow aggressively leading to secondary glaucoma. *E. cuniculi* lens infection (in utero infection) seems to be the most common cause although sessile cataract and spontaneous rupture is recognised with young dwarf breeds being predisposed to spontaneous rupture.

Occasionally only small focal lens ruptures occur with localised granulomatous response in the overlying iris. Topical steroids may be useful to control focal lesions. *Even topical use of steroids can be absorbed systemically and may lead to immunosuppression causing for example a* pasteurella *flare up. If steroids are used it is recommended to monitor haematology*.

Treatment

Treatment of choice in order to preserve vision is referral for removal of lens by phacoemulsification early in the course of the disease. If *E. cuniculi* infection is suspect (or the organism is detected in lens tissue post-phacoemulsification), systemic treatment with fenbendazole 20 mg/kg q24hrs for 28 days is recommended due to the high likelihood of central nervous tissue lesions. *There is a risk of bone marrow dysplasia with fenbendazole so consider monitoring haematology during and after treatment*.



Figure 2.67b Phacoclastic uveitis due to ocular *E. cuniculi*.

Enucleation

In cases of retrobulbar disease or a blind painful eye, enucleation should be considered. Personally, I struggle with the ethics of bilateral enucleation in a prey species, however colleagues who have performed bilateral enucleation report seemingly good quality of life, especially if part of a bonded, indoor housed group. Further ethical debate is outwith the scope of this book and is the remit of each individual vet as to where their ethical boundaries lay.

Transconjunctival technique

Rabbits have a large venous sinus behind the globe which can lead to a fatal bleed if uncontrolled. A transconjunctival approach is recommended, this helps with easier access to tying off the optic nerve and venous sinus and also ensures maximum amount of soft tissue left behind, enabling closure in several layers creating back pressure. A more cosmetically appealing finish is achieved as the socket is full of tissue meaning that once it is healed there is much less of a sunken eye socket. The transconjunctival approach is now my favoured approach in all species requiring enucleation.

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Transconjunctival approach means cutting through the conjunctiva circumferentially, working as close to the sclera as possible (Figures 2.68a–f). Transect the four muscular attachments to the globe. The globe will now be loose in the socket with only the optic nerve holding it in place. Place curved haemostats behind the globe and clamp the nerve. Ligate with 2/0 (3 m) Vicryl (or similar), remove globe. Occasionally I find it easier to remove the globe without removing the haemostat, then ligate the nerve and tissue. There should be no bleeding, if there is bleeding, use a Collaclot Haemostat sponge (Cambcol). Collaclot is an avian collagen sponge that will stop life-threatening bleeds. Even upon removal of the sponge (which is not necessary), and flushing of the site there should be no rebleed due to the unique haemostatic mode of action.

Remove the third eyelid and the very large Harderian gland associated with it. The gland is approximately two inches in length, knobbly in appearance and has a pale pink and a creamy white portion. There can sometimes be bleeding associated with this – which may be due to puncturing the venous sinus. Either place a Collaclot sponge or control with digital pressure using a swab then using 1.5 m (4/0)

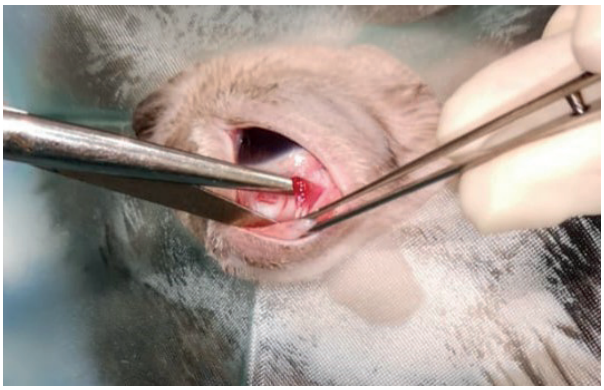


Figure 2.68a Using scissors, cut through the conjunctiva circumferentially, working as close to the sclera as possible.

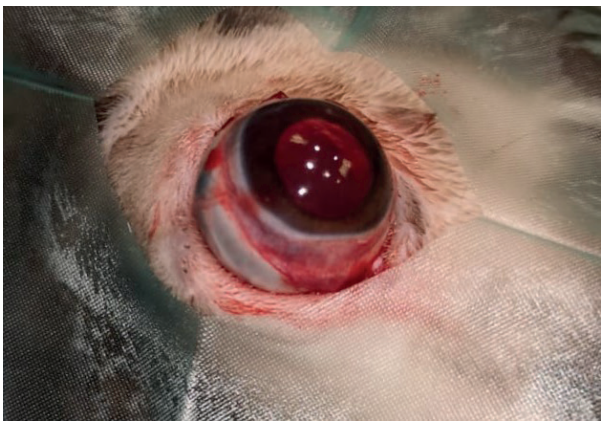


Figure 2.68b The conjunctiva has been cut through and the ocular muscles are now visible in each quadrant.

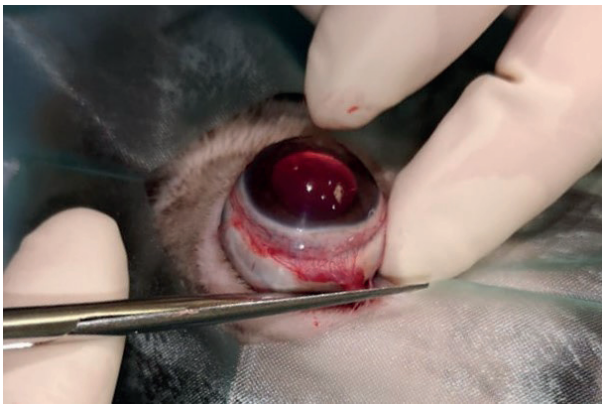


Figure 2.68c The ocular muscles are transected using scissors.

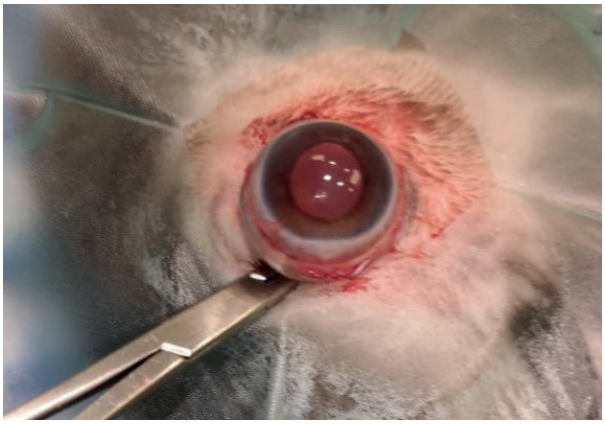


Figure 2.68d Place curved haemostats behind the globe and clamp the nerve and ligate.

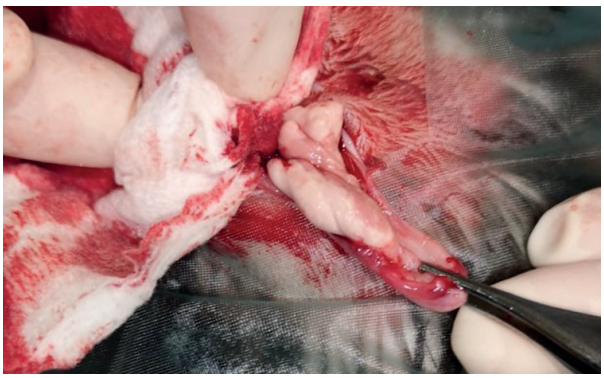


Figure 2.68e Remove the third eyelid and the very large Harderian gland associated with it. The Harderian gland is knobbly in appearance and has a pale pink and a creamy white portion. The swab is over the area the medial canthus would be.

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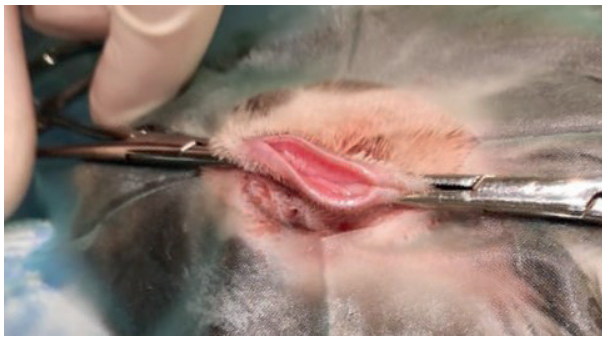


Figure 2.68f Remove the eyelids last and repair using intradermal suture pattern. I use haemostats to make removal of the eyelids easier.

Polyglactin 910 with a swaged-on needle (Vicryl, Ethicon) place simple continuous row in the deep socket tissues to provide back pressure. This will not be required if Collaclot is used but is good practice to ensure a more cosmetic finish. Place at least two simple continuous rows in the tissue to close the soft tissues of the socket. Remove the eyelids last and repair using an intradermal suture pattern. Postoperative analgesia should be continued for at least 1 week. Possible complications include chronic bouts of fluid build-up if part of the Harderian gland has been left behind. Repeat surgery can be performed if it is bothering the rabbit or the owner, but in my experience, using the transconjunctival approach, leaving part of the gland behind is very unlikely.

In cases of retrobulbar abscessation or retrobulbar tumours, exenteration rather than enucleation is required. This carries an increased risk of haemorrhage and diagnostic imaging to check for bone involvement should be performed before surgery.

Ear disease

Ear mites

Psoroptes cuniculi infestation is very painful for rabbits. Severe infestations can result in transfer of the mites to the perineal area during grooming (Figure 2.69). Ivermectin drops applied every 2 weeks for three treatments or a single selamectin 15–25 mg/kg treatment will kill the mites and any that hatch. *The crusting that forms inside the pinnae should not be manually removed no matter how tempting as to do so would be extremely painful.* Analgesia should be given for one week. Any secondary infection can be treated topically with a solution of 1/3 silver sulfadiazine (Flamazine, Smith Nephew) mixed with 2/3 of saline. Owners can instil 2 ml of the mixture into the ear canal daily.

Ear-based abscess/aural diverticulosis

Rabbits evolved having upright ears which are easily movable to allow for better hearing, regulating body temperature, and signalling. Nature did not design lop



Figure 2.69 The Psoroptes cuniculi infestation was so severe in this rabbit that the mites were transferred to the groin during grooming.

eared rabbits. Lop eared rabbits (dwarf lop refers to length of the ears not the size of the rabbit!) have a much narrower ear canal than nature intended, due to the softened cartilage folding over to cause the desired visual appearance of floppy ears, so much so that often the normal wax cannot migrate out and can build up causing stretching of the ear canal which is painful.

The intense pressure caused by the build up causes damage to the delicate skin lining the canal, ulceration, inflammation, and infection can cause pus to form. Rabbits lack the enzyme that makes liquid pus causing their pus to have the consistency of thick cream cheese. The pressure may be enough to rupture the eardrum and cause an inner ear infection. This results in a 'pocket' of tissue forming between the distal end of the cartilage and the start of the bony canal. *Many texts refer to the swellings as ear-based abscesses but aural diverticulosis is a more accurate term.*

Sometimes these swellings are bilateral but, in my experience, they are asymmetrical in size. Even when quite large they are easy to miss if not actively sought for. Often, they are tender to palpate and contain wax or a mixture of wax and pus (Figure 2.70a). Imagine how painful it must be to produce wax that builds up to an extent it actually stretches your ear canal and gives you an ear infection? Have you ever had a ruptured eardrum? If so then you have an idea of how very painful it is, yet rabbits will pretend they are absolutely fine and suffer in silence.

Visual examination of the external ear canal is often unrewarding until gentle, massaging pressure is put on the swelling. This usually results in the normal wax and discharge moving up the ear canal with a toothpaste-like consistency. The external ear canal can develop kissing ulcers which is *very painful*. Analgesia should be given.

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Figure 2.70a The swelling at the ear base is often referred to as an abscess but aural diverticulosis is a more accurate term.

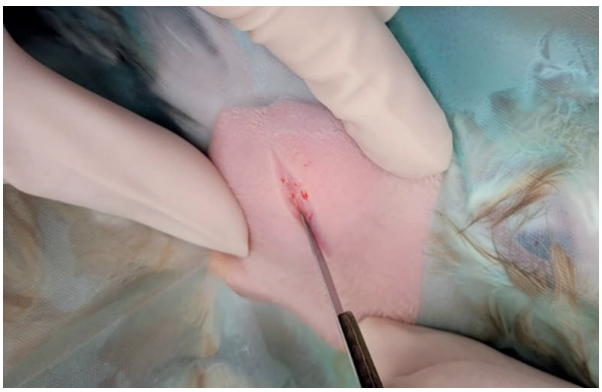


Figure 2.70b Perform an incision in the skin over the pocket.

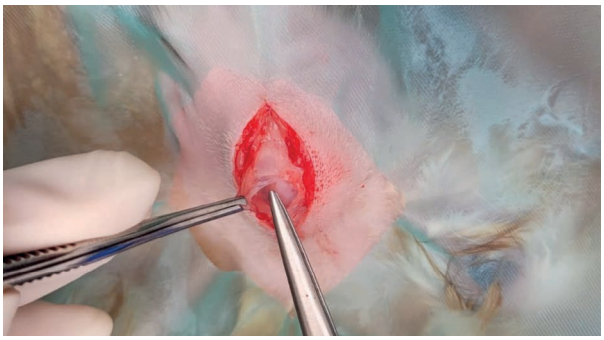


Figure 2.70c Blunt dissect through the soft tissue until reaching the ear canal which has been stretched by the swelling.



Figure 2.70d Incise into the pocket.



Figure 2.70e Remove any discharge. I use sterile cotton buds and forceps.

Frustratingly, it is possible for rabbits to have severe inner ear infections and have no obvious clinical signs. Other signs related to inner ear infections are facial asymmetry (known as a facial grimace, due to irritation of the facial nerve, see Figure 2.70), nystagmus, or a head tilt.

Medical management

If there are no other clinical signs associated (e.g. head tilt or facial grimace), medical management using saline or cat ear cleaner (cats are sensitive to smells therefore their ear cleaners are usually tolerated better by rabbits compared to dog ear cleaners) to flush the canal while massaging the pocket daily and a silver sulfadiazine ear flush can be used to treat any kissing ulcers and otitis externa twice daily. *Ear preparations that contain steroids should be avoided*. There is a risk that the pressure caused by the build of wax and discharge will cause perforation of the tympanic membrane and middle ear involvement. *Medical management alone will not always result in good control as fundamentally this is a*

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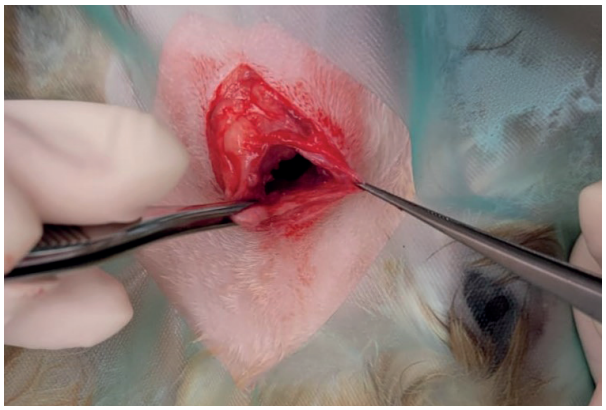


Figure 2.70f Evert the pocket.

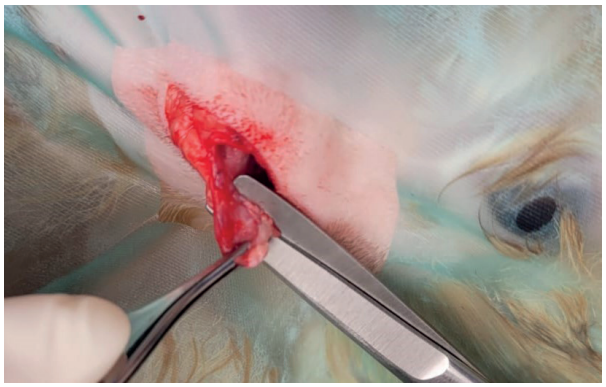


Figure 2.70g Remove any excess soft tissue.

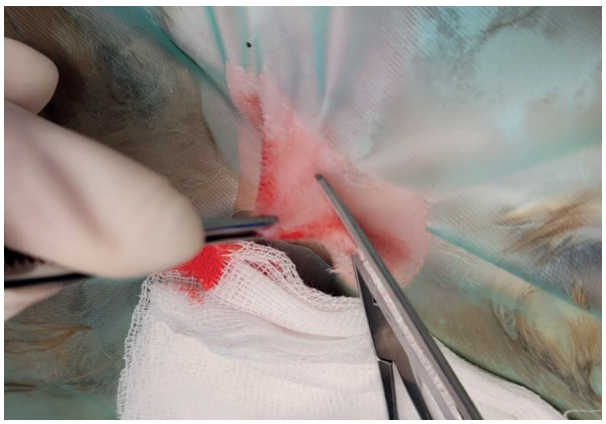


Figure 2.70h Remove the portion of skin.

conformational issue. Otitis externa is rare in prick ear rabbits but when present *Psoroptes cuniculi* or a foreign body, should be sought.

Surgery

In cases where medical management is unsuccessful or either the rabbit or the owner finds the procedure too stressful, surgical intervention should be considered. Simple cases without middle/inner ear involvement often respond well to partial lateral wall ablations. Effectively a permanent stoma is surgically created using the stretched soft tissue of the 'pocket' at the base of the pinnae. The size of the pocket will determine the amount of tissue that can be removed.

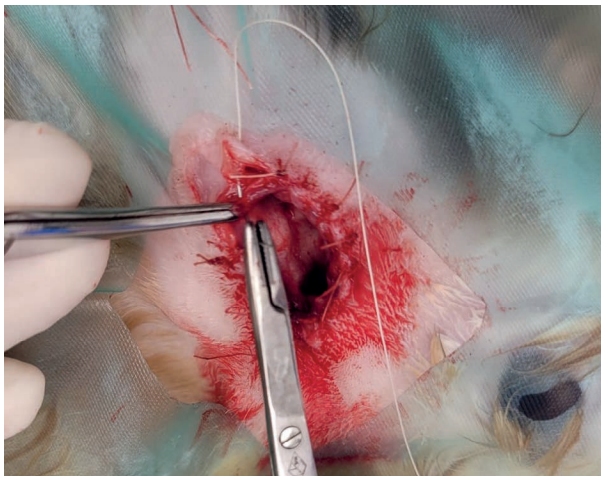


Figure 2.70i Stitch the internal ear canal to the skin.

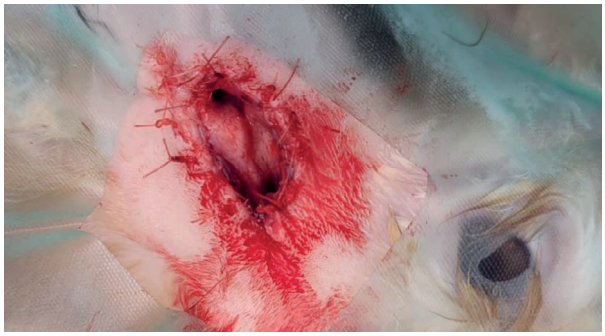


Figure 2.70j The finished stoma.

Essentially: perform an incision in the skin over the pocket. Incise into the pocket and remove any discharge. Evert the pocket, remove any excess soft tissue (some rabbits require removal of part of the cartilage if there is not a lot of stretched ear canal to work with), remove the portion of skin, and stitch the internal ear canal to the skin (Figures 2.70b–k) *Make the stoma as large as possible, rabbits heal quickly and wound contracture can result in the stoma almost closing fully*. Once healed any discharge or wax should easily come through the stoma. Antibiotics will frequently be needed but ideally these should be based on culture and sensitivity. The bacteria within rabbit pus are often dead, so a swab is best taken from the wall of the infected ear canal during surgery or a small sample of tissue can be sent to the lab for culture.

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Figure 2.70k The same stoma healed. Note how much smaller the hole is than the initial wound.

Head tilt

A head tilt is one of the most common neurological presentations in rabbits and yet still fills many of us with dread. As with more common species, it is highly suggestive of vestibular disease. History taking should include questions about exposure to new rabbits, any stressful incidents, appetite, faecal output, water intake, speed of onset, head shaking, ear scratching, rolling/spinning, weight loss, any treatment the owner has given, age of house if the rabbit is a house rabbit. Owners should be advised that even with diagnosis, treatment and recovery, a residual head tilt is not uncommon.

'Normal' scanning behaviour

Scanning behaviour is usually seen when the rabbit is at rest and to me it looks like the rabbit is vacantly staring into space as its head moves ('scans') one side to another. Sometimes there is a fast and slow phase, sometimes both are the same. It is important not to be caught out by this strange but completely normal behaviour. It is common in red eyed white (REW) rabbits but can be seen in any rabbit, and may be mistaken for head-nystagmus by the uninitiated, and the rabbit will not have nystagmus. When questioned most owners will report that this is completely normal for their individual rabbit.

Head nystagmus

Although visually it may resemble scanning behaviour, it is of a sudden onset. This will be very obvious if the rabbit does not normally exhibit scanning behaviour but even in rabbits who do, owners may notice a difference and it is unusual for rabbits to 'scan' on a consulting room table although it may be noted in hospitalised rabbits when they are resting (see Video 2.8). The fast and slow phases match phases of the ocular nystagmus. Look closely at the rabbits face for asymmetry, presence of a facial grimace helps locate the pathology as a peripheral vestibular issue.

Differential diagnosis and clinical exam

The most common differentials to consider in a rabbit are otitis media/interna and *E. cuniculi*. Other differentials may include encephalitis or abscess, heat stroke, head trauma, or neoplasia. Some people find it easier to think in terms of peripheral vestibular syndrome (otitis media/interna, traumatic rupture tympanic bullae, aminoglycoside ototoxicity, middle/inner ear neoplasia, idiopathic vestibular disease) versus central vestibular syndrome.

- Infectious:
  - Fungal: *E. cuniculi*
  - Bacterial: *P. multocida*, Staph, *E. coli*, Pseudomonas, Listeria
  - Protozoal: Toxoplasma gondii (uncommon)
  - Viral: Human Herpes Virus 1 (rare), Rabies (not in UK)
  - Parasitic: *Baylisascaris procyonis* (not in UK)
- Non-infectious: lead toxicity (chewing old painted skirting boards), cerebral infarcts, hepatic encephalopathy, lymphosarcoma).

Differentiating between them can be challenging. *As vets we often forget that a thorough clinical exam (including a full skeletal pain examination) is one of the best diagnostic tests you can perform!*

Assessing: mentation, appetite, proprioception, nystagmus (is it positional?), head tilt, ataxia, and the cranial nerves can help with locating the lesion. Check the ability of the rabbit to blink with damp cotton bud as facial nerve damage may lead to an inability to blink and exposure keratitis. Watching how the rabbit moves or asking the owner if they have any video footage of the rabbit moving at home is helpful, is the rabbit circling for example or suddenly hugging the wall when moving around the room (which suggests a loss of balance)? Is there any sign of a purulent nasal discharge? *Presence of a nasal discharge is indicative of upper respiratory infection and increases the likelihood of bacterial otitis media/interna.*

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The cause of any paresis, plegia, or ataxia depends on which limbs are affected: focal spinal disease, disease along cerebral-brainstem-spinal axis. Impairment of voluntary motor control of the limbs localised to the spine: spinal infarct, vertebral scoliosis, vertebral fracture. Subtle clinical signs such as facial grimace (will only happen with peripheral vestibular disease), or bald area on the dorsum of the pinna on the same side as the head tilt, or head shaking may suggest otitis, whereas reduced mentation and/or quadriparesis is suggestive of a central problem.

Treatment of head tilt rabbits (regardless of cause)

Much of the treatment required is the same regardless of cause, additional treatment will be needed depending on the definitive diagnosis.

Rabbits that are unable to eat need assisted feeding and IV fluids. Prochlorperazine maleate (used to treat nausea and vertigo in humans and marketed as Stemetil in the UK) is useful to include in the treatment protocol to treat suspected nausea/dizziness at a dose of 0.2–0.5 mg/kg q8hrs. Meclizine hydrochloride is an antihistamine that is commonly used in the USA to treat suspected nausea/dizziness at a dose of 12.5–25 mg/kg q12hrs.

Monitor the eyes for corneal oedema/ulcers as the lower eye is more prone to trauma and rabbits will not frequently squint with corneal ulcers the way dogs generally do. Using ocular lubricants may be beneficial. Padding the cages can help prevent injury if rabbits are rolling. Inserting a rolled up towel into a pillow case in a U shape will give some support to a rolling rabbit. Midazolam or diazepam may be beneficial in rabbits with severe vestibular signs as stress will definitely make the rolling worse. Ensure recumbent rabbits are given deep, soft bedding to spread weight and are regularly turned to prevent decubital ulcers which are painful and difficult to treat.

Multimodal analgesia should be used: NSAID, paracetamol, maropitant, tramadol, gabapentin (especially if neuritis) can all be utilised. Acupuncture can be used as part of a multimodal approach.

Therapeutic exercise, supervised on a non-slip area for 10–30 minutes several times a day has been shown to be extremely important to improvement of clinical disease and earlier return to more normal function. Supporting the rabbit in a normal standing position and passive physiotherapy therapy of the limbs as well as allowing hopping.

Showing owners how to massage the neck muscles to help relieve the muscle spasms caused by the head tilt, also gives the owner something else to do and makes them feel more involved.

Famotidine or omeprazole can be used if there are concerns about stress induced gastric ulcers.

Frank conversation

Sometimes, no matter what the diagnosis is and no matter how hard you try, not every owner will be able to emotionally or financially cope with the care, not every case will respond (poor mentation often will have poorer outlook) and we should be striving for quality not quantity of life. It is important to have end of life care conversations especially if treatment is not causing an improvement within two weeks.

Encephalitozoon cuniculi

Encephalitozoon cuniculi is a microsporidia, which research suggests is closely related to fungi. It is considered zoonotic and is recognised in several species including immunocompromised humans. Three different strains are recognised. Strain 1 affects rabbits and humans, strain 2 affects rodents, and strain 3 affects dogs and humans. There is a direct life cycle both horizontal and vertical.

The infective spores are spread in urine, which remain viable in the environment for an average of four weeks. The spores are sensitive to many types of disinfection *but the contact time required to kill them is highly variable*. A 0.1% bleach solution required a ten-minute contact time compared to the thirty second contact time required by 70% ethanol. This can have potentially serious implications in the clinic e.g. *where ethanol should be used for cleaning the consult table if a rabbit has passed urine on it during the clinical exam to prevent potential exposure of other patients to infective spores*.

Clinical signs

Exposure is widespread (52–83.6%) and the vast majority of rabbits will have no clinical signs, providing that parasite and host immune response are in balance. *Onset of clinical signs may be seen a few days after a stressful event (e.g. change to environment, new rabbit, loss of cage mate, predator exposure). One study showed 60% of rabbits developed clinical signs associated with* E. cuniculi *72 hours after a stressful event*. The predilection sites are the lens of the eye (in utero infection), brain, spine, lung, myocardium, and kidneys. Rupture of cells releases many spores into the surrounding tissues which results in a granulomatous inflammatory reaction. Therefore, *the clinical signs associated with E. cuniculi are more attributable to the inflammatory response by the host* rather than the presence of the organism itself.

Clinical signs vary widely depending on which predilection site the inflammatory response is in. Seroconversion occurs approximately 3 weeks post-exposure and is detectable before any histopathological changes are apparent. Therefore, a *negative IgG IgM titre test will rule out* E. cuniculi *as the cause of current neurological clinical signs in an immunocompetent rabbit*.

Clinical signs can include any combination of head tilt, torticollis, hindlimb paresis, paralysis, urinary incontinence, renal failure (rare), cataracts (with or without

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lens induced uveitis), retarded growth, lethargy, weight loss, collapse, tremors, convulsions, and death. Vestibular dysfunction due to *E. cuniculi* usually does not affect the rabbit's appetite.

Diagnosis

Despite *E. cuniculi* first being detected as an infectious agent in rabbits in 1922, over 100 years later definitive diagnosis antemortem remains challenging especially as the rabbits immunological competence determines the course of the disease. All immune competent rabbits exposed develop antibodies that may last for the life of the animal but do not confer protection. Severity of inflammation seen histologically does not mirror the severity of the clinical signs and the level of antibody titres does not mirror level of infection.

Exposure is so widespread that a single titre test is not useful. Even a negative IgG titre in a healthy rabbit could mean either the rabbit has not been exposed, or has only recently been exposed and will not show a titre for another 2 weeks. A positive IgG does not mean *E. cuniculi* is the cause of disease.

Demonstrating that IgM and IgG titre levels are rising is much more indicative of an active infection therefore it is recommended to measure IgM and IgG levels 2–3 weeks apart. This holds true for immunocompetent rabbits only. Immunocompromised rabbits may not mount an antibody response. Which is another reason why it is important to rule out other differential diagnoses before diagnosing *E. cuniculi* as the cause of clinical signs.

If a rabbit has a positive IgM, owners should be advised their rabbit may shed spores in the urine for up to 18 weeks. A urine PCR for spores is available but as spores are shed intermittently a pooled sample is advised (3 days). False negatives are possible as the rabbit may not be shedding spores. A positive result definitely confirms an active infection (but not that this is the cause of the neurological disease).

Presumptive diagnosis of *E. cuniculi* is based on having the relevant clinical signs, exclusion of other underlying causes, elevated IgM and IgG, and/or positive PCR urine. Definitive diagnosis relies on histopathological examination of tissue and is usually only performed on postmortem, although theoretically this could be performed on a renal biopsy.

Treatment

There have been no controlled studies on treatment of chronic *E. cuniculi*.

Treatment of presumptively diagnosed cases involves supportive care (varies depending on clinical signs, see treatment section under head tilt) and a 28-day course of fenbendazole 20 mg/kg q24hrs. Fenbendazole is not a benign drug and should only be used if we have ruled out other differential diagnosis or ruled in active *E. cuniculi* infection e.g. positive urine PCR, rising titres. Monitoring complete blood counts during treatment (weekly) is advisable.

Fenbendazole toxicity

Fenbendazole is not a benign drug and adverse reactions such as crypt necrosis of the small intestine and bone marrow hypoplasia/aplasia have been reported in several species including rabbits.

Graham *et al.* (2014) reviewed 13 cases of benzimidazole toxicosis in rabbits. They suggested that concurrent liver disease may increase risk of toxicity and possible drug-induced intestinal inflammation or necrosis allows greater absorption of the drug, contributing to toxicosis. Common clinical signs of toxicity included sudden lethargy, inappetence, GI stasis, petechiation, pale mucous membranes, haemorrhage, elevated body temperature, and sudden, unexpected death. The prognosis for rabbits with suspected benzimidazole toxicosis is poor as in only one case did the rabbit survive despite intensive treatment.

I have not seen a case of bone marrow dysplasia attributable to fenbendazole in a rabbit but I have seen several cases in birds and reptiles. This may be due to my tendency only to prescribe fenbendazole in presumptively diagnosed cases of *E. cuniculi* for many years rather than prescribing in every case of head tilt.

Otitis media/interna

Even in prick ear rabbits the tympanic membrane can be a challenge to visualise without the aid of an endoscope. The facial nerve runs very near to the tympanic bullae and the exact position varies with individuals (either ventrally or superficially). Proprioceptive deficits would not be expected in cases of otitis media/interna. Many rabbits with clinical signs will have both otitis media and interna.

One study demonstrated that up to 27% of rabbits with otitis media have absolutely no clinical signs (seen on CT as an incidental finding) so the condition is most likely underdiagnosed. Up to 80% of rabbits with upper respiratory tract infections will also have otitis media due to migration of bacteria up to eustachian tubes.

Clinical signs

Clinical signs associated with otitis media/interna can include asymptomatic, weight loss, persistent or recurrent gastrointestinal hypomotility, reduced appetite due to pain in the temporomandibular joint, facial neuritis, vestibular disease, facial grimace, or ipsilateral facial nerve paralysis.

Facial nerve paralysis initially looks like one side of the face is swollen due to the drooping of the upper lip, but over time the facial muscles atrophy and contract leading to twisting of the face towards the affected side. Conversely,

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inner ear disease can result in facial nerve irritation initially leading to a facial grimace, i.e. the lip is held higher on the affected side and may become paralysed leading to a droop in later cases (Figures 2.32a & b).

Diagnosis

Although advanced imaging such as CT scans will be much more sensitive, it may be outwith financial reach for many owners. In my experience, good quality radiographs can help with diagnosis (Figures 2.32c, 2.57 & 2.71).

Treatment

Antibiotics will be required. Options include SC injections of penicillin q48hrs in combination with oral metronidazole, or doxycycline or azithromycin (alternative first line) or ciprofloxacin can be considered due to the effectiveness against *Pasteurella*. Antibiotics should be continued for at least 4 weeks. See above for other treatments for head tilt and section on ear-based abscess/aural diverticulosis.

Lumps and bumps

Investigation of masses is performed using the same tools already utilised in more familiar species.

Abscesses

Abscesses are extremely common, present as subcutaneous firm, cool, and often non-painful masses. They are often missed in the early stages due to the initial small size, location and the fur. Some abscesses can become very large, with resulting pressure necrosis of the overlying skin. Rupture of the abscess may occur, especially if there is necrosis of the skin.



Figure 2.71 This is a conscious radiograph of a rabbit with torticollis. The head is rotated but there is an obvious difference between the tympanic bullae.

Rabbits lack the lysosomal enzyme required to digest dead cells which results in their pus being very thick. The abscess itself will often have a dense fibrous capsule with poor blood supply, meaning antibiotics struggle to penetrate the capsule. This is why medical management alone is usually unsuccessful.

Diagnosis can be achieved by aspirating the mass, although a large bore needle may be required due to the nature of rabbit pus.

Surgical intervention is recommended in virtually all cases. The exact approach will vary depending on location and underlying cause of the abscess formation. Ideally part of the capsule wall should be submitted for bacterial culture and sensitivity as the pus itself (often sterile) is unlikely to yield a result.

Broadly speaking, abscesses should be treated like tumours and if possible, the entire abscess should be removed in toto. This is often achievable in many areas. Any facial or mandibular abscesses should have radiography performed to determine if they are dental in origin. Dental associated abscesses are more challenging and are covered in the dental surgery section.

Marsupialisation surgery

In areas where full resection is not achievable, marsupialisation of the abscess achieves good results. Owners should be counselled that initially the cosmetic appearance of an open wound may be unpleasant, some owners may find this quite traumatising. There will be frequent check-ups and it will take in the region of four weeks to fully heal.

My preference is to hospitalise the rabbit overnight to allow me to perform the first postoperative cleaning of the wound. Always warn owners there is a risk of recurrence. Pre-emptive analgesia and antibiotics should be given. A good first line choice while waiting for culture results would be a combination such as: penicillin injections and oral metronidazole or trimethoprim sulfamethoxazole orally and oral metronidazole. The combinations cover for aerobes and anaerobes.

Incise the skin over the abscess, and blunt dissect the tissue taking care not to penetrate the capsule of the abscess. Continue to use blunt dissection to separate as much of the abscess capsule from the healthy tissue as possible. If the abscess bursts, or you have reached the limit of what is resectable, it is important to remove all of the capsule of the abscess as *leaving any of the capsule behind will increase the risk of recurrence*. If necessary, the capsule can be stripped off underlying tissue. Debride any devitalised or necrotic tissue. Flush the area with warm saline or 0.1% chlorhexidine. The skin is sutured to the open edges of the soft tissue using a fine monofilament, leaving a large deficit to heal by secondary intention. This allows daily flushing, debridement, and any topical therapeutics.

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I advise owners to use saltwater (1 teaspoon full of salt to 500 ml cooled, boiled water) to flush the wound daily. Using a cotton bud to remove any discharge in the wound. Topical agents such as: manuka honey, F10 barrier ointment, or Flamazine cream can then be applied. Rabbits often heal too fast and the wound will contract dramatically in the first 7 days. Thick scabbing should be removed via soaking and gentle rubbing. The sutures can be removed at this stage.

Any granulation tissue present will bleed during cleaning, which owners may find frightening if not prewarned – remember they do not know granulation tissue is wanted and a good thing. Managing owner's expectations by explaining what is likely to happen will help alleviate their (and your) stress. In most cases, by 4 weeks postoperatively the entire deficit will have closed.

Thymoma

Thymomas are *relatively common* in the pet rabbit. They are benign, and although thymic lymphoma and thymic carcinoma occur, they are both reported as being much less common. They are a slow growing thoracic mass which is locally invasive.

Due to the location, thymomas are either diagnosed as an incidental finding (during investigation of unrelated issue) or fairly late in the course of the disease due to acting as a space occupying lesion (Figure 2.72).

Clinical signs

Typical clinical signs include: dyspnoea, tachypnoea, exercise intolerance (may be missed by owners as many rabbits live sedentary lives), anorexia (in humans thymoma causes chest pain which may present as anorexia in rabbits), bilateral exophthalmos with the third eyelid protruding (note that frightened rabbits can also have bilateral exophthalmos – ask if the rabbit exhibits this at rest when home). The exophthalmos is said to be due to the thymoma compressing the vena cava, resulting in pooling of blood in the retrobulbar venous sinus. Oedema of the head, neck and forelimbs may also be present.

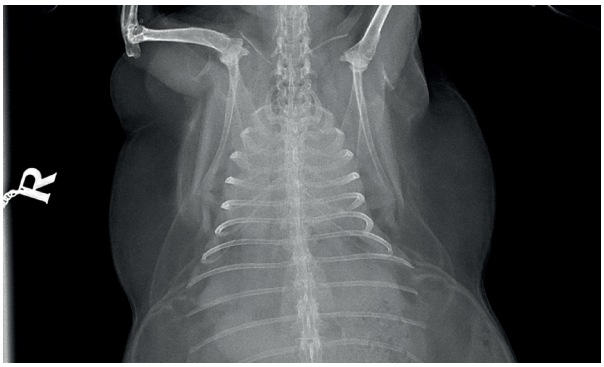


Figure 2.72 This rabbit was presented for a vaccination. The third eyelids were across the eyes bilaterally and there was reduced thoracic compliance.

The clinical exam will show reduced thoracic compliance (immediately alerts the clinician that thoracic radiographs are needed), the heart may be muffled and pulse quality may be poor. Oxygen should be available and stress kept to a minimum as these patients can quickly decompensate due to respiratory compromise.

Diagnosis

Radiography is indicated in all cases of reduced thoracic compliance. A cranial mediastinal mass, often with displacement of the heart and the trachea is seen. Advanced imaging such as MRI will give much more information. Not every owner can afford advanced imaging.

Ultrasonography can be used to examine the consistency of the mass as some thymomas have cystic compartments and needle guided aspiration of these compartments can temporarily improve the clinical signs. Ultrasonography can also be used to guide a fine needle aspirate (FNA), although a definitive diagnosis may not always be achieved.

Many thymomas are presumptively diagnosed on radiographic appearance.

Treatment

Several options are available and opinion can be divided on which option is best. Surgical removal is associated with high risk of complications and a high mortality rate which leads to many, including myself, not recommending it.

Studies have shown a variable morbidity (e.g. radiation induced pneumonitis, myocardial failure) and variable mortality associated with radiotherapy and the associated anaesthesia. Some cases have had a good clinical response and outcome with radiotherapy. The costs associated with this treatment may be prohibitive to some owners.

Palmer *et al.* (2021) looked at longevity when rabbits were treated with radiotherapy or prednisolone compared to rabbits who did not receive any treatment for thymoma with promising results. Their study suggested that prednisolone as a sole agent was the most effective treatment for extending the rabbit's life with little to no significant side effects. This was a small number of client-owned rabbits and further research is needed but prednisolone appears to be a viable option for treatment of thymomas in rabbits with minimal financial outlay for the owners.

> Owners should be advised that although no significant side effects were noted from prednisolone administration in this study, the immunosuppressive effects could exacerbate sub clinical infection such as Pasteurella for example. This would not stop me administering prednisolone and this is one of the very few conditions I recommend using this drug in rabbits.

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Skin masses

Non-viral skin masses are considered uncommon in rabbits. With increasing longevity and more owners seeking good veterinary care for their rabbit, this is likely to change over the next 20 years. The most common tumours are described in descending order of frequency seen. The list is not exhaustive but aims to cover the more likely diagnosis. Lipomas are diagnosed in the same way as for more familiar species.

Trichoblastoma

Trichoblastomas (formerly called basal cell tumours) are quoted as being the most common skin mass in rabbits. They are more frequently found on the head, neck and forelegs but can occur anywhere. Seen as solitary, often pigmented, well-circumscribed, skin masses that can grow to be quite large and may ulcerate. They are benign and complete surgical excision is curative. They may be confused (especially when small and pigmented) with malignant melanomas, therefore FNA or early surgical resection and histopathology is recommended (Figures 2.73a & b).



Figure 2.73a Trichoblastomas (formerly called basal cell tumours) are solitary, often pigmented, well-circumscribed, skin masses which are benign and complete surgical excision is curative. They may resemble malignant melanoma visually. Finances were limited so this rabbit had a wide surgical resection rather than diagnostics performed pre-surgery.

Spindle cell sarcomas

They are more frequently found in the axillae, elbow, or shoulder regions but can occur anywhere. *I have seen one case present as foreleg paresis before the tumour became appreciable in the axillae.*

The mass is firm, subcutaneous and often feels well attached to underlying tissues. Spindle cell sarcomas are fast growing and malignant. Cure can be difficult to achieve depending on location as a wide surgical excision is required (much like more familiar species).

Referral for post-surgical radiotherapy may be an option for some clients to try and achieve a complete cure. Many rabbits live good quality of life with palliative care as it only seems to be when the tumour becomes large that it clinically affects the rabbit. The exception being tumours in the axillae may affect the nerves to the leg, requiring earlier intervention.



Figure 2.73b Postoperative photo of the rabbit pictured in 2.73a. The wound healed perfectly and the histopathology confirmed a Trichoblastoma.

Collagenous hamartomas

They are more frequently found on the body and proximal limbs. The vast majority of these benign tumours are found in older male rabbits. The mass (or masses as multiple can be seen) present as gumdrop shaped firm nodules in the skin.

Squamous papilloma

These are most frequently seen on the pinnae although some may be seen on the oral mucosa. They present as warty type growths and may have keratin fronds. They are benign and usually self-limiting (Figure 2.74).

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Figure 2.74 Squamous papilloma are most frequently seen on the pinnae, are benign and usually self-limiting.

Mammary tumours

Just as with more familiar species, mammary tumours are more common in older, entire female rabbits, or female rabbits that were neutered late in life. There have been male rabbits with mammary tumours documented.

Kanfer and Reavill (2013) found that 70% of the female rabbits' mammary tumours were malignant, and may metastasise to the lungs and lymph nodes. Early detection, thoracic radiography, and surgical excision (mass excision if solitary or a mammary strip excision if multiple) is recommended to increase chances of cure. Palliative radiotherapy may be considered.

Malignant melanomas

Thankfully not very common as these are more malignant in rabbits than the ones seen in dogs and cats. They are more frequently found on the ear, eyelid and genital region but can occur anywhere and are locally invasive (including lymphatic). Head or genital locations have a much higher incidence of early metastasis resulting in death.

Initially may present as a small black freckle, which can be present for years before suddenly growing. In my opinion any small black skin lesion should be surgically removed, early surgical resection may have a higher survival rate. Complete resection may be difficult depending on location, radiotherapy may give better tumour control. A melanoma vaccine has been developed for dogs which may prove to be successfully used in rabbits. To date I have no experience (and neither have my colleagues) using the vaccine as it has so far proved to be cost prohibitive.

Dermatology

Skin masses

See the section on lumps and bumps.

Dandruff

Presence of dandruff with or without pruritus should make us suspicious of *Cheyletiella parasitovorax*. Tape tests are a quick, affordable, easy way to check for the mite and many owners are keen to see the mite under the microscope given the option. *C. parasitovorax* is a zoonotic and although most people will not be affected, any owner who is immunocompromised is at risk of a nasty infestation.

In humans, *C. parasitovorax* tends to affect the forearms and neck, and any owners displaying itchy scabs in these regions should be directed to their doctor with a written note of the parasite name.

It is important to note the distribution of the dandruff as well as its presence. Generalised distribution could indicate immune compromise or a severe, established infestation (multiple life stages may be evident on tape tests). Localised distribution e.g. a patch on the rump of lumbar region, is highly suggestive of an inability to groom that area. It is important to then do a full clinical exam including skeletal pain check as this is the rabbit equivalent of the 15-year-old cat with a matt on its lumbar spine. That being said, lack of a localised patch of dandruff does not mean the rabbit has no spinal pain.

Ivermectin (I use 1% spot on ordered from my wholesaler which works out as one drop/500 g body weight repeated every two weeks for three treatments (200–400 μg/kg)) or selamectin 15 mg/kg (6–20 mg/kg) has been shown to work against *C. parasitovorax* using a single application.

Treponema paraluis cuniculi

Treponema paraluis cuniculi is a sexually transmitted, species-specific, spirochete, which causes a condition commonly referred to as rabbit syphilis. Owners should be reassured that this is not zoonotic. Asymptomatic carriers may develop overt disease months or even years post-infection. Overt disease may be precipitated by a stressful event.

Clinical signs

Clinically it will typically present as crusting lesions at mucocutaneous junctions: nares, lips, eyelids, anus, and external genitalia. There may be swelling and erythema

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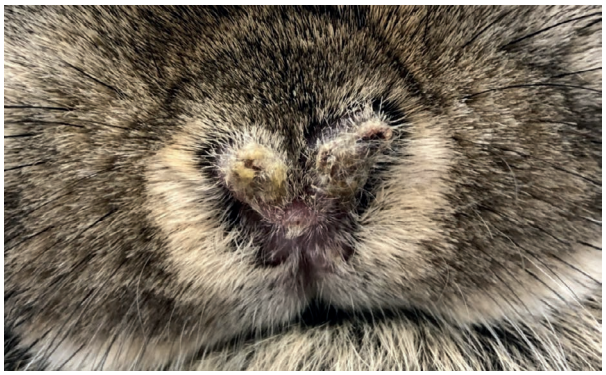


Figure 2.75a Rabbit syphilis is sexually transmitted (including dam to kits via birthing). Note the crusting lesions at mucocutaneous junctions in this case the nares.



Figure 2.75b The rabbit pictured in 2.75a showing crusting lesions at the external genitalia.

in the early stages progressing to crusting, which may be frond like at the nares/on the nose (Figures 2.75a & b).

Diagnosis

Diagnosis is often made presumptively on clinical signs. Subclinical infection is believed to be common therefore a positive antibody result may mean exposure not causation. Scabs or a swab can be taken from the lesion and submitted for a PCR test or examined using dark background microscopy for identification of the organism.

Treatment

The treatment of choice is injections of penicillin: 42,000–84,000 IU/kg SC is given once weekly for three treatments. I find international units (IU) confusing, but with penicillin 1 mg is approximately 1600 IU (depending on which salt, G sodium is 1667 IU, G potassium is 1595 IU). I have successfully used amoxicillin 20 mg/kg SC in place of penicillin. *Remember that oral ingestion of penicillin or amoxicillin can cause a fatal dysbiosis in rabbits therefore any spillage of the drug on the fur should be immediately washed off.* The scabs will usually fall off five days after the first injection.

> Any companion rabbits should also be treated as direct contact with the lesions can spread disease.

Fungal disease

Fungal skin disease typically presents as hair loss and crusting lesions around the eyes, nose, and extremities (although can present anywhere) (Figure 2.76). Nasal fungal disease can clinically resemble *Treponema cuniculi*.

Trichophyton mentagrophytes is reportedly the most common isolate but *Microsporum canis* (and other *Microsporum* spp.) have been documented.



Figure 2.76 Severe fungal skin disease, confirmed by biopsy.

Diagnosis and treatment

Diagnosis is via microscopic examination of the hair shaft and fungal culture. Treatment is always recommended due to the zoonotic risk for the owners. Topical treatment is not recommended due to rabbits finding dips and baths extremely stressful – this can lead to immunosuppression or fractures due to the rabbit panicking. Focal topical treatment is not recommended in isolation as dermatophytes will be present in areas not clinically affected.

Systemic therapy with either itraconazole 5 – 10 mg/kg q24hrs or terbinafine 10 mg/kg q24hrs should be started and continued until two negative fungal cultures are obtained a month apart. Disinfection of the environment with a suitable agent such as sodium hypochlorite or F10 is an important part of management. Wooden enclosures may be difficult to eradicate the dermatophytes from.

Ulcerative pododermatitis

A common, painful, and sometimes frustrating (to treat) condition that results in chronic ulcerative granulomatous dermatitis on the plantar aspects of the feet. It is mainly the hind feet that are affected but front feet should always

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be checked as they may develop as secondary lesions due to shifting of weight from hind to fore quarters.

Causes

Rabbits do not have protective pads on their feet, instead a thick layer of coarse fur protects the plantar surfaces. *When active, rabbits bear weight through their toes, conversely, at rest rabbits bear their weight on the area between the hock and the hind claws*. Overgrown claws also change how rabbits bear weight and owners should be advised on the importance of ensuring correct nail length.

Obesity, poor husbandry (wet bedding or wire grid flooring) and inactivity (which may be due to pain) are predisposing factors. Certain breeds and coat types, e.g. Rex and Satin, are more at risk due to the fine coat type.

Rabbits that suffer from anxiety and regularly 'alarm' thud with their back feet may be at an increased risk due to trauma of the soft tissues. Husbandry corrections (ensuring multiple open-ended hides, providing enrichment), products such as Pet Remedy or Rabbit Comfort plug ins and anti-anxiety nutraceuticals, e.g. Zylkene, Vetoquinol (rabbits <1.5 kg, 75 mg; rabbits 1.5–3 kg, 225 mg; rabbits >3 kg, 450 mg) should be utilised.

Holistic Exotic Veterinary Nutrition (HE'VN) produces a Wellbeing mix in conjunction with Hop to Forage. This herbal mix contains herbs and flowers which have been shown to decrease anxiety. It is palatable, only available to veterinary practices, and can be ordered by contacting either of these email addresses: hoptoforage@gmail.com, enquiries.hevn@gmail.com.

Painful rabbits are less active and may 'shuffle' when at rest. *This inactivity increases the amount of time the weight is borne on the area between the hock and the hind claws*. The protective layer of fur is damaged or lost as a result. The increased pressure on the skin leads to ischaemia and necrosis. As the skin closely overlays tendon and bone, it is easy to understand why tendon displacement and osteomyelitis can readily become a problem.

The location of the lesions means it is easy to forget to check for foot lesions, especially in flighty or painful rabbits. Therefore, this condition is underdiagnosed in general practice. Several studies have shown that the prevalence of lesions can approach 100% in obese rabbits but even rabbits of a normal body condition score have a high prevalence. Without a doubt, *the under diagnosis of this painful condition is a welfare concern* and I have been just as guilty in the past of not checking the feet. Even if there are no immediately obvious lesions, parting the fur on the plantar aspect of the metatarsal area is advised.

Scoring system and management

Mancinelli *et al.* (2014) developed the Pet Rabbit Pododermatitis Scoring System (PRPSS) for clinical assessment of hock lesions. They graded from: 0 (no lesions), 1 (mild), 2–3 (moderate), 4–5 (severe), to 6 (loss of function).

It is important to manage owners' expectations at the beginning. They need to understand that this will not be a 'quick fix', they will need to be willing to adjust their husbandry and their pet may be on analgesia for the rest of their lives. If the owner cannot commit to the time, financial, or emotional cost of the treatment then euthanasia should be discussed.

Sometimes owners are waiting for us to broach the topic of euthanasia, sometimes the mere fact we have broached the topic will make them understand how severe a condition is in a way no other explanation can.

Treating the lesions depends on the severity. Advising owners to provide deep bedding or providing several layers of fleece blankets/foam floor tiles under the bedding hay will help provide support to the feet.

As soon as lesions are observed, a full clinical exam with special attention paid to skeletal pain should be performed (see section on pain). Multimodal analgesia should be started and owners should be counselled that analgesia may be required long term especially in cases of spinal pain or osteoarthritis.

Mild lesions are seen as a small, circumscribed, circular area on the plantar aspect of the hock, bald, with minimal redness, and no infection or bleeding. It would be easy to miss mild lesions in my opinion. These may be resolved with weight loss (in cases requiring it), deep bedding for support and analgesia. Close monitoring is required to ensure there is no further progression.

Moderate lesions are red, extend along the foot, and can feel spongy to the touch (due to infection of the subcutaneous tissues). These will be very painful (can you imagine if the soles of your feet looked like this) (Figures 2.77a–c). Systemic antibiotics are indicated and ideally would be chosen on culture and sensitivity results. Broad-spectrum antibiotics such as trimethoprim sulfamethoxazole (20–30 mg/kg q12hrs) or enrofloxacin (10–20 mg/kg q12hrs) are suitable choices. I apply bandaging to help pad and support the feet. Allevyn is my primary wound dressing of choice in cases without broken skin. It is spongy, gives support to the tissues while healing and I can easily trim to the shape of the foot and extend beyond the point of the hock. I use Micropore dressing tape to apply the dressing. I describe them as having to wear a pair of slippers until healed and advise owners it could be many weeks until the dressings are no longer required. I continue with Allevyn (Smith & Nephew) dressings until the fur has regrown. Most rabbits leave the dressings completely alone in my experience. I change the dressings twice weekly although some owners are capable of doing this themselves.

Severe lesions occur when there is full skin thickness loss. Usually this is seen first on the point of the hock, the tissue resembles a doughnut, and underlying tissue bulges

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Figure 2.77a The rabbit had skeletal pain. The point of the hock had a full skin thickness sore.



Figure 2.77b Rabbit pictured in 2.77a with Allevyn slippers.



Figure 2.77c Rabbit pictured in 2.77a after a couple of weeks treatment.

from the centre. Infection is present, and the tissue may readily bleed when cleaned. The infection can extend into tendons and bone.

If concurrent bone involvement is suspected then radiography is indicated. *Osteomyelitis or tendon displacement carries a guarded to grave prognosis and we should consider whether it is in the best interests of the rabbit to continue with treatment.*

Necrotic or abscessed tissue should be surgically debrided under general anaesthesia and a swab taken from deep tissues for culture and sensitivity. Systemic antibiotics are indicated and ideally would be chosen on culture and sensitivity results. Broad-spectrum antibiotics with good tissue penetration such as enrofloxacin or parental penicillin are suitable choices. In cases of deep infections, antibiotics may be required for eight weeks or more.

In open or infected wounds my primary dressing of choice is Granuflex (Convatec). Granuflex is a hydrocolloid dressing and comes in a variety of thicknesses – I favour the thicker to provide more support. It absorbs exudate, supports the soft tissues, and keeps the wounds moist. It sticks to the wound (therefore should not be used in an area where stitches or staples are present), and removing the Granuflex will help physically debride the wound. I warn owners that when the Granuflex is initially removed, the wound may look worse as it will be covered in exudate, once cleaned with sterile saline the area will look much

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better. My preference is to hold the Granuflex in place with Micropore dressing tape. The dressings should be replaced every two days in the first two to three weeks post-debridement, decreasing to every five days once the tissue looks healthier. When the skin has healed, I change to Allevyn dressings until the fur has regrown.

End stage occurs when there is complete loss of function and euthanasia should be recommended on welfare grounds.

Fractures

Fractures are more commonly seen in rabbits less than 2 years of age and frequently owners will not be aware of the fracture, presenting the rabbit because of lameness. Failure of the owner to appreciate the severity of the injury can result in a significant delay in presentation. Some may present with a known history of trauma, e.g. jumping from owners' arms.

Pathological fractures can occur due to osteopenia (lack of exercise, lack of UVB, diet too high in phosphorus/too low in calcium, *a double cortical line is a radiographic feature*), osteomyelitis, and neoplasia.

Clinical examination for fracture location is very similar to more familiar species. Open fractures are stated to be more common in rabbits and careful palpation through the fur is required. Observe the rabbit from above to note any subtle change in weight bearing (see Figure 2.29a). The prey freeze response can make certain reflexes more difficult to interpret such as the pupillary light reflex. The paper slide test is useful to assess proprioception without risking iatrogenic damage.

Extreme care should be taken during spinal palpation to ensure unstable fractures/luxation of the spine are not exacerbated. Obvious scoliosis of the spine (if not previously noted and therefore not congenital) should prompt radiography and careful handling when sedated as the epaxial muscles will not be supporting the vertebrae.

Various options exist for fractures in rabbits and many owners are keen to explore options. *Pain causes anorexia and gut stasis so prompt analgesia and supportive care is required.*

The main differences in rabbit orthopaedics that we need to appreciate are the following.

- Rabbit bones are thin and brittle (skeleton accounts for 7% body mass compared to 16% in most other mammals), this makes comminuted fractures more common and the bone prone to iatrogenic fracture during repair – think glass!
- Rabbits rest and move with their limbs very close to their body and in much more flexion (elbow, stifle, and hock) than more familiar species. This makes external coaptation very difficult to effectively apply.
- Dogs and cats find it easy to lift a painful limb while ambulating normally on the remaining three limbs – rabbits find this very difficult. Rabbits also rely on their front legs for thorough grooming and hind legs for balance when grooming perianal regions or consuming caecotrophs.

Spinal fractures/subluxation

The injury is most commonly seen in the caudal lumbar vertebrae in rabbits, but can occur at any point along the spine. Presentation is dependent on location, just as with more familiar species. Clinical signs may include: paresis, paralysis, loss of reflexes, deep pain sensation present or absent, urine retention (may have overflow), anorexia, pain on palpation, faecal incontinence. Any voluntary urination and passage of faecal pellets should be noted.

Assess the gait of all limbs and take care during palpation to prevent iatrogenic damage. It can be more appropriate to progress to sedation and radiographs rather than palpate the spine.

Differential diagnosis includes intervertebral disc extrusion, vertebral traumatic fracture, vertebral pathological fracture, vertebral subluxation or *E. cuniculi*. *The most common cause of traumatic posterior paralysis in rabbits is vertebral fracture and luxation.*

In an ideal world, sedation and radiography (and myelography) should be performed. Complete fracture and luxation with obvious spinal cord transection (we cannot see the spinal cord on standard radiography, however in cases where there is complete separation of the spinal vertebrae, with one segment severely dorsally displaced and overriding, it is safe to assume transection in my opinion) carries a grave/hopeless prognosis and euthanasia should be recommended. In some cases, this can be evident on the clinical exam.

Many other spinal injuries (including individuals with multiple spinal injuries) have been shown to do well with analgesia, supportive care, and strict cage rest.

I have had owners argue that they would rather spend their limited funds on trial therapy rather than progress to sedation and radiography. Providing there are no concurrent limb fractures I personally do not have an issue agreeing to the trial therapy, although I remind the owner if there is osteomyelitis of the spine it will be missed and therefore not treated. In my experience osteomyelitis in the spine is very uncommon, and areas of infected bone need surgical intervention. Sedation and handling for radiography carries the risk of iatrogenic trauma to the spine due to the loss of the muscular internal support. I advise the owners it may not work and if it does work, they are looking at a minimum of six weeks cage rest with good nursing care being a necessity. It is recommended to hospitalise the rabbit for at least the first four days (Figure 2.78).

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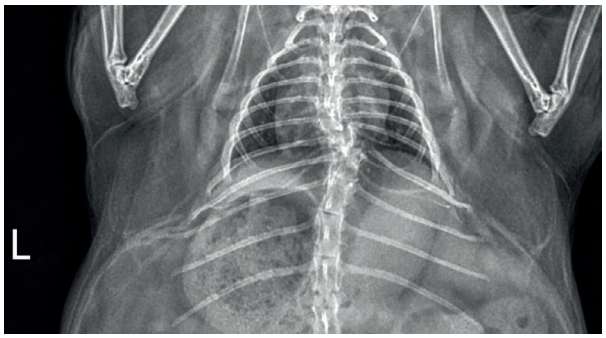


Figure 2.78 Note the old dislocation fracture of the thoracic vertebrae. The owner was completely unaware that the rabbit had previously damaged the spine. We were radiographing to assess for dental disease.

Analgesia

NSAIDs have been shown to improve spinal cord blood flow, which may help the healing process (McCullough *et al.*, 2012) and are commonly used as part of a multimodal analgesic plan. Opiates, e.g. methadone, should be included in the analgesic plan given the severity of the pain associated with spinal injuries. Thought should also be given to neurogenic pain control utilising gabapentin.

Supportive care

IV fluids are important to prevent hypotension and ensure good vascular supply to the spinal cord. Encourage the rabbit to eat by using favoured hay and food stuffs. Supplementary feeding may be required using the preferred syringe feeding feed (various flavours are now available). *Close monitoring of urine and faecal output is required*. Urinary catheterisation may be required in the short term if the rabbit is urinary incontinent.

Deep, soft bedding is advisable to prevent sores. Rabbits that are not mobile should be frequently turned. Once eating well, passing faeces, and passing urine, the rabbit may be discharged with oral medications for owners to administer at home. Strict cage rest should be continued and although the rabbit needs to be physically isolated from other rabbits, an ideal set up would include a mesh/wire wall that would allow limited contact.

Physical therapy may be beneficial in some cases although it is recommended this is done on a case-by-case basis *under the guidance of a veterinary physiotherapist* as some exercises may be detrimental in certain cases.

Regular check-ups are scheduled every fortnight or so, sooner if the rabbit stops eating or is losing weight. Some owners may be reluctant to revisit for fear of making the spinal injury worse. The owner should be made aware if there are any secondary issues, e.g. sore hocks or urine scald, that go untreated they are life threatening. Advise a non-slip, well-padded travel container that opens from the top as well as in front to minimise movement of the spine when entering/exiting the travel container.

If voluntary urination fails to return in one week or movement fails to return to hind limbs in three weeks, serious consideration needs to be given to euthanising the rabbit. Having rabbits on wheels is a serious welfare concern for me. The wheels prevent species-specific behaviour such as ingestion of caecotrophs and urine scald may be an issue.

Long bone fractures

Most reports quote the frequency of long bones fractured in descending order as femur, tibia, radius/ulna, and humerus. One report had tibia as the commonest presented long bone fracture.

Microfractures in the cortex extending from the main injury are common in rabbits and can be missed with conventional radiography. This can lead to fragments breaking off during repair.

The most commonly utilised technique for repair is external skeletal fixator (ESF), with or without an intramedullary pin tie-in. Some surgeons advocate plate usage but opinion is divided as the small size and brittle nature of rabbit bones can make adequate screw placement a challenge. Pre-drilling screw holes slightly smaller than required may help reduce iatrogenic fracture/fissures.

Recently transfixation pin splinting has been described as an alternative to external coaptation for treating fractures of the distal long bones (tibia, radius, and ulna) with good results albeit a small sample size.

External skeletal fixator

The basic principles of using an ESF are similar in all species. *Manual placement can result in wobble that is likely to lead to premature loosening of the pins therefore low rotational speed (150–300 rpm) insertion is advised. A recent study of the femur indicated that the pin diameter selected should not exceed 15% of the bone diameter* to reduce the risk of iatrogenic fractures. More work is needed to establish if the same holds true for the other bones but it is logical to assume it does until proven otherwise.

Positive profile threaded pins are preferred, utilising pre-drilled holes one size smaller than the pin to reduce the risk of iatrogenic fracture. If these are unavailable, smooth pins may be enough, provided they are inserted at an angle without predrilling.

Two to three pins spread evenly per segment is advised if no intramedullary pin is used, one pin per segment may be sufficient if an intramedullary tie-in is used. *The intramedullary pin should be no more than 30–40% the diameter of the bone to allow room to place the transfixation pins*. The connecting bar should be placed as close to this skin

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as possible while taking into account the likely degree of post-operative swelling. A lightweight connecting bar can be fashioned from epoxy resin or epoxy putty. A postoperative radiograph to check placement is advised.

Hospitalisation for a few days postoperatively will allow daily cleaning and inspection of the pin/skin interface while allowing monitoring analgesia level for efficacy.

Skin wounds can be checked weekly then extended to every 2 weeks if stable. One study suggested the ideal time to remove ESF was 6 weeks postoperatively. Clinically it is advised to stage removal when confident the fracture is stable, has normal stiffness, but may not yet be at full strength. Staged removal is the same as for other species, starting with any loose pins first if only ESF. In cases where an intramedullary pin is used (and the ESF pins are stable), cut the intramedullary in where it joins the connecting bar and tape in place to prevent migration. Remove the intramedullary pin a week later, and gradually remove the ESF over the next two weeks. If an intramedullary pin is used and the ESF pins are loose, leave the intramedullary pin and deconstruct the ESF first.

Complications are common and include fissures/fracture of bone during pin placement, infection of pin entry wounds, osteomyelitis, and non-union. A swab should be taken for culture and sensitivity from entry wounds and antibiotic selection made in conjunction with the results. Start on enrofloxacin while waiting on the results. Clean the entry wound and flush with sterile saline. In cases of osteomyelitis surgical debridement may be required due to the thick nature of rabbits' pus and any necrotic bone will act as a sequestrum.

Non-union can still result in a functional limb in rabbits. If ambulation is good and any pain is controlled, this may be preferable to repeated limb saving surgery or amputation.

Transfixation pin splinting

A modification of the transfixation pin cast used in large animal orthopaedics for treatment of comminuted fractures of the distal limb. Ciwko *et al.* (2018) presented two case reports on the use of transfixation pin splinting (TPS) as a method of reducing the complications of current fracture repair techniques *for distal long bone fractures* in the rabbit.

Pin size selection in that report was less than 20% of the diameter of the bone but given previous studies, *I aim for 15% of the diameter of the bone*. Pre-drill the holes with a small diameter drill bit to reduce risk of iatrogenic fractures and premature pin loosening.

The location of the entry site should be chosen with minimisation of damage to soft tissues in mind. Pins should be placed in the proximal fragment (more than half the bone diameter away from the fracture site) and engage both cortices but only exit from the insertion side of the limb again to minimise soft tissue damage. The distance separating the two pins should be equal to six times the pin diameter.

The pins are encased in a fibreglass splint which transfers forces from the pins directly to the ground through the splint, bypassing the fracture site. The bone fragments are held in alignment when the splint is applied. Ciwko *et al.* (2018) recommends using a partial circumference thermoplastic splint to allow careful dressing changes and monitoring of the underlying skin. They experienced soft tissue morbidities associated with the cast when they utilised a full circumferential cast. A postoperative radiograph to check placement is advised.

Closely monitor the site for dressing associated soft tissue trauma. Rabbits have very little soft tissue on their distal limbs and as they have no protective pads on their feet, any soft tissue trauma can potentially have devastating results. Removal of the pins and cast was performed when there was radiographic evidence of a bridging callus which was within six weeks.

Metacarpal/metatarsal fractures

Providing that not all of the metacarpals/metatarsals are fractured, these are likely to heal with a partial circumferential splint, analgesia, and rest.

When all of the metacarpals/metatarsals are fractured surgical intervention will increase the chances of healing (Figure 2.79). Intramedullary pinning using a 'dowel' technique similar to the technique utilised in cats is recommended (Degasperi *et al.*, 2007; Zahn *et al.*, 2007). Owners should be made aware that it is extremely difficult/impossible to remove the pins if there are post-operative complications but this is a quick technique that has shown good results.

Unlike dogs and cats (intramedullary pin size being 70–75% of the diameter of the bone), in rabbits it is recommended to choose *an intramedullary pin approximately 50% of the diameter of the bone* to prevent iatrogenic fractures (Barron *et al.*, 2010).

Place the pin into the medullary cavity of the longest fragment first and cut it with a few millimetres protruding. Then lever the shorter fragment onto the protruding end of the pin. Ideally pin all of the affected bones and perform a postoperative radiograph to check placement is advised (Figure 2.80).

A padded gutted splint or a padded partial circumferential fibreglass/thermoplastic splint should be applied for at least 4 weeks postoperatively and removed when there is radiographic evidence of union. Closely monitor the site for dressing associated soft tissue trauma. Rabbits have very little soft tissue on their distal limbs and as they have no protective pads on their feet, any soft tissue trauma can potentially have devastating results.

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Figure 2.79 All four of the metatarsal bones are fractured.

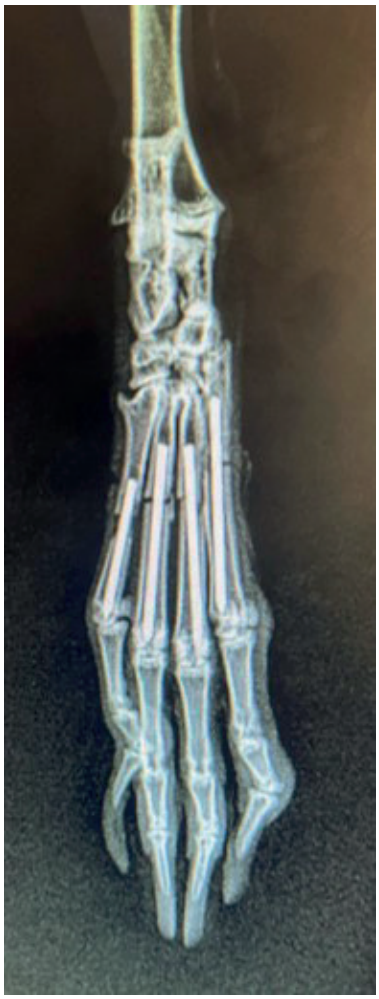


Figure 2.80 Intramedullary pinning using a 'dowel' technique.

Amputation

Opinion is divided within the profession on the ethics of performing limb amputation in rabbits. Personally, I have more of an issue with the ethics of bilateral enucleation in rabbits than I do with limb amputation in carefully selected cases.

Northrup *et al.* (2014) performed a retrospective study on limb amputation in rabbits and although it was only 34 cases, the majority of rabbits tolerated the procedure well and 91% of owners were happy with the outcome. Over half the rabbits had chronic issues with grooming or ambulation. However, some of these rabbits were overweight or had other co-morbidities (e.g. osteoarthritis).

Full body radiographs should be taken to assess for spinal disease and degenerative joint disease. Presence of spinal disease and degenerative joint disease will not stop me from performing the amputation. I once amputated the hind leg of a 6-year-old continental giant rabbit who had radiographically awful stifle degenerative joint disease and hip dysplasia. He performed chronic alert thudding whenever there was wind or even birds in 'his' garden, did not respond to anti-anxiety medication, and performed it to such an extent he gave himself such severe hock disease it was amputation or euthanasia. He lived on multimodal analgesia with good quality of life and no complications for another 18 months.

The aim of the survey radiographs is to identify any underlying conditions we may not be clinically aware of and preemptively treat them. Although they may not be obviously clinically affecting the rabbit (we should always remember they are masters at disguising pain so it does not mean they are not affected by it), amputation will increase the load bearing on the remaining limbs, alter the centre of gravity and increase the likelihood of clinically apparent pain.

Overweight rabbits are at a massively increased risk of developing pododermatitis in the remaining limbs especially if it is a hind limb amputation. This makes complete sense and the rabbit should be on a weight loss programme to prevent or reduce this complication. If pododermatitis is present on any of the remaining limbs preoperatively

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and delaying the amputation is possible, treat the pododermatitis first.

Post-operatively and for the rest of the rabbit's life it is advised to provide deep, soft bedding material and ensure the play area has suitable non-slip flooring (e.g. foam or rubber supportive tiles) to support the remaining limbs during ambulation. Monitor the remaining feet for pododermatitis during the healing period and quick intervention, support and treatment is required.

The forelimbs are of particular importance for routine grooming in rabbits. If a forelimb amputation is required, I advise *the owners they may need to groom the rabbits daily*. If a companion rabbit is present, they will often be allogrooming anyway. Rabbits particularly enjoy being groomed around the eyes, head, and ears as this is an evolutionary safe place to be touched – i.e. that is where companion rabbits groom each other. Therefore, *if the patient is a single rabbit, the owner will need to take over that role*, washing around the rabbit's eyes (especially the side of the amputation), massaging the ear base and removing any wax build-up with cat ear cleaner.

FORELIMB AMPUTATION

For forelimb amputations I favour removing the scapula in all species. This provides a fast and cosmetic repair. Owners find the skeletal appearance of the scapula (once atrophy of the remaining musculature occurs) distressing. The brittle nature of rabbit bones makes transecting the bone for a diaphyseal amputation more challenging as shattering can occur and sharp fragment ends may need filing down. Once the limb is removed there is no longer any orthopaedic pain either due to removal of the entire limb.

HINDLIMB AMPUTATION

I favour a coxofemoral joint disarticulation amputation or proximal diaphyseal amputation. Diaphyseal amputation is more challenging as shattering can occur and sharp fragment ends may need filing down.

> Coxofemoral joint disarticulation amputation in entire males may increase the risk of trauma to the scrotum/testis on the affected side due to increased exposure. Consideration should be given to castration at the same time both to prevent this and reduce any mounting behaviour which will now take place on one hind leg.

Previous literature has advised a mid-diaphyseal fracture to allow a stump the rabbit can utilise for weight bearing when at rest. However, Hulth and Olerud (1962) noted that none of the rabbits stood on/used the stump when at rest or walking and some developed ulceration and infection at the site of the amputation. The infection and ulceration may have been due to uncontrolled pain.

Leaving a large stump behind increases the weight that the remaining leg will have to carry. With these points in mind, I cannot see why we would favour a midshaft diaphyseal amputation in rabbits.

Analgesia

Regardless of the technique utilised, it is important to discharge the rabbit with multimodal analgesia for the owner to continue at home. For non-spinal fractures I advise meloxicam, paracetamol, and tramadol, I will add in gabapentin if there is a spinal fracture. Personally, I advise continuing on with the analgesia until the leg is healed then gradually weaning off the tramadol or gabapentin before stepping down the paracetamol and meloxicam. I do not believe that the frequently quoted 10 days of analgesia is sufficient as humans will take analgesia for much longer with comparable injuries – rabbits cannot tell us they are still sore unless it is severe.

Antibiotics

Despite reducing antibiotic usage, many texts still recommend prophylactic antibiotic use following orthopaedic surgery in rabbits. This may be partially due to the caseous nature of rabbit pus which makes drainage difficult and osteomyelitis frequently requires either aggressive debridement surgery (with prolonged courses of antibiotics), or amputation of the limb.

Ideally antibiotic choice should be dictated by culture and sensitivity and open fractures should be swabbed at point of surgery. Trimethoprim sulfamethoxazole 30 mg/kg q12 hrs for 5 days postoperatively was recommended by Ciwko *et al*. (2018).

Urinary scald

In its mildest presentation, before true scalding, it can present as only staining of the fur in the groin, medial aspect of hind legs. Untreated it will progress through to urine-soaked fur extending cranially along the ventral abdomen resulting in scalded, macerated skin (Figures 2.81a & b). The fur holds the urine next to the skin, burning the skin, keeping the area damp, and a secondary bacterial infection can quickly take hold. The secondary infection and stale urine can create a very unpleasant smell for both the rabbit and the owner and the fur will start to fall out easily. Chronic cases will have folds of infected skin on the medial aspect of the hind legs. This is extremely painful.

Rabbits adopt a particular posture when urinating. They dip their back and tilt their pelvis up so that they can pass urine in a jet up and backwards. Pain in the spine, hips or stifles can prevent proper posturing during urination resulting in the rabbit urinating on themselves rather than behind them. *In my experience, by far the most common cause of urine scald is lumbar spine pain, hip, or stifle pain*. Other causes include renal calculi, bladder calculi, or *E. cuniculi*.

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Figure 2.81a In its mildest presentation, before true scalding, it can present as only staining of the fur in the groin, medial aspect of hind legs.



Figure 2.81b Urine-soaked fur is extending cranially along the ventral abdomen resulting in scalded, macerated skin.

An important consideration is that a rabbit with pain preventing proper posturing for urination is unlikely to urinate as frequently as normal (it is painful to do), therefore they will 'hold' onto the urine for longer than normal, leading to urine which is much more concentrated. As rabbits excrete any calcium excess to requirements via the kidneys in urine, concentrated urine can be supersaturated, leading to precipitation of calcium sand in the bladder (often called 'sludge').

Large build ups of 'sludge' in the bladder can result in cystitis, stretched painful bladders (with potential atony), and the inability to fully empty the bladder leading to further urinary tract issues. The bladder may be palpable as a firm, small pear-shaped structure in the caudal abdomen. The bladder is relatively thin walled compared to dogs, however gentle massage can result in the rabbit passing urine and sludge during the consultation. Discrete bladder stones are uncommon in rabbits, but when they occur, they need surgical intervention once the rabbit is stable. Cystotomy is performed using the same technique utilised in dogs and cats (Figures 2.82a–c).

Encouraging natural diuresis by providing cooled dandelion or nettle teas as drinking water will promote further fluid intake. I also advise fresh grass, and fresh dandelion leaves/flowers daily. This will help flush the bladder naturally. In severe cases, sedation, passing a tom cat urinary catheter, and flushing the bladder with sterile saline will help return the bladder to normal function faster. Holistic Exotic Veterinary Nutrition (HE'VN) produces a urinary mix in conjunction with Hop to Forage. This herbal mix contains herbs and flowers which have been shown to help support bladder health. It is palatable, only available to veterinary practices, and can be ordered by contacting either of these email addresses: hoptoforage@gmail.com, enquiries.hevn@gmail.com.

Analgesia is required (multimodal) and, in many cases, will be lifelong treatment. I have seen several referral cases where the rabbit heals up beautifully but it is recurrent. The reason being that the analgesia is stopped when the scald heals without the referring vet/owner realising that the skeletal pain is what caused the urine scald in the first place, therefore it is understandable that it will recur as once the analgesia stops, the skeletal pain prevents normal posturing and the vicious circle continues.

> Rabbits do not like being bathed and find their ventral abdomen being touched extremely stressful. There is no safe evolutionary pathway for this area of their body to be touched – other rabbits do not touch them there (unless they are fighting), and predators touch this area to kill. So, it is not difficult to understand why rabbits find touching this area difficult to deal with.

Ideally, we should be cleaning this area and trimming the fur to prevent the fur acting like a sponge and holding urine against the skin. Owing to the stress, how painful the area is and how fragile rabbit skin is, cleaning (use warm water to prevent chilling the rabbit) and clipping/trimming of the fur under a deep sedation is recommended in severe cases. It also gives a perfect opportunity to radiograph the rabbit (lateral and ventro-dorsal views) to check for: renal calculi (advise renal bloods if present), intervertebral disc disease (if narrowed disc spaces – use gabapentin also), and degenerative joint disease of the hips and stifles (Figures 2.83a & b).

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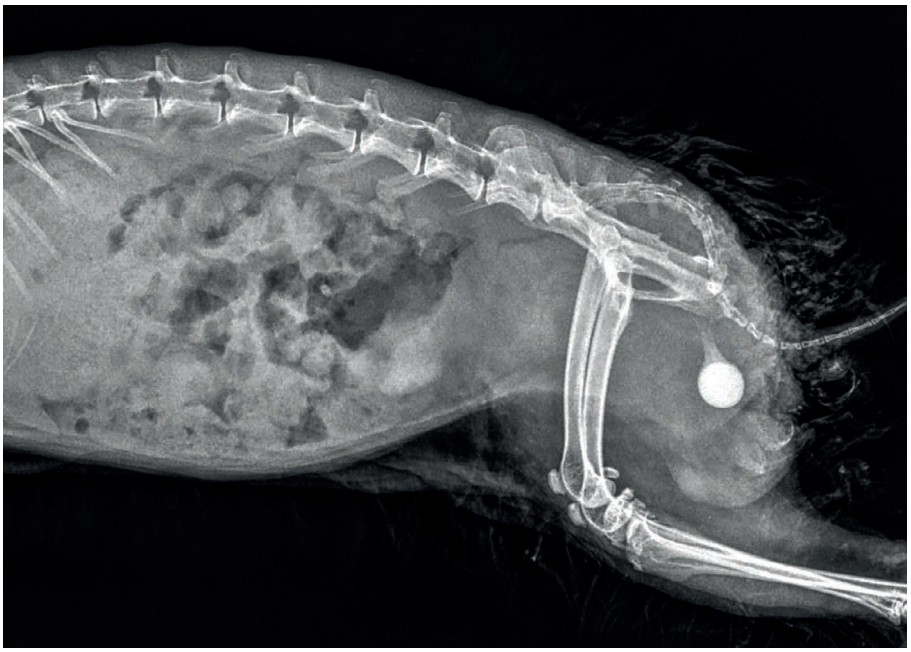


Figure 2.82a An unusual case of urinary scald which was referred to me. Skeletal pain was evident on the clinical exam but there was also a hard area in the penis. This is the lateral radiograph.

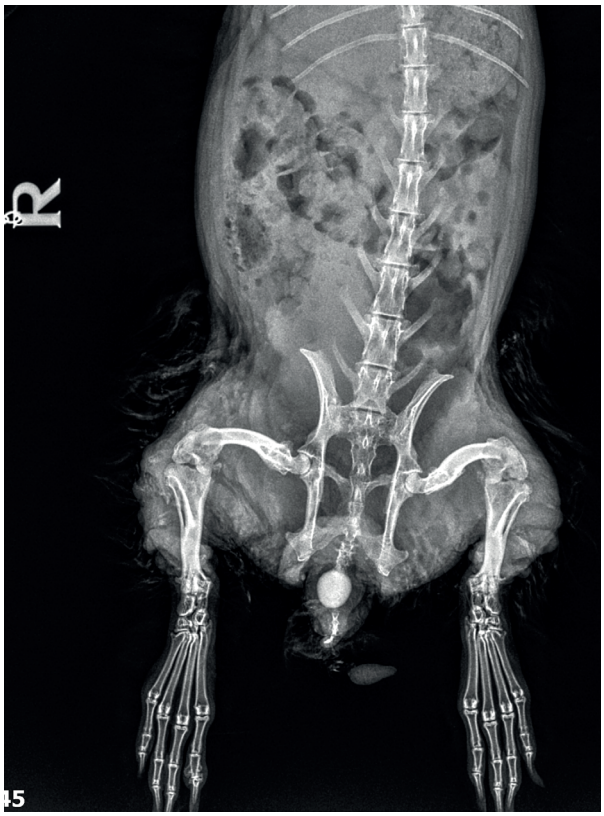


Figure 2.82b A VD radiograph of the rabbit in 2.82a. Note the urolith in the urethra, the narrowed disc spaces, and the osteoarthritic changes to the stifles.



Figure 2.82c The urolith was adhered to the mucosa and acted like a stop cock, occasionally allowing urine to dribble out. The stone was incised over through the penis, stay sutures were then placed in the urethra prior to removing the stone. The urethra was then repaired with a simple continuous suture pattern and the penis was closed in two layers. The rabbit made a full recovery and was kept on meloxicam and paracetamol long term.

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Figure 2.83a This VD radiograph shows multiple narrowed disc spaces and bilateral osteoarthritic changes in the stifles.

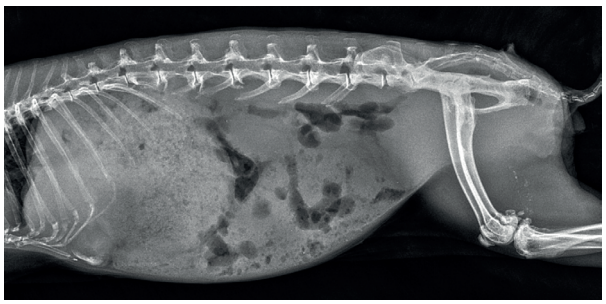


Figure 2.83b A lateral radiograph of the same rabbit in 2.82a, confirmed multiple narrowed disc spaces, spinal spondylosis, and osteoarthritic changes in the stifles.

Once the area is clean and dry, the owners can apply Flamazine cream twice daily to help with the scald. Using unscented baby wipes to remove the previous application of Flamazine is recommended, *advise owners not to put their rabbit on its back to perform this treatment*. I advise one person to safely restrain the rabbit vertically while the second person cleans and applies the cream. In white or light coated rabbits, warn owners the silver can precipitate out of the cream causing bluish grey staining to the fur or skin – this is cosmetic only.

Using puppy pads on top of their usual substrate or bedding may help wick the urine away from the rabbit faster during urination, however some rabbits dig them up or chew the pads.

Blood in urine (dipstick any that is passed on table if owner struggles to get a sample) warrants systemic antibiotics. Broad-spectrum antibiotics such as trimethoprim sulfamethoxazole (20–30 mg/kg q12hrs) or enrofloxacin (10 mg/kg q12hrs) are suitable choices. Most cases will require antibiotics for 7–10 days. Renal infections may require longer courses. Renal calculi will commonly act as a nidus of infection – examination of urine sediment will help establish if there is an infection, in the same way we would in dogs and cats.

Other, less common causes of urinary scald in rabbits include: urinary incontinence, renal disease, and spinal trauma. Spinal trauma can result in urinary retention and overflow if the innervation to the bladder is affected – much the same as in more familiar species. The resulting scald is due to the fact the rabbit is unaware it is dribbling urine (this will also be the case in urinary incontinence but is dribbling urine for a different reason.) therefore is not posturing properly and the urine will soak along the fur. *Encephalitozoon cuniculi* can cause renal or spinal issues as these are also predilection sites.

Prognosis for spinal trauma is dependent on what the trauma is and time to presentation. Non-displaced spinal fractures, with deep pain sensation (may be paretic), may do well with strict cage rest (for 6 weeks minimum) and analgesia providing the nerves to the bladder and colon have not been affected. Owners should be advised to monitor urine and faecal output carefully. Disc disease should similarly be assessed.

Displaced fractures, loss of deep pain sensation with paralysis are grave prognostic indicators and euthanasia should be advised.

Diarrhoea

Many owners are unaware of caecotrophs; therefore, it is important when presented with a rabbit the owner claims has diarrhoea, *that we remember to ask if any normal faecal pellets are being produced at all*. If they are still producing normal faecal pellets then logic dictates it is uneaten caecotrophs the owners are confusing for diarrhoea and reasons for this should be investigated.

Uneaten caecotrophs/claggy bum

Ingestion of caecotrophs (caecotrophy) is triggered by the smell and sensation of the caecotrophs being passed. Therefore, if the rabbit has chronic rhinitis (cannot smell), or spinal/nerve issues (cannot feel) they may well be unaware of the passage of caecotrophs.

If owners feed a diet including too much concentrates and low in fibre, this will reduce caecotrophy. Reducing the level of concentrates and increasing good quality fibre content of diet will lead to increased consumption of caecotrophs.

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Obesity may cause a physical obstruction to ingestion of caecotrophs and a full dietary review is advised. Spinal pain (spondylitis, disc disease, osteoarthritis) is very common and underdiagnosed in rabbits, as is hips and stifle pain. Pain in these areas prevents or reduces the frequency of caecotrophy. Balance issues such as otitis media/interna or weak hind legs will also cause issues with caecotrophy, which is completely understandable.

Another consideration is painful conditions such as dental disease, urine scald or infected perianal skin folds. If it is painful to ingest caecotrophs for any reason then it is understandable why the rabbit will choose not to.

Chronic build-up of caecotrophs on the ventral aspect of the tail and around the anus can prevent passage of normal faeces (Figure 2.84a). The skin can become burnt and infected, flies are attracted to the smell and the risk of fly strike is very high in these individuals (Figure 2.84b).

A full clinical exam including skeletal pain check and oral examination should be performed to diagnose any underlying causes and appropriate treatment instigated. Full dietary review and adjustment as required.

In severe cases, sedation, and careful removal of the faecal build-up, careful clipping (rabbit skin tears easily) of fur and cleaning of damaged skin should be performed. Scald of skin can be treated with Flamazine cream application twice daily. Analgesia should be given and F10 wound spray with insecticide (toxic to cats) can be used on the perianal region to try and prevent fly strike. F10 wound spray with insecticide is much more cost effective and easier to apply than products such as Rearguard (Novartis).

Fly strike (myiasis)

This is an emergency. Rabbits with faecal build ups, urine scald, or wounds are at the highest risk. Flies such as bluebottles and greenbottles are attracted to the smell and lay eggs. These hatch in 24 hours into maggots and essentially eat the rabbit alive (Figure 2.85a).

Even rabbits who appear bright initially are in shock. IV fluids should be given, warmth, sedation administered (increased risk but needed), maggots should be manually removed. Clip the fur and flush the wounds with warm saline (Figure 2.85b).

I administer meloxicam on the basis it will help with pain and may help with endotoxins. Secondary bacterial infection is very common so I will give a broad-spectrum antibiotic. Topical Flamazine application may help in some cases. Administer ivermectin (0.4 mg/kg if injection) to kill any remaining maggots or any that freshly hatch from missed eggs. F10 wound spray with insecticide can be used as part of the treatment protocol, it will also kill any remaining maggots as well as prevention (Figure 2.85c).

In severe cases euthanasia should be considered.



Figure 2.84a Uneaten caecotrophs/claggy bum.



Figure 2.84b A different rabbit with uneaten caecotrophs resulting in fly strike.

True diarrhoea

True diarrhoea is more frequently seen in juvenile rabbits, although I have also seen it in older rabbits following a stressful incident. Incorrect antibiotic choice can also cause a gut dysbiosis leading to potentially fatal enterotoxaemia (*Clostridium* spp. or *Escherichia coli* oral penicillin will cause this, parental penicillin is unlikely to cause this, however if the injection leaks drug onto the fur and is not washed off this will lead to oral ingestion during grooming).

Large volumes of liquid faeces are passed seemingly with little to no voluntary control and the rabbits hind quarters may be covered in diarrhoea (Figure 2.86). These rabbits will often be dull, dehydrated, may have electrolyte

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Figure 2.85a Flystrike in a rabbit with urine scald.



Figure 2.85c The same rabbit seen in 2.85b 10 days later.



Figure 2.85b A rabbit after treatment for flystrike. Note the open wound on the tail after removal of the maggots.



Figure 2.86 In cases of true diarrhoea large volumes of liquid faeces are passed seemingly with little to no voluntary control and the rabbits hind quarters may be covered in diarrhoea.

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imbalance, and have abdominal pain (may be hard to elicit due to demeanour). Enterotoxaemia can occur without being antibiotic induced, and can present as peracute and sudden death with tarry brown liquid diarrhoea. Mucous can be seen in cases of diarrhoea without it actually being mucoid enteropathy (see below).

Start IV fluids (Hartmann's), monitor rectal temperature in dull rabbits, provide warmth, administer Buscopan Compositum Solution for Injection (Butylscopolamine + Metamizole, Boehringer Ingelheim Animal Health) 0.1 ml/kg SC in cases of abdominal pain. If Buscopan is unavailable, give maropitant 4 mg/kg SC instead. Although maropitant is not an antispasmodic, it has been shown to help relieve visceral pain. Paracetamol administration may also help with analgesia.

> Diarrhoea in rabbits regardless of inciting cause will result in a gut dysbiosis. Metronidazole is the drug of choice to help correct the dysbiosis and should be administered 20 mg/kg q12hrs for 10 days.

Examine a smear of the diarrhoea under the microscope, addition of a drop of lugol's iodine may make visualisation easier. Faecal flotation using super saturated saline (use boiled cooled tap water and keep adding salt until it no longer dissolves – I keep a premade bottle in the practice) can also be used if the smear is not diagnostic.

I am looking mainly for the presence of coccidia but any worm eggs should be noted. It is unusual for worms to cause enteric pathology in pet rabbits. Although coccidia is more commonly seen in young rabbits, any rabbit experiencing a stressful situation may succumb (see section on coccidia).

Cyniclomyces guttulatus

Another common finding when examining faeces is the yeast *Cyniclomyces guttulatus* (previously called *Saccharomyces guttulatus*). These look like cigars with two circles/ovals inside them (Figure 2.87). In small numbers (one or two per ×40 field) this yeast is considered commensal, and previously in the literature it was stated even in large amounts it should be considered non-pathogenic.

This never sat well with me and if there is diarrhoea and high numbers of *C. guttulatus* I treat with Nystatin oral suspension (100,000 units/ml) 1 ml/kg q12hrs (which equates to 20 mg/kg) for 10 days. The objective is not to eliminate the yeast but to reduce numbers to a normal count. It is now recognised that while in small numbers *C. guttulatus* is commensal, with concurrent disease (e.g. coccidia) it will act as an opportunistic secondary infection and high levels should be treated (Shi *et al.*, 2021).

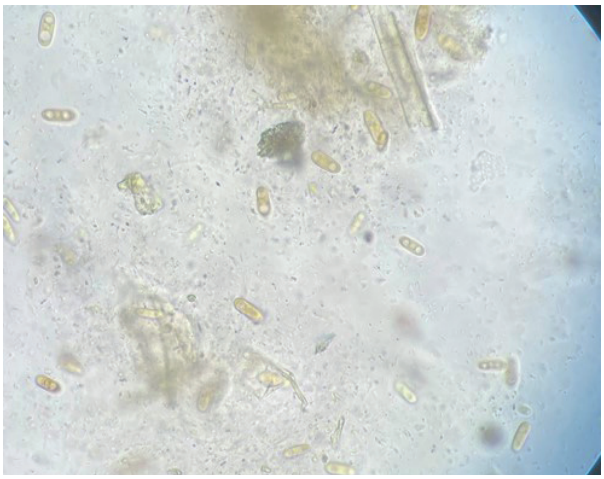


Figure 2.87 Cyniclomyces guttulatus.

Coccidia

As many as 16 different species of coccidia are known to affect rabbits. If coccidia are identified on faecal examination treatment with an anticoccidial should be started. Trimethoprim sulfamethoxazole 30 mg/kg q12 hrs for 10 days or toltrazuril can be given at a dose of 3 mg/kg once daily for 2 days, stop for 5 days then give once daily for 2 days. *Treatment of coccidiosis without administering metronidazole (as above) will most likely fail due to the continuing dysbiosis*. The coccidia will be treated but the diarrhoea will not resolve.

Freshly picked grass should be offered as a source of palatable fibre. Assisted feeding should be commenced if the rabbit is not eating voluntarily and donor caecotrophs can be included to repopulate the gut flora. Probiotics can be given (e.g. Bio Lapis, or Bene Bac) but efficacy is debated.

Mucoid enteropathy

Mucoid enteropathy is seen in juvenile rabbits and breeding does. It appears to be a non-inflammatory condition which is poorly understood and usually fatal. The resulting hypomotility of the caecum and colon results in caecal impaction with large volumes of mucous in the colon.

Clinical signs develop gradually, initially the rabbit will still be eating. The caecum becomes impacted and the stomach becomes distended with fluid and/or gas. This results in abdominal distension, tooth grinding (due to pain), depression and a hunched stance. Diarrhoea mixed with mucous progressing to only mucous will be passed rectally.

Megacolon/chronic diarrhoea syndrome

This is seen in rabbits over three years old and 'eye liner' breeds and spotted breeds such the English Spot seem over represented. Owners will often describe their pet as ravenously hungry and bouts of anorexia with caecal tympany

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are common. These rabbits often have a poor body condition score and produce faeces that are either cow pat like or soft large marble shaped (Figure 2.88). They are often paler than normal faecal pellets. The aetiology is unknown and owners should be counselled on expected control rather than cure.

Biochemistry may show anaemia (due to chronic disease), hypoproteinaemia and hyperglobulinaemia. A dietary trial is recommended as certain foods will trigger worse bouts and it is highly variable between individuals which food types will do this.

Medical management with domperidone or cisapride to help with motility is recommended. My preference is cisapride 0.5–1 mg/kg q8–12hrs (sourced from compounding companies such as Summit or Bova in UK), and this can be given daily or only during bouts. I advise daily in the hope to prevent bouts but that is opinion rather than evidence based. During bouts of anorexia or abdominal pain then multimodal analgesia (meloxicam, paracetamol, (opiates if severe)) should be given. The condition is progressive but many rabbits will achieve several years of good quality life with management.



Figure 2.88 Faeces from a rabbit with megacolon.

Anorectal papilloma

A red, cauliflower-like growth at the junction between anal and rectal mucosa. This is a benign growth with unknown aetiology and results in chronic inflammation with bleeding of the mucosa. Due to the frequent 'winking' of the anus (due to subtle straining) when I first see these, I am sure they are uncomfortable if not painful for the rabbit. Surgical excision may be curative but recurrence is common. I have had good clinical outcomes with meloxicam – reduction in the swelling, redness, and 'winking'. It is possible for these lesions to spontaneously regress.

Renal disease

The increasing longevity of rabbits means diseases associated with old age such as renal disease are being seen much more commonly in the pet rabbit population. Acute and chronic renal disease are both seen in rabbits and may be underdiagnosed, especially if owners or vets are reluctant to perform biochemistry or further investigation.

> Stress (environmental, inappropriate handling or severe pain) has been shown to cause a marked and prolonged reduction in renal blood flow and glomerular filtration rate (adrenaline release causes vasoconstriction of renal arterioles) and oliguria may result. Stress also causes gut stasis and we need to be conscious that acute renal failure can accompany any severe life-threatening disease in rabbits.

Polydipsia/polyuria is an inconsistent finding in rabbits with renal disease and one that is often missed by owners, even if present. Rabbits will much more commonly demonstrate obvious polydipsia due to pain from e.g. dental disease in my experience.

Renal stones are painful and passage of renal stones through the ureter (with or without blockage) may present with clinical signs consistent with acute abdominal pain such as sudden onset hunched body position, dull, lethargic, haematuria and anorexia. Anorexia is seen due to virtually any condition causing pain.

Interestingly, *rabbits with chronic renal disease often cope well for years without anyone realising they have the condition*. The majority of rabbits with chronic renal failure display a good appetite until the late stages of the disease, despite this chronic weight loss in the face of a good appetite, with a cachexic appearance should increase our index of suspicion for chronic renal disease (I have also seen chronic weight loss despite good appetite in chronic pain cases with normal renal function).

Clinical signs may be misleading (they are often the same regardless of the disease process) or relatively mild compared to the severity of renal disease. Urinary scald

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may be present but is more commonly present in rabbits with healthy urinary tracts due to skeletal pain.

Abdominal palpation can be useful to assess the size, shape, texture and if there is pain associated with kidney and bladder palpation. Abdominal guarding due to pain can be very subtle in rabbits compared to more familiar species. A *repeatable* flinch (can be a subtle tightening of abdominal muscles), fidget, or restlessness when a specific organ is palpated is indicative of pain.

Small kidneys are suspicious of chronic renal disease and if they feel pitted, I will be considering *E. cuniculi* infection. The kidneys are a predilection site of the parasite, and disease is usually seen in the older rabbit. This is simply because it takes a prolonged time for the granulomatous interstitial nephritis and fibrosis caused by *E. cuniculi* to reach severity causing clinical disease. Treatment of *E. cuniculi* with fenbendazole (see section on *E. cuniculi*) will eliminate the organism but the renal changes are irreversible.

A large, painful kidney (unilateral more common but bilateral possible) may be associated with renal stones, pyelonephritis (may be normal sized but painful), renal neoplasia (not always painful), or hydronephrosis. Polycystic kidneys are usually large, uneven but non-painful in my experience unless concurrent pyelonephritis.

A large, painful bladder especially with a history of reduced, or no recent urination should be treated as an emergency just like a blocked tom cat. Urethral stones, bladder stones, bladder neoplasia are all possible causes of a blocked bladder in a rabbit. Renal failure can result due to post-renal blockage.

I advise a comprehensive biochemistry and haematology in every rabbit with anorexia who has a painful abdomen. This will help assess organ function (liver, kidneys, and pancreas) as well as give us information on electrolytes, calcium, and phosphorus levels. In renal disease, urea and creatinine levels will not rise in rabbits until 50–75% of the renal function is lost, therefore even with small increases in creatinine there may be significant disease. Prerenal azotaemia can occur as per more familiar species. A haematocrit of 45–50% may indicate dehydration.

Urinalysis

A urine sample can be obtained similarly to a cat, especially in litter trained rabbits. Alternatively, careful manual expression or urinary catheterisation can be used to obtain a sample. Cystocentesis should be performed with care and via ultrasound guidance in case of inadvertent caecum puncture, I personally do not perform cystocentesis due to the large voluminous intestinal tract. Any urine sample obtained using the last three methods has a higher risk of red blood cells being present through iatrogenic trauma. See Table 2.9.

Table 2.9 Urinalysis.

Urinalysis normal	
Colour	Highly variable, diet dependant
Protein	Trace
pH	8–9
Specific gravity	1.003–1.036 (fixed range 1.008–1.012)
Sediment examination	Calcium crystals, struvite crystals
UPC	0.14–0.4

Urine colour

Normal urine varies in colour depending on diet: pale straw colour, yellow, orange, brown, and red. Porphyrins from plant pigment breakdown are responsible for the wide variation in normal colour for rabbit urine. The intensity and which foods cause it will vary between individuals. Turbid white urine can also be a normal finding due to the presence of calcium sediment.

Clear urine (unless lactating doe or young growing animal) could mean that the rabbit is not fully expressing the bladder during urination, leaving behind the sediment, or that end stage renal failure has occurred and there is no longer any renal clearance of calcium.

Specific gravity

Specific gravity should only be measured once the sample has been centrifuged to remove the sediment, and performed using a refractometer.

The normal specific gravity for rabbit urine varies between 1.003–1.036. Measurements outwith their fixed range of 1.008–1.012 indicates renal function, whereas, a measurement within the fixed range in a patient who is azotemic and dehydrated would indicate renal failure.

Dipstick examination of urine

Urine testing positive for blood on dipstick could be indicative of haematuria, haemoglobinuria, or myoglobinuria. If the urine is visibly red, centrifuging will differentiate haematuria from haemoglobinuria. Haematuria will result in the urine going yellow again as the red blood cells will form a pellet during centrifuging. Myoglobinuria tends to cause the urine to be brown.

Like all herbivores, rabbits have alkaline urine, 8–9 pH. Neutral or acidic urine is a poor prognostic sign.

Trace to small amounts of protein are normal, however small amounts of protein in very dilute urine is more significant. Laboratory analysis or urine protein creatinine ratio (UPC), with concurrent sediment exam, can be used to help diagnose or monitor for progression of renal disease. Reusch *et al.* (2009) established the UPC reference

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range as 0.14–0.4 for healthy rabbits. *Persistent proteinuria with inactive sediment indicates tubular or glomerular disease, much the same way as we monitor for more familiar species*.

Sediment exam

This can be performed in-house as per more familiar species or sent to an external laboratory. If performing in-house it is important to remember that calcium crystals and struvite (ammonium magnesium phosphate) crystals are a normal finding. Some antibiotics e.g. trimethoprim sulphonamides can result in sulphonamide crystals forming in the urine, so if an unusual crystal shows up during the sediment exam it is important to check the recent drug history.

The presence of casts is indicative of glomerular or tubular disease and the presence of white blood cells suggests infection especially if bacteria are also seen. Sending urine for bacterial culture and sensitivity is recommended.

Red blood cells may be artifactual depending on technique used to collect the urine.

Diagnostic imaging

In any suspected case of renal disease, radiographs are strongly recommended. This is not only to check for renal/bladder stones.

In contrast to carnivores, high blood calcium is common in end stage chronic renal failure in rabbits (renal function is required for excretion of calcium). Intestinal absorption of calcium continues despite the reduced renal excretion caused by chronic renal failure, and the resulting hypercalcaemia can cause ectopic mineralisation affecting the aorta, myocardium, bronchus, and renal cortex. Hypercalcaemia can also cause increased bone density (osteosclerosis). *Calcium is not as tightly regulated in rabbits compared to other species as it is more depending on diet compared to other species. Remember that many reference ranges available were worked out in laboratory rabbits that have tightly controlled diets and therefore tightly controlled dietary calcium levels*.

The presence of increased bone density on a rabbit radiograph (will not be every bone and affected bones will be much whiter with blurred outlines due to mineral deposits) should trigger an investigation into renal disease if that was not the primary reason for radiographing.

Ultrasonography is useful to assess the renal parenchyma. Hydronephrosis or renal cysts are readily visualised as expected for other species. Renal stones may cause an acoustic shadow.

Treatment

Metoclopramide can reduce renal blood flow exacerbating any underlying renal issues. Clinically I have not experienced any issues with using metoclopramide but to minimise risks, if renal disease is suspected in an anorexic rabbit, cisapride is my preferred prokinetic.

As with renal disease in more familiar species, dehydration is commonly present. Dehydrated rabbits will need IV fluids. Care should be taken to minimise stress during rehydration to prevent a reduction in renal blood flow and potentially a resulting over perfusion.

Warmed saline given at 4× maintenance rate (150 ml/kg/day) which is: 25 ml/kg/hour, 600 ml/kg/day for the first 24 hours.

The rabbit should be closely monitored (every 30 minutes) for signs of overperfusion: breathing, tissue oedema (scrotal sac useful to check for this in male rabbits), urine output. If signs of over perfusion are present the fluid rate should be decreased to 2× maintenance and some cases will require cessation of fluids and diuretics. Assisted feeding should be commenced at 20 ml/kg/feed to encourage gut motility and prevent hepatic lipidosis.

With high levels of urea, uraemic ulcers in the gastrointestinal tract are a recognised risk. Treatment with famotidine (0.5–1 mg/kg q12–24hrs) should be started. Grinding tablets to dissolve in a known quantity of water (makes a mg/ml solution) can allow easier administration. I also administer paracetamol (acetaminophen 15–20 mg/kg q8–12hrs) for analgesia.

After the first 24 hours I decrease to 2× maintenance and recheck renal parameters. The rabbit should be brighter, passing urine and starting to nibble food. If the renal parameters (especially potassium) are lower and the rabbit is brighter I continue on with IV fluids for another four days. The last two days it is important to gradually decrease the fluids to maintenance only to prevent medullary washout. Repeating the renal blood tests is recommended once the rabbit appears stable and eating. I advise owners it is important that their rabbit can maintain lower renal parameters on their own, without the IV fluids.

Increasing fluid intake in the longer term can be encouraged by: using nettle or dandelion tea (natural diuretic) instead of drinking water, offering water from a bowl rather than a bottle, offering freshly picked grass, and soaking hay prior to feeding. Periodically (just like cats with chronic renal disease), the rabbit will benefit from IV fluids. I do not agree with subcutaneous fluid administration as it is painful. It is important that owners realise chronic renal disease is irreversible whereas acute renal disease may be. Chronic renal disease may present with an acute flare up, however presence of anaemia should alert us to the chronicity.

Persistent proteinuria can be treated with benazepril 0.25–0.5 mg/kg q24hrs. *There are some reports that rabbits may be more susceptible to the hypotensive effects of benazepril so some authors suggest starting at 0.1 mg/kg and monitoring for signs of hypotension*. Hypotension will further exacerbate renal disease. The normal systolic blood pressure in a rabbit using a Doppler in a clinical setting is 120–180 mmHg. Benazepril, like so many of the medications that

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we use in rabbits, has not been validated for use in rabbits. I have found it useful in several cases as have colleagues anecdotally.

Liver disease

A rabbit's liver is anatomically split into right and left lobes. The right lobe is further divided into four lobes (lateral, medial, quadrate, and caudate) and the left lobe is divided into two lobes (lateral and medial). The caudate lobe is the smallest lobe, is circular in shape and has a narrow attachment to the rest of the liver. The gallbladder is found deep within the right lateral lobe and empties via a duct into the duodenum. Rabbits produce a large volume of bile daily (125ml/kg/day!) and interestingly a rabbit's main bile pigment is not yellow (bilirubin) like ours, but is green (biliverdin) which is one of the reasons why jaundice (yellowing of the whites of the eyes, skin, and gums) is rare in rabbits, although it does not explain why they do not go green!

As the liver is integral to many processes in the body it is easy to understand how variable clinical signs associated with liver disease can be. Another consideration is whether we are dealing with a disease of the actual liver (primary liver disease), or disease of another body system which the liver is then impacted by (secondary liver disease). Examples of primary liver disease are: infections (e.g. bacterial or viral), trauma, cancer, toxins (e.g. lead poisoning), lobe torsion. Examples of secondary liver disease are: fatty liver disease (secondary to reduced appetite) and metastasis. The treatment required can be very different for primary liver disease compared to secondary liver disease although there may be some similarities.

Clinical signs

Rabbits with liver disease may present with any combination of: anorexia, poor growth, lethargy, depression, listlessness, weight loss, diarrhoea, polydipsia, polyuria, jaundice, swollen belly, difficulty breathing, bleeding disorders, neurological signs, seizures, collapse, or sudden death.

Diagnosis

Tests

Running a full biochemistry and haematology is recommended. Interpreting the biochemistry may be challenging so the parameters used for liver involvement are discussed below.

ALT (alanine transaminase): is an enzyme commonly measured for liver disease in dogs but is of limited use for liver disease in rabbits. This is because in rabbits, their heart muscle has as much ALT as their liver does! When raised, it is only measurable as raised for a relatively short period of time but is still included in the tests as significant elevations may be seen in conditions that cause severe damage to the liver such as severe fatty liver disease or liver coccidia infection.

ALKP (alkaline phosphatase): is an enzyme found in bone, placenta, liver, and the highest levels are found in the intestine and kidneys. In rabbits' livers, the highest activity of this enzyme is in the membranes of the biliary tree therefore increases in ALKP may be seen in cases where there is slowing of the flow of bile (bile stasis).

AST (aspartate transaminase): is an enzyme found in heart muscle, kidneys, pancreas, and with the highest levels found in skeletal muscle, and the liver. Increases in AST are seen in cases of liver damage which results in cells within the liver dying.

BA (bile acid): are made in the liver from cholesterol and are essential for fat digestion and absorption. Bile acids will only rise if the liver function is impaired.

TBIL (total bilirubin): the main product of haemoglobin breakdown in rabbits is biliverdin but there is no test available to clinics to measure biliverdin. As some of the biliverdin is converted to bilirubin, we can measure bilirubin easily and this gives us information on liver function. Increases in bilirubin are seen if there is damage to the liver that is serious enough to impact how well the liver is functioning. Very high levels of bilirubin in the blood may indicate conditions which result in obstruction of the flow of bile or parasite infections such as coccidia.

GGT (gamma-glutamyl transferase): is an enzyme found in the liver and kidneys. Increases in blood GGT are only from liver origin as any increases due to kidney issues would only be measurable in urine. GGT increases indicate damage to liver cells and bile stasis.

TP (total protein): broadly speaking, serum protein is made up of two fractions: albumin and globulin. TP levels vary depending on breed and age of the rabbit but the albumin fraction should be approximately 60%. Low albumin may indicate liver disease as the liver is responsible for albumin production.

If diarrhoea is present an examination of the faeces under the microscope is advised.

Diagnostic imaging

Diagnostic imaging such as ultrasound is also advised. Hepatomegaly should be suspected during ultrasonography if an increased distance between the stomach and diaphragm is seen, an increased extension of the liver ventral to the stomach and if the liver margins appear rounded.

Radiographic signs of significant liver enlargement include: caudal projection of the liver beyond the rib cage margin, caudal displacement of the stomach.

Liver biopsy

Some cases may require biopsy of the liver to achieve a definitive diagnosis. It is important to fully assess the

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Figure 2.89a A mild fatty liver, there is infiltration of fat but it is not as obvious. The edges of the lobe are still sharp.



Figure 2.89b A rabbit with moderate fatty liver, there is obvious fat infiltration of the liver and the lobes are swollen but the edges of the lobes are still sharp.



Figure 2.89c A rabbit with a severe fatty liver, there is very obvious fat infiltration of the liver, the lobes are swollen and the edges of the lobes rounded. The liver was friable.

clotting factors before performing. Biopsy can either be performed under deep sedation via ultrasound guided percutaneous or under general anaesthesia using a punch, guillotine suture, or wedge biopsy technique. Regardless of technique used, positioning the rabbit in sternal recumbency afterwards uses the weight of the liver to aid in compression of the biopsy site which decreases the risk of haemorrhage. Monitor the rabbit for signs of haemorrhage for several hours post procedure.

Hepatic lipidosis/fatty liver syndrome

Any condition that causes a rabbit to stop eating can quickly result in fatty liver syndrome. Essentially, when there is no food intake there is a negative energy balance so the body moves fat from storage into the blood and transports it to the liver to make energy.

Overweight rabbits are at a higher risk simply because they have a higher level of fat storage. The fat from the blood gets absorbed into the liver, the hepatocytes swell which causes the liver to become rounded and more fragile (think blancmange). The liver may be friable and it can be damaged by even the gentlest examination of the abdomen causing an internal bleed (Figures 2.89a–c).

Liver lobe torsion

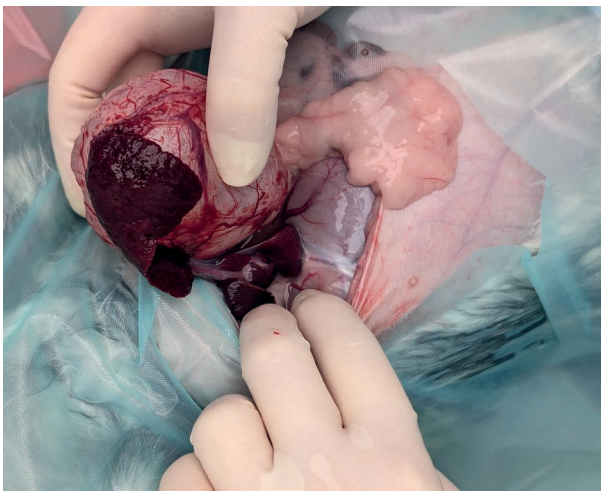
Liver lobe torsion is an increasingly recognised condition in pet rabbits. See previous section on liver lobe torsion.

Liver cysts

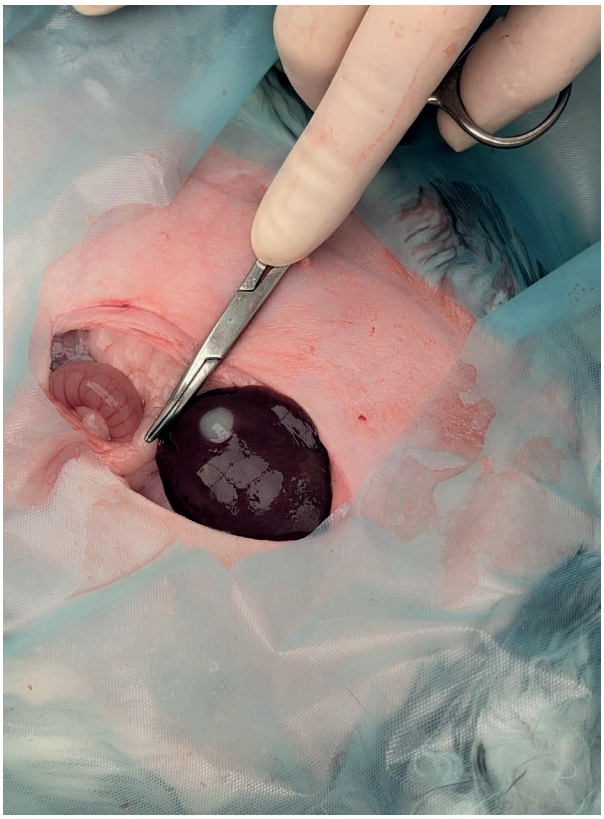
Rabbits can act as an intermediate host for the dog and fox tapeworms, *Taenia serialis* and *T. pisiformis*. Larve (*Coenurus serialis* and *Cysticercus pisiformis*) can form large cysts in the liver or the mesentery which can be felt during a clinical examination. Unless these are very large

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there may be no clinical signs for owners to tell this is an issue but may be felt during routine clinical examinations. The cysts may contain focal mineralisation which can be seen on radiographs. Treatment is surgical removal and praziquantel 5–10 mg/kg PO repeated in 10 days (see Figures 2.90a–c).



Figures 2.90a Intraoperative photo of removal of a large liver cyst. Credit: Dr Laura Cawley



Figures 2.90b Intraoperative photo of same rabbit with a smaller cyst in another liver lobe.Credit: Dr Laura Cawley



Figures 2.90c Both cysts successfully removed. Credit: Dr Laura Cawley

Liver coccidiosis

Numerous species of coccidia (see section on coccidia) can infect the rabbits' intestine but *Eimeria stiedae* invades the liver. *Eimeria stiedae* is seen more commonly in intensive breeding establishments but may be seen in individual pet rabbits.

The clinical signs vary (depending on the severity of the infection, the age of the rabbit, and the immune status of the rabbit) from inapparent through to depression, anorexia, emaciation, abdominal distension, diarrhoea, jaundice, and occasionally death.

Microscopy of faeces will show numerous ellipsoidal unsporulated oocysts. They are smooth, colourless, and have a two layered wall with a centrally positioned zygote. Biochemistry and haematology is recommended as significant liver disease may be present.

Treatment includes supportive care (see below) and either trimethoprim sulfamethoxazole 30 mg/kg q12hrs or toltrazuril 25 ppm in drinking water or 25 mg/kg PO q12hrs for 2 days, repeated in 5 days, to control the coccidia until immunity develops. Owners should be advised that in severe cases, despite treatment the prognosis may be grave.

Liver neoplasia

Primary hepatic neoplasia reported includes lymphoma, bile duct adenoma, and carcinoma. Secondary metastatic

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disease is commonly seen with uterine adenocarcinoma. The prognosis is usually grave as surgery is generally not performed. It has been suggested that chemotherapy may be tried in cases of lymphoma, but the immunosuppressive effects of chemotherapy may precipitate other conditions.

Treatment of liver disease

Other than torsion or cysts which require surgery, the rest of the treatments are very similar. Specific conditions such as coccidia infection or abscessation will require additional specific treatments.

Hospitalisation

IV fluids are often required to maintain hydration and correct any electrolyte imbalances. Assisted feeding will help prevent or reverse hepatic lipidosis, mucosal atrophy, and GI stasis. As anorexic rabbits do not eat their caecotrophs, either add their caecotrophs to the syringe feed slurry or consider vitamins such as B and K as their normal source of those vitamins would be from their caecotrophs and they may become deficient otherwise.

Offer timothy hay, fresh dandelions, fresh grass and rabbit's own food. Minimise stress by ensuring out of sight and smell of predators. Admit the bonded companion and provide a hide.

Medications

Prokinetics which can be given include metoclopramide, cisapride, ranitidine, and domperidone. It is advisable to continue prokinetics until the rabbit is eating and passing faeces.

Supportive liver supplements are advisable which usually contain antioxidants such as S-Adenosyl methionine (SAMe) and silymarin 50 mg/kg q 24hrs as they have been shown to have a protective function.

Euthanasia

End of life conversations can be a difficult area to broach with some clients. Occasionally owners will wait for us to broach the topic as they are 'worried' if they do so we will think they are not dedicated enough. Reassuring clients that sometimes we need to love our pets enough to let them go when the time is right can be invaluable.

Many owners will want to stay with their rabbit during this procedure and the process should be fully explained including possible events such as sudden jumping, the need to change veins or gasping. I administer a pre-med (e.g. medetomidine/butorphanol injected SC or IM), once the rabbit has fully relaxed, I will use the lateral saphenous vein by choice, to inject the pentobarbitone, to allow the owners to stroke the rabbits head. In some cases, I will burrito the rabbit first.

I advise allowing the bonded cage mate to see its companion after euthanasia. I advise clients that I believe it allows the cage mate to accept that its companion will not return. Over the years, many owners have reported to me they felt the cage mate was looking for its companion constantly after losing them and certainly anecdotally this is reduced when they get the opportunity to 'say goodbye'. Whether this is purely anthropomorphic or not is up for debate, however it demonstrates to your owners that you care and you listen to their concerns.

Many owners will opt for private cremation with return of their pets' ashes and all options that your practice offers for dogs and cats should also be made available to your rabbit clients. I offer a lock of fur. I send sympathy cards if I know the patient and owner well.

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3 Guinea pigs

Guinea pigs are an extremely popular pet and are often kept by adults rather than only being a child's pet. They are hystricomorph rodents with continuously growing incisors and cheek teeth, a long gestation period (65 days – the same as a bitch!) and birth precocial young who start to eat solid food from as young as 2 days old. Their average lifespan is 4–8 years, and the oldest guinea pig I have treated was 9.5 years old. Owners bond very tightly with their guinea pigs and they are frequently kept indoors. The domestic guinea pig (*Cavia porcellus*) is thought to have the Montane guinea pig (*C. tschudii*) as its main ancestor as the domestic guinea pig does not exist in the wild. Guinea pigs were introduced to Europe in the 16th century and were kept as meat production animals or sacrifices for approximately 5,000 years.

Guinea pigs evolved to live in the Andes of South America occupying terrain such as grassland savannahs, mountainous regions, and brushland. They evolved to eat grass, leaves, plants, and flowers, and although they do not burrow, they will rest in burrows of other animals, rocky crevices or hide in vegetation.

As a prey species, they have evolved to hide disease and pain to prevent predation. Guinea pigs appear to hide illness much more effectively than rabbits do, leading to many owners perceiving that their guinea pig either suddenly got ill or died without any illness. Advising weekly weighing of their pet is advisable as changes in body weight can be an early indicator of disease. Interestingly, one study showed that owners thought skin disease needed a more urgent same day appointment than lethargy or anorexia in guinea pigs which highlights the need to educate our clients on what constitutes an emergency in guinea pigs.

> Fawcett (2011) stated that the leading cause of morbidity and mortality in guinea pigs was poor husbandry and poor diet. It stands to reason that with good husbandry and a balanced diet they will rarely get ill as very few conditions are infectious. They are exquisitely sensitive to the effects of stress and pain.

Educating owners on husbandry and how to improve the welfare of their pet (e.g. environmental enrichment) should be part of a standard veterinary visit. New pet owners may be more likely to seek advice but every day is a school day and we need to be aware that owners may confuse love and dedication to their pet with appropriate welfare, this is where husbandry registration forms are invaluable to allow us to have a non-confrontational overview of how the pet is being kept.

Companionship

As a prey species, they evolved being a food source for many predators. If you have evolved to be 'food' you will feel safer if there are two or more sets of eyes looking out for predators than if you are on your own. If you have evolved to live in a single species colony then you have an innate requirement for conspecific companionship. *Put simply: guinea pigs need guinea pig companions*. Research has shown that *conspecifics reduces stress, this is important as chronic stress is implicated in common pathologies*.

A minimum of two guinea pigs should be kept together although some owners choose to keep a higher number. A single male can be kept with several females, although neutering of the male should be performed if breeding is not desired. The pubic symphysis of female guinea pigs ossifies at approximately 5–7 months of age and it is recommended if breeding is planned to ensure the first pregnancy occurs before this age. This is due to large precocial pups that guinea pigs' birth, closure of the pubic symphysis results in a narrow birth canal which may increase the risk of an emergency caesarean section being required.

A common myth is that male guinea pigs cannot be kept together. Male guinea pigs are more likely to live peacefully if they are raised together from a young age, although if one dies an older boar will more likely accept a young boar than an adult (as young boars are more likely to be submissive). This setup will only work if there are no female guinea pigs also in the household.

Rabbits do not make suitable companions for guinea pigs. We all know people who have 'gotten away with it' and claim that their rabbits and guinea pigs live together in harmony and love each other. In reality: guinea pigs and rabbits evolved to live in different niches, have different dietary requirements, different behavioural needs, and different communication methods. Rabbits also harbour *Bordetella*

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bronchiseptica as a normal nasal commensal which can cause fatal bronchopneumonia in guinea pigs. The powerful kick of rabbits can result in fractured ribs if aimed at the guinea pig and bullying by either species is possible.

Enclosure

Enclosures need to be designed to provide an outlet for normal behaviour. Guinea pigs do not like wide open spaces (which makes sense when you are a prey species whose main defence is to run away) and they will tend to huddle next to the wall or in a tunnel. This also explains their tendency to move around the periphery of an enclosure rather than cross the middle. A fully enclosed area is a requirement in the enclosure to reduce stress, ideally with two entrances/exits.

Regardless of whether guinea pigs are kept indoors or outdoors, *the minimum enclosure footprint size is: 1.5 m × 1 m with access to an exercise area of 2 m × 1 m*. Most commercial cages are far too small, and larger enclosures resulted in demonstration of more positive behaviours in various studies. The floor of the enclosure should be solid as wire-based floors contribute to foot problems.

Outdoor living areas can be made out of converted sheds with an outdoor run attached. Indoor enclosures can be made using puppy pens to increase flexibility of size and shape. Their wild ancestors had large home ranges for their size; quoted as being up to 2.5 km^2.

Several tunnels or hides should be provided to reduce stress and provide multiple resting areas. *Guinea pigs are known to create a mental map of their hiding spots (to reduce risk of predation) therefore owners should be advised that rearranging their hides during cage cleaning may be a source of stress for their pet*, as they have to relearn the locations of their hiding spots.

Areas with large amounts of hay will provide environmental enrichment allowing them to make hides within the hay, stomp on it as well as consume it. A thick layer of substrate such as: scent free shavings (some guinea pigs will be allergic to any type of shavings), wood- or paper-based pulp, or recycled paper (that is used for horse bedding) will help absorb any urine, and provide support for their relatively small feet. I do not recommend straw as injuries to their eyes and male guinea pig genitalia is more commonly seen with straw-based substrates. Some owners, especially if the guinea pigs are kept indoors, opt for layers of fleece-based bedding or vet bed, rather than a loose substrate. I advise they still use a lot of hay as a substrate on top of the fleece.

Cardboard boxes and fruit tree branches (not cherry) with the bark left on can be provided for chewing opportunities (they will often eat the bark) although it should be noted that *guinea pigs use their incisors more as scissors rather than true gnawers like rats*. Various foraging toys can be easily and cheaply created with a little imagination.

Thought should also be given to the position of the enclosure. Outdoor enclosures must be well shaded as they are very prone to heat exhaustion and *will experience heat stroke with temperatures over 26 °C (78.8 °F)*. The ideal temperature range is 18–22 °C (64.4–71.6 °F). Indoor enclosure position needs careful consideration too. Many owners do not realise how sensitive guinea pig ears are. *They can hear well outwith our audible range and being kept next to electronic devices can cause immense chronic stress*. Guinea pigs can hear the high-pitched electronic hums produced by electronics such as fridge freezers and televisions/stereos. The vibrations and noise produced by washing machines will also have a negative impact. *Chronic stress leads to immunosuppression and then even commensal bacteria can become opportunistic pathogens*. I have seen cushingoid symptoms in a colony of guinea pigs (pot belly, thinning skin, easily epilated fur, alopecia, massive adrenal glands) due to chronic stress which did resolve when the stressor was removed.

Guinea pigs are generally crepuscular but feed regularly throughout the day and night. They are messy, indiscriminately defecating and urinating throughout their enclosure (including food and water bowls). They do perform caecotrophy and most guinea pigs will consume these directly from their anus. Obese or heavily pregnant guinea pigs will consume the caecotrophs from the floor of their enclosure. *Caecotrophy is normal and although can be distressing for owners, educating owners that they gain vitamin B_{12} and folic acid among other nutrients from performing this behaviour can help the owners understand*.

Diet

Guinea pigs develop strong food preferences early in life and can become neophobic with food types as adults. Offering a wide variety of hay, greens, vegetables, and several brands of guinea pig nugget to young guinea pigs will make feeding as adults much easier. *They do not cope well with sudden changes to diet or water provision and any changes should be done slowly*. They have been known to fatally dehydrate themselves if presented with an unfamiliar source of water suddenly.

The diet advised for captive guinea pigs is 85% forage (hay and grass), 10% dark leafy greens and vegetables (e.g. broccoli), 4% good quality concentrates (nuggets or pellets – not muesli as this has been shown to contribute to dental disease), and 1% healthy treats (small quantities of fruit, strawberries are a particularly good source of vitamin C).

Many owners are still unaware that hay should be fed ad lib and that the absolute *minimum consumption should be the guinea pigs own body size in hay daily*. Another consideration is type of hay and which cut. It is surprising how many owners really do not know about different types of hay.

While one guinea pig may prefer timothy hay, another may prefer meadow hay and yet another may prefer orchard. The quality/taste of hay will vary throughout the year depending on when it was harvested. This is due

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to the fact plant composition varies throughout the year. Therefore, it is understandable that guinea pig's preference may also vary throughout the year. Alfalfa hay should only be fed to young, growing animals or pregnant sows.

> Educating owners on the available types of hay and potential reputable sources, e.g. Hop to Forage (www.hoptoforage.co.uk), can help with – 'Oh, my guinea pig will not eat hay.'

Wild guinea pigs spend over 80% of their waking time foraging – compare this to domestic guinea pigs. Using bedding hay and scattering the daily allowance of concentrates through the hay provides environmental enrichment. As humans we mainly eat our food from plates or bowls and we like to see our pets do the same. Unfortunately, the bowls made for guinea pig food are massively big when you compare them to the recommended portion size. This leads to inadvertent over feeding in many cases as owners will often completely fill the guinea pig bowl with concentrates.

Fresh grass should be offered daily. Many owners claim to feed grass daily but on further questioning they may be referring to dried grass. If guinea pigs belong to owners without a garden, encourage the owners to grow turf in trays, which can be rotated allowing grazing while the other trays regrow.

Guinea pigs evolved eating a diet that varied throughout the year depending on seasonality. We should try to replicate this in captivity to encourage more natural behaviour and for a list of safe greens they can buy or forage, owners can be directed to websites such as: https://www.pdsa.org.uk/pet-help-and-advice/looking-after-your-pet/small-pets/your-guinea-pig-s-diet#faqs or https://www.guineapigmagazine.com/what-weeds-can-you-feed-to-guinea-pigs/

Vitamin C

Guinea pigs cannot produce their own vitamin C and require 10–30 mg/kg/day for maintenance, ill or pregnant guinea pigs have a higher vitamin C requirement. Good quality guinea pig nugget/pelleted concentrates contain added vitamin C but as vitamin C is a very labile vitamin, the vast majority of it has deteriorated ninety days post-manufacture. Vegetables such as dark leafy greens, broccoli and orange or red bell peppers are high in vitamin C and should be given daily. *Guinea pigs that are offered vegetables high in vitamin C on a daily basis have been shown to be healthier and have less morbidity*. Carrots will also increase environmental enrichment by providing a suitable outlet for incisor wear. Many owners supplement vitamin C in the drinking water but it will break down to negligible amounts within two hours of exposure to light and therefore should be discouraged as a method of supplementation. *A supplement that is widely available to veterinary clinics and marketed as being suitable for use in the water for guinea pigs, when I challenged the stability of their product they confirmed that it may be negligible by 6 hours which means owners would need to change the water four times daily to ensure adequate vitamin C levels. Even marginal hypovitaminosis C will predispose guinea pigs to almost any disease state*.

Guinea pigs fed on rabbit pellets will develop scurvy (see Scurvy section) and may develop metastatic calcification of organs due to the high vitamin D levels in rabbit diets. Guinea pigs need guinea pig concentrates.

Water

Wild guinea pigs frequently ingest grass and plants that are soaked in dew or rain, thereby consuming more water while eating. In captivity the main food source is hay, which is a great source of fibre and nutrition but is extremely low in water content. Guinea pigs that eat a lot of fresh grass or greens will drink less than those who do not. However, neither group is likely to consume as much water as the wild guinea pigs. The maintenance water requirement is 100–200 ml/kg/day.

Studies have shown that *unlike rabbits and chinchillas, guinea pigs prefer to consume water from water bottles*. The action of drinking from the water spout mimics their natural chewing pattern and has been *shown to actually increase the volume of water consumed*. It is also thought to provide environmental enrichment. As guinea pigs display the behaviour of consuming food, then drinking while having food still in their mouths, the resultant slurry can sometimes be blown up the nozzle of the water bottle while drinking. This can lead to blockages and *so the bottle should be regularly checked to ensure it is working correctly. Guinea pigs do not show the usual adaptations required to concentrate urine and conserve water, therefore failure to drink will quickly result in life-threatening dehydration*. Ideally give both a bowl and a bottle to ensure constant access to water.

Guinea pigs *absorb all calcium from ingested food and excess levels are excreted via the kidneys in the urine*. Inadequate water consumption can lead to calcium sand precipitating in the bladder leading to 'sludgy' urine, cystic calculi, renal calculi, and potentially life-threatening renal disease. *I believe that 'sludgy' urine etc. is not due to dietary intake of calcium but rather it is down to the fact that wild guinea pigs have a much higher water intake*. Many domestic guinea pigs have skeletal pain due to osteoarthritis, or hypovitaminosis C. They are therefore reluctant to pass urine as frequently and may not be as mobile, leading to an increased risk of urine becoming concentrated and supersaturated, in turn leading to further precipitation.

Encouraging natural diuresis by providing cooled dandelion or nettle teas as drinking water will promote further fluid intake. Bottles of plain water should also be provided until owners are sure that their guinea pigs are readily consuming the flavoured water. Fresh dandelions can also be given daily to promote natural diuresis.

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Ultraviolet (UV) light

Guinea pigs are mainly crepuscular, but they can regularly be seen basking in the sun. Indoor only guinea pigs, or guinea pigs who rarely are given outdoor access, benefit from a source of UVA/B.

Many of us associate the sun with 'feeling good'. This is due to the release of beta endorphins triggered by exposure to UV light. Improved skin immune function and conversion of vitamin D to the active form D_3 are also benefits provided by provision of UVB. Dermal conversion of vitamin D to D_3 is self-regulating and therefore it is impossible to overdose!

We know guinea pigs evolved to live in South America, in particular the Andes and that these areas are exposed to high levels of UV light. Providing an artificial sunbeam (UVA/B and heat) may give indoor guinea pigs physical benefits, mental health benefits and allow more expression of normal species-specific behaviour. It has already been proven to increase endogenous vitamin D levels (safely) and Watson *et al*. (2014) also showed no adverse effects in guinea pigs exposed to appropriate levels of UVB over a 6-month period. Day night ratio should be 10:14 or 12:12.

Sexing

Guinea pigs are relatively easy to sex by looking at the shape of the genitalia. Both sexes have a pair of nipples.

Female

Female guinea pigs have a Y shaped genital region (Figure 3.1a & b). The anus sits at the base of the Y, the vulvar opening (covered with a membrane unless in oestrus) is at the junction of the Y and the arms of the Y surround the slit shaped urethral opening.

Male

Male guinea pigs have what has been described as two circles or an i shaped genital region. In sexually mature entire boars there are two large testicles, one on either side and the inguinal canals are open (Figure 3.2). The top circle (or the dot of the i) is the prepuce and the bottom circle (or line) is the anal sac. The penis can be extruded with gentle pressure.



Figure 3.2 A male guinea pig.

Preventative health care

There are no vaccinations available for guinea pigs and routine worming or ectoparasite control is unnecessary. *Owing to their amazing ability to mask disease, 6 monthly routine check-ups are advisable* but many owners will only present their guinea pig when there is a problem.

Nail care

Regular nail trimming may be required depending on the substrate type. It is common for their front toes to deviate laterally and curl to the side as they age. Keratin overgrowths are common on the lateral aspect of the feet and are commonly referred to as spurs (Figure 3.3). These





Figure 3.1 Female guinea pig.
a The female guinea pig genital anal region may look like a V shape as in this image or Y as in 3.1b.
b The female guinea pig genital anal region may look like a Y shape as in this image or V as in 3.1a.

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Figure 3.3 Lateral spur on front foot.

should be trimmed to prevent trauma associated with catching and tearing on things. The actual growths contain no sensitive material or blood supply and can be trimmed with a sharp pair of nail clippers. I prefer straight nail clippers for the spurs and shaped nail clippers for the nails.

Neutering

Routine neutering of guinea pigs remains controversial within the exotic's world. Castration of boars to prevent unwanted litters is the most commonly performed elective neutering of guinea pigs in my practice.

Elective ovariohysterectomy or even very early ovariectomy is advocated by some due to the high incidence (100% in some studies but commonly quoted as 50–80% in others) of ovarian cysts. The vast majority of these cysts are not hormonally active and at worst, will cause clinical signs due to the size they get to. Follicular cysts are hormonally active and are responsible for the classical clinical signs associated with ovarian cysts, i.e. symmetrical bilateral flank alopecia, crusting of the nipples.

Uterine pathology can also occur, especially with follicular cysts (they are hormonally active) and is more common in guinea pigs over three years of age but does not have as high an incidence level.

In my experience *very few guinea pigs show clinical signs* and I struggle with the ethics of advising an elective surgical procedure as a blanket approach for a species that has been shown to have a 22% occurrence of follicular cysts. *See the section on reproductive medicine for further information to form your own opinion*.

Hospitalisation

As a prey species, extremely sensitive to stress caused by the sight, sound and smell of predators, there are several factors we need to take into consideration. Stress is immunosuppressive for any species, but *guinea pigs are particularly sensitive to stress*, and as such we aim to minimise it.

In an ideal world we would all have prey species wards. For many practices that is not the reality. Other options include setting up pens/collapsible kennels in a quieter area of the clinic or utilising the cat ward provided the kennels are not facing each other.

The ward should be kept warm, but care should be taken *not to heat above 22 °C (71.6 °F)* as above this temperature heat stroke becomes a real risk. I aim to keep my ward between 18–20 °C (64.4–68 °F) as this ensures the animal is not having to expend energy to stay warm. A digital max/min thermometer and hygrometer (purchased inexpensively from a well-known online retailer) allows me to monitor the temperature and the humidity within the room.

Prey species do not like absolute silence. In the wilderness there is always birdsong or other animal noises unless there is a predator around. For this reason, playing music at a low level is recommended. I use Pet Remedy (Pet Remedy Ltd) plug-ins in each of my wards to try and help mitigate stress while hospitalised.

How the kennel/pen is set up is also important. Slippery surfaces are stressful and can result in injuries. Lining the floor with paper and providing a deep bed of hay is a good choice. If the guinea pig is used to having blankets or towels at home, then they can also be used.

A hide should be given which can be a cardboard box or even half the front of the enclosure being covered by a blanket.

Check with the owner how they normally provide water. *Guinea pigs do not react well if their usual method of water delivery is suddenly changed and they can fatally dehydrate*. Fresh grass should be offered to every inpatient and can act as an appetite stimulant to many guinea pigs.

For elective procedures vets or front of house staff can request the owners bring their guinea pigs a 'packed lunch' of favourite and familiar foods. Owners often love this phrase and I have experienced some well packed and personalised lunch boxes! If the guinea pig is being admitted as an emergency, then I will ask the owner to bring in some of their normal food if it is not a food I already have. Familiar food is important even if it is not ideal, for example, muesli. Once the pet has recovered, that is the time to gradually change the diet to a more advisable one.

The bond between guinea pigs is scent based. Failure to admit the bonded cage mate can result in stress-induced anorexia (in either or both guinea pigs), the breakage of the bond, fighting, injuries, upset owners and it may be difficult to re-bond the guinea pigs. *I always advise admitting the bonded cage mate at the same time*. Owners are, in my experience, happy to bring the cage mate in once they understand why.

In cases where there are more than three bonded guinea pigs (three cage mates is my limit), I will ask the owner to bring in the one it spends the most time with. Scent swapping (rubbing each group with a clean towel then using the towel to rub the other group transferring the scent) to ensure both groups are marked with the communal scent can help ensure a smooth reintroduction at home.

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Hospitalisation sheets should be used, and the animals weighed twice daily.

Venipuncture sites

Wrapping the guinea pig in a towel (same technique that you use in rabbits, see Video 2.1) using EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) makes venipuncture much easier.

My vein of choice is the cephalic vein, both for intravenous (IV) catheter placement and blood sampling. (see Figure 3.4) I regularly obtain 2 ml samples from this site although I often use only a 23 g needle and allow the blood to drip directly into the blood collection tube. *Having your nurse massage the vein or 'pump' the vein by very slowly raising and releasing while you flex and unflex the carpus allows good flow of the blood. Ensuring you utilise gravity by holding the leg directed ventrally rather than horizontally also helps*.

As with more familiar species one vein is often bigger than the other, therefore I clip fur over the vein in both legs. I apply approximately 1 mm thick layer of EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) enough so there is a noticeably white patch, then I get the rest of my equipment organised. In my experience, EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) takes around five minutes to cause venodilation, some local anaesthesia and does not require a dressing to work. *Pick your catheter size judged on the size of the vein, not the size of your patient*. For most guinea pigs I will end up using a 22 g IV (blue in UK) catheter, but occasionally I have had to use a 24 g (yellow in the UK).

Placing IV catheters in guinea pigs is not that difficult! The skin is thick, and some clinicians find it easier to nick the skin with another needle prior to inserting the catheter. Winged catheters are much easier to tape in as you can bend the wings around the antebrachium. Dressing tape such as Micropore (3M) is applied proximal and distal to the cannula in exactly the same manner we would in our canine patients.

Elizabethan collars should not be used in guinea pigs. *Elizabethan collars make eating food more difficult (not what you want in an anorexic patient!), the eating of caecotrophs impossible, and are an unnecessary source of severe stress.*

Alternative sites for venipuncture are the lateral saphenous vein (Figure 3.5) or the jugular vein. I rarely utilise the jugular vein anymore as guinea pigs tend to find it stressful, they have short necks and their jugulars are relatively deep. *Many exotics texts describe anaesthetising the guinea pig and performing a cranial vena cava stick to draw blood. This carries the risk of death due to uncontrollable haemorrhage*. Laboratory vets and technicians have perfected this technique with very few bad outcomes, but they are performing this very regularly whereas clinical veterinary staff do not. Personally, I would not do this to one of my own guinea pigs therefore I will not do it to a client's. I have never needed to anaesthetise a guinea pig to draw blood. In highly stressed patients, midazolam may be advisable to reduce stress levels.

The maximum volume of blood that can be safely taken is 1% of their bodyweight. Therefore, in a 1 kg guinea pig we could safely take 10 ml which is more than we would ever need to. Many biochemistry analysers can run a profile on 0.3 ml of blood.



Figure 3.4 Cephalic vein is large in the guinea pig.



Figure 3.5 Lateral saphenous vein.

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Table 3.1 Guinea pig biochemistry.

BIOCHEMISTRY (Heatley & Russell, 2020)	REFERENCE RANGE (UK units)	REFERENCE RANGE (US units)
ALKP	0–418 IU/l	0–418 U/l
ALT	10–25 IU/l	10–25 U/l
AST	0–90 IU/l	0–90 U/l
AMYL	212–424 IU/l	212–424 U/l
GLDH	0–17 IU/l	0–17 U/l
GGT	0–13 IU/l	0–13 U/l
LDH	0–515 IU/l	0–515 U/l
CK	0–2143 IU/l	0–2143 U/l
LIPASE	0–152 IU/l	0–152 U/l
GLU	3.3–6.9 mmol/l	60–125 mg/dl
CHOL	0.52–1.11 mmol/l	20–43 mg/dl
TRIGYLCERIDES	0–1.4 mmol/l	0–145 mg/dl
BA	0–84.5 µmol/l	4.94 mg/dl
TBIL	5–15 mmol/l	0.3–0.9 mg/dl
UREA	3.2–11.2 mmol/l	9–31.5 mg/dl
CREA	53–194 umol/l	0.6–2.2 mg/dl
TP	46–62g/l	4.6–6.2 g/dl
ALB	21–39 g/l	2.1–3.9 g/dl
GLOB	17–26 g/l	1.7–2.6 g/dl
CA	1.32–3 mmol/l	5.3–12 mg/dl
PHOS	0.97–3.87 mmol/l	3–12 mg/dl
K	4.5–8.9 mmol/l	4.5–8.9 mEq/l
Na	132–156 mmol/l	132–156 mEq/l
FRUCTOSAMINE	134–271 µmol/l	
T4	29.6–45 nmol/l	2.3–3.5 ug/dl

Biochemistry and haematology profiles can be run 'in-house' using our usual mammalian profiles or sent to an external laboratory. Both options have pros and cons. The main pro for running in-house is that you obtain your results quickly whereas the main con is that you have to interpret whereas many labs will provide interpretation upon request.

See Tables 3.1 and 3.2 for reference ranges for biochemistry and haematology.

Drugs contraindicated in guinea pigs

If in doubt, always check the formulary as it will often tell you whether it is safe to use a drug in any given species. My favourite formulary is James Carpenter's *Exotic Animal Formulary* now on its sixth edition (Carpenter & Marion, 2022).

Antibiotics to avoid in guinea pigs include penicillin, amoxicillin, clavulanated amoxicillin, ampicillin, clindamycin, lincomycin and erythromycin. These drugs, especially if given orally, will cause a fatal antibiotic induced gut dysbiosis. The last three mentioned antibiotics are not safe to be given by any route.

> Penicillin and amoxicillin can be given safely by subcutaneous injection, although I always warn the owner there is a small risk. We need to remember that if the drug leaks onto the coat, it needs to be thoroughly washed off as grooming equals oral ingestion!

Species-specific anatomy/behaviour

Guinea pigs are hystricomorphs with elodont – hypsodont dentition, (Elodont: continuously growing throughout life and hypsodont: long anatomical crown compared to the root). They have two incisors in the maxilla and two in the mandible. Dental formula: 2 × (I 1/1; C 0/0; P 1/1; M 3/3) = 20.

The *tight cardiac stomach sphincter prohibits vomiting or eructation*. They are hindgut fermenters with a large caecum. They have very long gestation periods (65 days) and birth precocial young.

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Table 3.2 Guinea pig haematology.

HAEMATOLOGY (Heatley & Russell, 2020)	UNITS	REFERENCE RANGE
Red blood cells	10^{12}/l	4.5–7
Packed cell volume	%	37–48
Haemoglobin	mmol/l (g/l)	6.82–7.14 (110–115)
Mean corpuscular volume	fl	80.3–89.1
Mean corpuscular haemoglobin concentration	mmol/l	18.1–19.7
Mean corpuscular haemoglobin	fmol/l	1.5–1.7
Platelets	10^9/l	250–850
White blood cells	10^9/l	7–18
Differential blood count (absolute values)		
Monocytes	10^9/l	0–0.657
Lymphocytes	10^9/l	1.401–10.665
Neutrophils	10^9/l	0.899–5.097
Neutrophils, bands	10^9/l	0–0.072
Eosinophils	10^9/l	0–1.563
Basophils	10^9/l	0–0.106

Using a towel or a blanket on the table to reduce the slippery nature of the surface makes guinea pigs feel safer and they are less likely to struggle.

Guinea pigs have relatively small feet compared to body size.

Male guinea pigs have open inguinal canals. Failure to appreciate this and take steps to close the canal during castration can lead to inguinal herniation of abdominal organs.

Guinea pigs do not show the usual adaptations required to concentrate urine and conserve water, therefore failure to drink will quickly result in life-threatening dehydration.

Guinea pigs hide illness and pain. They seem to be even better than rabbits at hiding illness leading owners to believe it was a sudden death. Again, from an evolutionary point of view this makes complete sense because if you show illness or weakness, you get eaten! So, by the time a guinea pig looks overtly ill or painful, they literally no longer care if they live or die. Many owners underestimate this and unfortunately many of us, the veterinary profession also underestimate it.

Pain in guinea pigs

One of our jobs as veterinary professionals is to actively look for pain. In Chapter 1 we discussed that all vertebrates have the same anatomical and physiological pain pathways.

Guinea pigs show very subtle behaviour changes associated with post-operative pain and studies have shown that in rodents the most significant signs of pain occur within the first 12–24 h post-operatively, therefore *application of a local anaesthetic at the site of the incision could greatly reduce post-operative pain*. Abdominal contraction, back arching, twitching, and weight shifting at rest are all signs associated with post-operative pain. Guinea pigs will often freeze when in the presence of an observer so these signs can easily be missed if video surveillance is not an option.

Acute and surgical pain is easier to appreciate but what about chronic pain especially arthritic pain?

Chronic pain negatively impacts quality of life severely. It causes chronic maladaptive stress, activation of stress hormones, sleep disruption, immunosuppression, and reduces social interactions (which for an innately social species further exacerbates stress levels). Rabbits, rats, and mice have grimace scales that can be utilised to pain score but unfortunately the same cannot be said for guinea pigs, such is their ability to hide pain. Stress and fear may increase the degree of pain perceived by an animal and steps should be taken to minimise these emotions.

The first signs of pain commonly seen in guinea pigs are: rough coat (from reduced grooming), a hunched body posture (slight arch of the back often thoracolumbar), a slower possible shuffling gait, pacing/restless when normally would be sleeping, abnormal sleeping position (stretched out instead of tucked), self-isolation from the group, personality change (previously friendly now aggressive or vice versa), drooling (if oral pain), anorexia and associated weight loss. In today's busy world these signs are commonly missed by owners until they become very obvious.

Ensuring that during our history taking we target our questions to include mobility can help. We already do that

with cats as they tend to show more behavioural adaptations to osteoarthritic pain compared to dogs, but we often forget to do that with guinea pigs (stop hitting your panic button!) *Recognition and alleviation of pain are an essential component of every animals' care*.

How many veterinary professionals genuinely perform the exact same clinical exam in a guinea pig that you do in a dog? How many do a full skeletal exam, spinal palpation, and limb range of motion in every guinea pig patient? If you do not do this – why not? *Osteoarthritis and spinal pain is massively underdiagnosed in guinea pigs simply because we do not always think to look for it*.

Placed on a towel for security, the neck flexion tests are performed exactly the same as in more familiar species. Extending the forelimbs one at a time allows you to differentiate if more pressure was required to extend one limb therefore identifying pain. View the guinea pig from above, does the guinea pig lean more to one side (taking pain off the sore side) or is a limb abducted?

Spinal palpation requires the exact same level of pressure is needed that you would use on a Jack Russell terrier. Failure to perform correctly will lead to failure to spot pain. Remember that the thoracic spine runs between the scapula and needs palpating too!

Guinea pigs may well vocalise if you palpate a painful area but sometimes, they vocalise because they object to being handled. If the vocalisation is repeatable when the exact same area is palpated and especially if they vibrate when you press on an area of their spine then we can be confident it is pain. Back pain can sometimes be confused with abdominal pain and vice versa (same as more familiar species).

Self-injurious behaviour (e.g. chewing through skin) can be seen especially with chronic neuropathic pain.

Osteoarthritis In guinea pigs

Owners can be shocked or initially disbelieving of a diagnosis of osteoarthritis in a guinea pig of less than 2 years of age, yet I frequently diagnose osteoarthritis in that age group. How I explain this seemingly strange occurrence is discussed below.

Osteoarthritis is a degenerative condition affecting the cartilage and bones of a joint. It is commonly referred to as the wear and tear arthritis. Wild animals do not live as long as captive animals (many die before even reaching reproductive age) due to a combination of pressures such as: predation, disease, or trauma (cars etc.).

In captivity (protected from such pressures) wild animals live longer. Therefore, the evolutionary pressures on prey species have selected for fast moving individuals that breed at a young age, and produce many young.

From an evolutionary point of view, by the time a guinea pig is 2 years of age it is already skeletally old. To me it makes complete sense why 'young' domesticated guinea pigs develop wear and tear arthritis, as evolutionary pressure did not select for a robust skeleton designed for longevity. The added pressures that domestic guinea pigs face such as: enclosures that limit proper movement (sedentary guinea pigs develop more severe stifle osteoarthritis), lack of UVB light, hypovitaminosis C, inappropriate substrate (type and depth), and obesity, further compound the damage and pain caused by the wear and tear arthritis.

Once this is explained to owners, I find they completely understand why this has occurred in their beloved pet and they are much more willing to work with me. Companies such as Oxbow now produce joint supplement treats, which may provide some support to the joints. Reports are contradictory as to whether it is beneficial in naturally occurring osteoarthritis, although evidence does exist that it may be beneficial in reducing cartilage swelling, and reduce pain if given early in the course of the disease. Ultimately, it should cause no harm, may work, and many owners like to give supplements.

Chronic pain associated with osteoarthritis is a major welfare concern. It is underdiagnosed in practice mainly due to the guinea pigs astounding ability to hide pain. Keeble *et al.* (2017) reported 100% radiographic prevalence of osteoarthritis (albeit in a small study of 23) with the stifle joint being the most commonly affected. There is a higher prevalence of osteoarthritis in guinea pigs over 2 years of age, but it can occur at a relatively young age too. I frequently diagnose osteoarthritis both during clinical exams and radiographically from 1 year of age.

Pain associated with osteoarthritis can be found when a full skeletal exam is performed (see section on examination). In my experience guinea pigs often are presented for secondary reasons that develop due to the pain associated with osteoarthritis, such as: weight loss, perineal faecal/urine staining, vocalisation while toileting, stranguria/dysuria, and pododermatitis.

Owners find vocalisation during toileting particularly distressing and often worry that there is a problem in the bladder. There may indeed be an issue in the bladder, but equally the pain caused by the posture required to urinate and defecate can result in vocalisation during these behaviours. I find repeatable pain on lumbar, sacral, hip or stifle manipulation in these cases.

Pain in these areas is the most common cause of perineal faecal/urine staining. Inability to adopt the correct posture to pass urine and faeces results in both becoming stuck to the fur and may result in scald of the skin.

Guinea pigs *absorb all calcium from ingested food and excess levels are excreted via the kidneys in the urine*. Inadequate water consumption can lead to calcium sand precipitating in the bladder leading to 'sludgy' urine, cystic calculi, renal calculi, and potentially life-threatening renal disease. *I believe that 'sludgy' urine etc. is not due to dietary intake of calcium but rather it is down to the fact that wild guinea pigs have a much higher*

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water intake (wild guinea pigs frequently ingest grass and plants that are soaked in dew or rain, thereby consuming more water).

Reluctance to pass urine until absolutely necessary will result in supersaturation of the urine with calcium crystals. Precipitation of the crystals results, irritation of the lining of the bladder with subsequent development of cystitis and increased risk of urolith formation can often be traced back to osteoarthritis or spinal pain.

Encouraging natural diuresis by providing cooled dandelion or nettle teas as drinking water will promote further fluid intake. Bottles of plain water should also be provided until owners are sure that their guinea pigs are readily consuming the flavoured water. Fresh dandelions can also be given daily to promote natural diuresis.

Osteoarthritis of the temporomandibular joints will cause secondary dental disease due to pain caused by chewing. Osteoarthritis of any joints may cause secondary dental disease due to a decrease in appetite, which as it happens slowly, will often not be noted by the owner. *In our clinic we have noticed a sharp decline in dental disease cases since a more unified approach to multimodal analgesia was adopted*.

Radiography can be utilised to investigate osteoarthritis or spinal conditions in exactly the same way as more familiar species (Figure 3.6).

Treatment of osteoarthritis

Multimodal analgesia should be started (see section on pain management). Urolith formation can be easily diagnosed in most cases via a conscious dorsoventral radiograph (Figure 3.7). Most uroliths require surgical removal in guinea pigs (see section on uroliths), although small ones may pass naturally.

Husbandry changes

Lifestyle changes can make a big difference to quality of life. Ensuring the floor of the enclosure and exercise areas are non-slip and padded will reduce concussive injuries. Yoga mats or children's play tiles can be useful. Avoid using ramps in the enclosure, reduce weight slowly in obese individuals and increase the daily vitamin C in the diet via fresh foods (also increases water intake). Ensure regular short periods of exercise to prevent stiffness of the joints and gradually increase muscle mass. Scatter feeding concentrates through the hay will gently increase exercise. *Ensuring deep layers of substrate will help cushion the joints and providing heat discs, e.g. Snuggle Safes, during cold spells will reduce pain associated with the cold*.



Fig. 3.6 Severe osteoarthritic changes in both stifles but the right is worst.

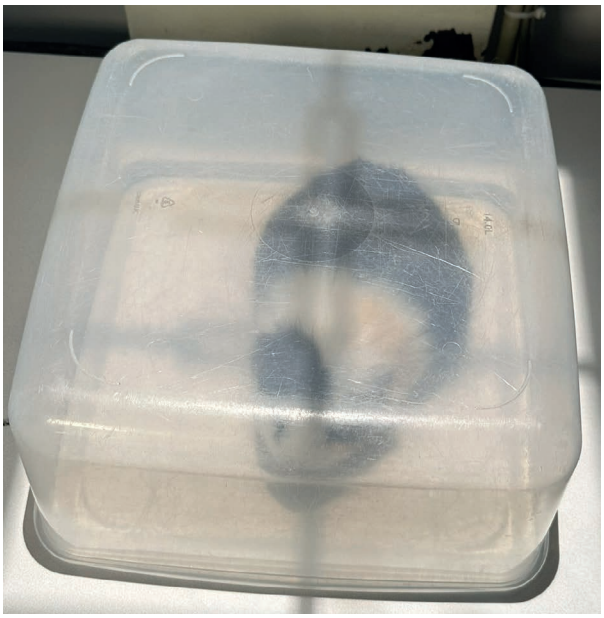


Fig. 3.7 An upside-down plastic tub may be placed over a conscious guinea pig for a dorsoventral radiograph.

Analgesia in Guinea Pigs

Consideration of the type of pain we are looking to control will lead to logical choices of the type of analgesics we select. *Multimodal analgesia is recommended* just as in more familiar species as it has been shown to provide better relief from pain in guinea pigs and other species (Oliver *et al.*, 2017).

Guinea pigs have a much higher metabolic rate than our more familiar species and as such their required doses will be much higher. *I manage owners' expectations* by explaining that 'pain winddown' can take 8 weeks in chronic pain cases but that I reassess after 2 weeks. *Asking owners to focus on normal behaviours for their pet can also help their perception of whether their pet has improved.*

Oral or parenteral vitamin B has been shown to reduce inflammatory and neuropathic pain in many species and can be used as part of the multimodal analgesic approach.

My preferred non-steroidal anti-inflammatory (NSAID) is meloxicam. I have been using meloxicam at 0.6–1.5 mg/kg q12hrs for around 20 years now. Meloxicam has recently been licensed for use in guinea pigs but at a very low dose that is not sufficient in my opinion (and many other exotics vets' opinion) to achieve effective analgesia. Personally, I evaluate response to analgesia and adjust as

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Table 3.3 Guinea Pig Analgesia

Analgesic	Dose
Meloxicam	0.6–1.5 mg/kg q12hrs PO
Paracetamol (acetaminophen)	15–20 mg/kg q8–12hrs PO
Buprenorphine	0.2mg/kg q4–6hrs OTM 0.03mg/kg q 6hrs SC
Tramadol	10–30 mg/kg q6–12hrs PO
Gabapentin	10–30 mg/kg q8–12hrs PO
Maropitant	2–4 mg/kg SC
Methadone	1–2 mg/kg SC q4hrs dependant on pain scoring
Amantadine	3–5mg/kg PO q24hrs.
Amitriptyline	5mg/kg PO q24hrs
Lidocaine	2mg/kg (maximum 4mg/kg)
Bupivacaine	1mg/kg (maximum 2mg/kg)
Acupuncture	

required. Just as with more familiar species I utilise meloxicam in many types of pain. *As guinea pigs' stomachs always contain food, I am not concerned about administering with food in this species*.

Paracetamol (acetaminophen) works well if given in conjunction with meloxicam. I order mine from my usual wholesaler and use 15–20 mg/kg q8–12hrs.

In my experience tramadol works extremely well for various types of moderate pain. *In humans, around 20% of the population do not get any analgesic effects from tramadol and I am highly suspicious the same will be true in some animals due to the lack of a particular enzyme*. Liquid tramadol is often more cost effective when obtained from a compounding company rather than human liquid tramadol. However, many guinea pigs will take the contents of 50mg capsules dissolved in a known quantity of water (therefore providing you with a mg/ml solution) which the owners can make up daily for oral administration. I use 10–30 mg/kg q6–12hrs.

Gabapentin works well for neurogenic pain and is my go-to analgesic if I am suspicious of disc disease. The dose may need adjusting up if the patient has been taking the medication for a long time. *This further highlights the importance of regular pain scoring*. I start my gabapentin at 10 mg/kg q12hrs and will go up to 30 mg/kg q8hrs.

Maropitant can be useful for visceral and skeletal pain, administered by subcutaneous (SC) injection at a dose of 2–4 mg/kg. The exact mechanism is unknown and more work needs to be done on how much analgesia is provided but I have certainly found it to be useful in some cases. Also, just because guinea pigs cannot vomit does not mean they cannot feel nauseous.

Buprenorphine 0.2 mg/kg q4–6hrs can be given by the oral transmucosal (otm) route. Only the single use vials can be used in this manner as the multidose bottle contains a preservative which is bitter tasting. If used by SC injection I give 0.03 mg/kg q6hrs.

Methadone has a short half-life in guinea pigs but it is useful in cases of extreme pain. I use 1–2 mg/kg SC, this will last 4 hours and further dosing is dependent on pain levels. Opioids can cause problems with gut motility but so can pain. Pica has been observed in rodents that have been administered opioids.

Amantadine is an antiviral which works on the NMDA receptors and provides analgesia as part of a multimodal approach. I have not used it often in guinea pigs, the dose recommended is 3–5 mg/kg PO q24hrs.

Amitriptyline 5 mg/kg PO q24hrs can be used to help break the cycle of self-injurious behaviour as part of a multimodal approach (Green, 2021).

Acupuncture has shown great success in alleviating chronic pain as part of a multi-modal approach and is frequently used in my practice. It has been shown to work by inducing endogenous opiates and reportedly naloxone will reverse analgesic effects of acupuncture (Figure 3.8).

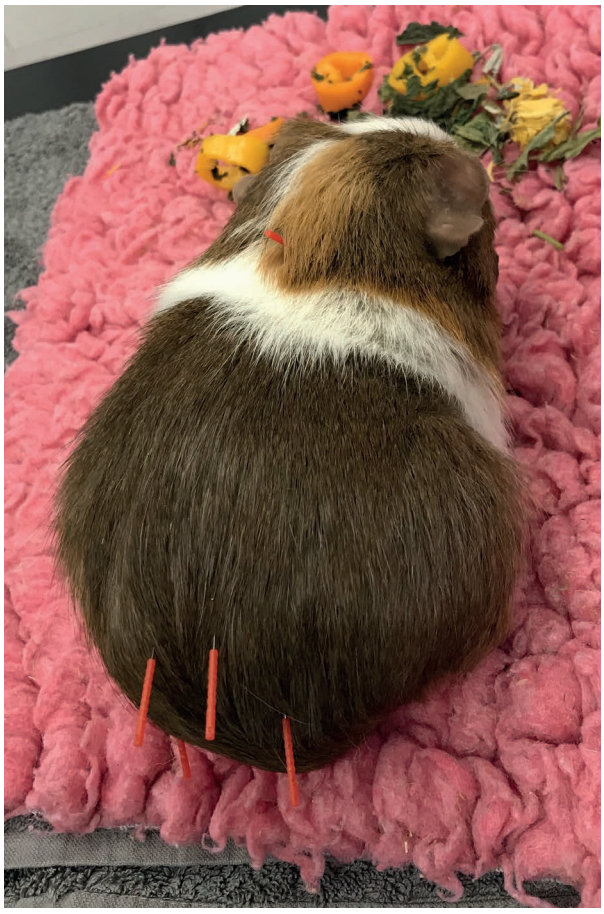


Fig. 3.8 Most guinea pigs can be given acupuncture conscious. Photo: Dr Krissy Green.

Clinical exam

I always place a towel or blanket on the table to reduce the patient's stress. Prey species have evolved to be prey, therefore have an innate requirement to escape predation at a split-second notice. This is why they find a slippery surface so frightening, if they cannot find purchase, they cannot escape predation. Using the blanket or towel reduces the chance of them injuring themselves or you as their handler.

If possible, take the top off the carrier to enable easier, less stressful removal of the guinea pig from the carrier. I will look at the guinea pig face on to check for facial symmetry.

Personally, I prefer to perform the rest of the examination from behind so that the guinea pig is facing its owner. I perform the same clinical exam, in the same order that I do for any other species (except that I perform oral examination of the molars last). See Table 3.4 for biological data.

Is there any evidence of nasal discharge? As obligate nasal breathers, guinea pigs fastidiously groom their face and nose, so a minor nasal discharge may not be evident at the nares – check the medial aspect of their front legs by running your thumbs down the inside of the front legs. If there is a nasal discharge, we will often feel crusting on the fur. I will assess the length of the nails on the front feet at the same time.

Palpate the mandible for any masses which may or may not indicate dental disease. Check for peripheral lymphadenopathy and palpate the ventral neck (guinea pigs can develop goitres).

I check for ocular symmetry from above the head, is there any evidence of exophthalmos? Are the eyelids normal? Rex coated guinea pigs are overrepresented for development of entropion (see Ophthalmology section for more detail).

Take note of any ocular discharges or crusting around the eyes (flaky conjunctivitis may be the first sign of hypovitaminosis C). Is there any inflammation? Are any cataracts present? Apply fluorescein to check for corneal ulcers.

Table 3.4 Biological data.

Parameter	Reference range
Average longevity	4–8 years
Respiratory rate	40–120 bpm
Heart rate	226–300 bpm
Body temperature	38–39.5°C (100.4–103.1°F)
Maintenance fluid therapy	100–150 ml/kg/day
Sexual maturity	4–6 weeks female, 9–10 weeks male
Gestation	59–72 days (63 average – which is the same as a bitch)

Examine the ears, is there any sign of: crusting, wax (normal wax is often the same colour as the skin in guinea pigs and can vary from light brown to grey black), inflammation/pruritus, foreign bodies (grass awns etc.), masses, pus (may require cytology as may be hard to differentiate from wax but other clinical signs will help). Remember – they are just ears!

A skeletal examination performed the same way as more familiar species and as previously discussed in the Pain section is my next step. I also check for thoracic compliance by compressing the thorax laterally mid thorax, although it *should be remembered their thorax is not as flexible as a rabbit's*. Lack of thoracic compliance indicates that there is pathology in the thorax such as: thymoma, abscessation or dilated cardiomyopathy to name a few. Sometimes this subtle finding is the only clinical sign indicating radiography.

Thoracic auscultation is performed on both sides and listening to several areas on each side. Take note of any adventitious noises but also of any areas of dullness. If you are finding it difficult to differentiate between upper respiratory sounds and lower respiratory sounds – listen to the ventral neck. We do this all the time in brachycephalic dog breeds. I do not count heart rates in guinea pigs as I find it impossible with heart rates between 226–300 beats per minute. If it is beating, I cannot count it, and the rhythm sounds normal, I move on. Heart murmur may sometimes be audible.

Check through the coat and examine the skin. We are looking for the same issues we would see in any examination of fur and skin: lumps, scabs, alopecia, dandruff, pustules, sores, wounds, and ectoparasites (the fur mite, *Chirodiscoides caviae*, is commonly seen in immunocompromised individuals and is easily missed if not actively looked for, visually appearing as dark specs on the fur frequently on the shaft of the hair approximately a third of the way down from the tip – see section on dermatology for more information).

Examine the scent gland, which is found on the caudal aspect of the rump approximately where a tail would be expected.

Both male and female guinea pigs have nipples and the mammary region should be palpated for masses. Male guinea pigs are at an increased risk of malignant mammary neoplasia (see Reproductive disorders section).

Examine the genital region. Is there any evidence of urinary scald or faecal build-up? If the patient is an entire male palpate the testis. Examine the anus, boars have an anal sac and aged boars sometimes lose the tone leading to faecal build-up (see Reproductive disorders section).

Examine the palmar and plantar aspects of the feet.

Ulcerative pododermatitis (hock sores), can quickly become life threatening as the skin is thin, the tendons and bone lay in close proximity and can become secondarily

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infected. Overgrown nails alter how they walk and can exacerbate this condition.

Gently palpate the abdomen. Fatty liver disease occurs quickly in the ill guinea pig (especially if obese), the enlarged and friable liver can rupture and cause abdominal bleeds. Take note of any areas producing repeatable flinches (pain) and any abnormal masses. If the guinea pig is female *gently feel for ovarian cysts taking care not to rupture them*. I have occasionally had cysts rupture during palpation and although I always warn the owners there could be an issue due to haemorrhage or hormonal overload if the cyst is follicular rather than cystic rete – thankfully I have never had an issue. This is likely due to the fact most of the cysts are cystic rete therefore hormonally inactive (see section on reproductive disorders).

Check for tympany or gas build-up. Are there faecal pellets palpable? It really is just a gentler version of the same examination we do in other species. I often use abdominal palpation to assess hydration status as discussed below.

I perform the conscious oral dental examination last as most guinea pigs will find this stressful. Many owners are unaware that guinea pigs have molars and that all their teeth grow continuously. I advise owners that we can miss up to 50% of dental disease cases during conscious oral exams as the guinea pig chews the scope and sticks their tongue down the end of it. That being said, due to the oral anatomy (the tongue has a soft tissue attachment to the cheek), I find oral clinical exams much easier in the guinea pig compared to the rabbit.

Performing conscious oral clinical exam

After wrapping the guinea pig in a burrito, I will ask an assistant (which may be the owner) to hold the guinea pig vertically with its back nestled against their breastbone supporting the thorax and its hindquarters. I use the medium metal otoscopic cone.

Restraint of the head is important. I use my non-dominant hand (for me that is my left hand) to restrain the head, I place the palm of my hand flat onto the skull and use my index (pointer) finger and thumb to hold onto the point of the zygomatic arch just distal to the eyes. I then pull gently caudally with my index and thumb.

> Guinea pigs always have food in their mouths, so this is completely normal. However, it does mean that the oral cavity must be swabbed out with cotton buds (Q tips) before the teeth can be visualised.

Guinea pigs have a normal occlusal plane of 30-degree oblique with the lower arcades angled towards the tongue and the upper arcades angled towards the buccal mucosa.

I place the otoscope cone into the right diastema just caudal to the upper incisors. Owing to the soft tissue attachment between the tongue and the cheek, I always aim towards the hard palate when I first enter the mouth then tilt the scope back down, this ensures I 'jump' over the attachment, otherwise it is likely all you will see is tongue. Guinea pigs also have a mandible which is wider than their maxilla meaning that their lower molars are situated more laterally than in rabbits.

Examine the soft tissues for any wounds, ulceration, or bleeding. Move around the guinea pig, without taking the scope out, and perform the rest of the clinical exam to include the both upper and lower arcades.

Maxillary molars flare towards the cheek although the spikes formed in my experience are more like shelves than the more dramatic spikes seen in rabbits. The lower arcades grow towards and over the tongue with advanced cases causing complete bridging of the tongue and a complete inability to chew or swallow effectively.

Many vets (including myself) used to focus on looking for spikes when performing this exam. Now I advise looking for asymmetry (seeing spikes is a bonus). Asymmetry is often easier to appreciate. If they have pain on one side, they will preferentially chew on the other side which leads to asymmetrical wear of the continuously growing molars. If in doubt I advise full oral exam under sedation or general anaesthesia (once stabilised if anorexic) including skull radiographs. This is discussed fully in the section on dentistry.

Anorexia and gut stasis (ileus)

Anorexia is a true emergency in guinea pigs which can lead to gut stasis, a negative energy balance and fatty liver syndrome (very prone to this especially if overweight) developing within 12–24 hours. It is important to remember that *anorexia is a symptom* not a condition in guinea pigs.

Unfortunately for us, anorexia is a nonspecific reaction to stress which can be physical (pain anywhere and illness) or psychological (separation from cage mate, change in environment). A good clinical history and thorough clinical exam will often help direct your diagnostic tests. *I assume any anorexic guinea pig is in pain somewhere*.

For example, if the guinea pig has a painful or bloated abdomen without an obvious discrete mass, I will recommend a full general health profile and a conscious dorsoventral radiograph to check for gas in the gastrointestinal tract. This is important to differentiate cases of anorexia which may require immediate surgical intervention from those that should be stabilised before any anaesthetic or surgical procedure is considered.

Gastric dilatation and volvulus (GDV) is not uncommon in guinea pigs and on a radiograph, a gas filled stomach silhouette on the right side of the abdomen should raise the index of suspicion that fast surgical intervention is required. (see section on gastrointestinal issues).

The cardiac sphincter of the stomach prevents vomiting or burping in the guinea pig. Any gastrointestinal foreign body will quickly result in obstructive stasis with

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dire consequences without prompt diagnosis and intervention. Gas and fluid will build up proximal to the blockage, resulting in gastric and/or intestinal distention and severe pain. Gastric distention will impinge on the diaphragm making respiration more difficult. The stomach will be easily palpable.

Foreign bodies are rarely seen in guinea pigs. Radiographs will show fluid and gas filled viscera. *Stress related gastric ulceration is commonly seen* and avascular necrosis of the gastric or intestinal wall can occur.

Pancreatitis

Pancreatitis is underdiagnosed in my opinion. This seems to be spontaneous in guinea pigs and although the literature reports that obesity predisposes, all of the cases I have diagnosed have been in guinea pigs of a normal body condition score.

Clinical signs are nonspecific and are often attributed to gut stasis for another reason: anorexia, tooth grinding (due to pain), abdominal pain (sometimes localised to cranial abdomen), peritonitis, fat necrosis. Biochemistry will often show an elevated amylase, frequently several times the upper reference range limit (212–424 IU/l). Unlike more familiar species, amylase in guinea pigs is highly organ specific to the pancreas.

As with any case of pancreatitis in any species, sudden death can occur. However, I have had good clinical success with hospitalisation, multimodal analgesia (meloxicam, paracetamol, maropitant and occasionally methadone), famotidine, metoclopramide, and assisted feedings (10–20 ml/kg/feed). If I have used methadone, I will usually tailor the frequency of doses to pain scoring, and many cases will only require the methadone for 24–48 h. Multimodal analgesia and famotidine should be continued for 2 weeks to ensure resolution of any secondary gastric/duodenal ulcers or peritonitis. In my experience, guinea pigs have not had repeated bouts of pancreatitis, but it would be advisable to advise it could be possible.

Stabilisation of the anorexic guinea pig

The mainstays of stabilisation are analgesia (previously discussed), correction of dehydration, assisted feeding, and prokinetics.

Dehydration

Hydration status in guinea pigs can be challenging to ascertain. Skin tent, eye position and consistency of caecal contents (normal will feel like a 'bag of runny porridge') can all be utilised but in my experience are only obvious in severely dehydrated patients, albeit the caecal contents is often the easiest to appreciate. It should be remembered that the abdominal viscera are thin walled, any anorexic guinea pig may have fatty liver syndrome and organs can be ruptured if palpation is too rough. I advise using fingertip pressure, gradually massaging the abdomen cranial to caudal with a very gradual increase in pressure as needed. *It is recommended to assume that any anorexic guinea pig is at least 5% dehydrated.*

Oral rehydration with assisted feeding should not be underestimated but given that maintenance is believed to be 100–150 ml/kg/day intravenous fluids should be used in any moderately to severely dehydrated guinea pig. *I do not agree with subcutaneous fluid administration*. It is *incredibly painful* and dehydrated patients will not absorb fluids via this route as efficiently. Guinea pigs hide pain so relying on the fact your patient 'allows' you to perform subcutaneous administration is not rationale for it not being painful. IV access is relatively easy in guinea pigs (see Venipuncture). Peritoneal injection of fluids is risky due to the risk of inadvertently puncturing the large caecum. If IV access cannot be achieved, then sedation (if not moribund), local anaesthesia and intraosseous (IO) administration of fluids should be considered as in more familiar species.

> Stress (environmental, inappropriate handling or severe pain) also causes gut stasis. Conscious guinea pigs should not be placed on their backs as this will further stress and immunocompromise an already ill patient.

IV catheters can be connected to giving sets or boluses can be administered every 30–60 minutes. If they chew through their second giving set, I will move to boluses and make a note in the patient's file for future reference that this individual should get boluses. *Hydration status should be assessed throughout the day* which enables you to alter the rate and volume of fluids therapy as required. My intravenous fluid of choice in most cases is saline.

Assisted feeding

The term assisted feeding is preferable to force feeding when discussing with owners. There are several high-quality, purpose made foods available such as: Oxbow Critical Care, Supreme Recovery or EmerAid Herbivore. All three are high in fibre to promote gut motility, are palatable (although individuals may prefer one over another), and easy to prepare. Feeding syringes with widened nozzles are available from wholesalers made by Supreme Pet Foods and Oxbow.

Some guinea pigs will require wrapping in a towel (burrito technique) for assisted feeding, with minimum recommended volumes of 10–20 ml/kg/feed three to four times daily to support gut motility. Some individuals will tolerate smaller, more frequent feeds better.

Freshly picked grass, hay and the guinea pigs' normal diet should always be readily available to encourage the guinea pig to eat on its own.

Prokinetics/gastric protectants

Drugs to help promote normal gastrointestinal motility can be administered. Metoclopramide (1 mg/kg q8–12hrs,

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increases gastric emptying and small intestinal motility) and cisapride (1 mg/kg q8–24hrs, increases gastric emptying, small intestinal motility but has no effect on large intestinal motility) are the two most commonly used drugs. Metoclopramide can reduce renal blood flow, which may exacerbate any underlying renal issues. Clinically I have never known this to cause a problem.

Cisapride is available from compounding companies such as Summit or Bova (UK) or Doctors Foster and Smith (USA). Domperidone can also be used at a dose of 0.5 mg/kg q12hrs. I will use both cisapride and metoclopramide until the guinea pig is eating and passing normal faeces.

Stress-induced ulcers seem to be common in guinea pigs and are frequently seen during post mortem examinations. It can be difficult to know if administration of NSAIDS contributed to the gastric ulcers, however it does not stop me administering NSAIDS if clinically appropriate. I will add in a gut protectant if I am concerned about the presence of ulceration.

Ranitidine (2–5 mg/kg q12hrs) was my drug of choice due to its dual effect of being a prokinetic as well as lower gastric acid production. At time of writing this drug is only available from compounding companies. Other viable options are cimetidine (5–10 mg/kg q6–12hrs) or famotidine (0.5–1 mg/kg q12–24hrs).

Sucralfate 25–50 mg/kg q8–12hrs can be used in suspected cases of gastric or duodenal ulceration. Sucralfate requires an acidic environment to work therefore should be given at least 30 minutes before ranitidine, cimetidine, or famotidine. Sucralfate can be sourced from compounding companies such as Summit or Bova (UK) or Doctors Foster and Smith (USA).

Some owners prefer grinding the tablets and mixing with water for administration. Alternatively, the pill can be administered via a pill giver or a 1 ml syringe with the nozzle and top cut off. Compounding companies make paste versions of many drugs.

Once stable (eating and passing faeces) some owners will decide against further investigation. Depending on your findings during the clinical exam this could be an acceptable approach. For example, if you found spinal or hip pain then continuing on with long term analgesia without radiographs is absolutely fine if the guinea pig is clinically stable.

However, *if dental disease is suspected then owners should be counselled this approach would be completely inappropriate*, full investigation is required for prognosis, treatment, and management decisions to be taken.

General anaesthesia

Reducing risks associated with general anaesthesia starts before admission.

Guinea pigs cannot vomit and need a near constant supply of fibre to maintain gut motility therefore they should not be starved before an anaesthetic. Owners are used to starving themselves or their dogs and cats prior to surgery, so we need to be clear when we book guinea pigs in for surgery that they should not be starved. Advise owners to bring a 'packed lunch' of favourite and familiar foods.

> Bonded cage mates should always be admitted, not only will this reduce stress during the hospitalisation period, help protect the bond between conspecifics, it also has been shown to reduce healing times and increase coping ability for aversive or painful experiences (Denommé and Mason, 2022).

Allow guinea pigs at least an hour to acclimatise to the ward after travelling to the clinic to reduce stress levels prior to induction.

With only a few exceptions (bloat, or a liver lobe torsion), ill guinea pigs should be fully stabilised (rehydrated, passing faeces and starting/trying to eat) before an anaesthetic should be administered.

Pre-anaesthetic blood tests

The need for these is debated among vets. Personally, I advise they are an option but acknowledge in the young healthy individual the vast majority will be within reference ranges. The benefit is that it will give you a personalised reference range for this individual patient and as *guinea pigs are so good at hiding disease, occasionally we get unexpected results which may alter our plans*. Blood can be taken from the cephalic vein or the lateral saphenous vein. The jugular is not commonly used due to the short neck and the relatively deep position. There is nothing wrong with aseptically dripping the blood from a needle into the collection tube. Biochemistry can be run 'in-house' using the same pre-op/comprehensive profiles used for dogs and cats (see Tables 3.1 and 3.2 for reference ranges).

Pre-emptive analgesia/prokinetics

Once admitted (and if not already on medications) administer meloxicam 0.6–1.5 mg/kg and/or another analgesic if appropriate depending on the surgical procedure, and metoclopramide 1 mg/kg. These can either be given orally or by SC injection. SC injections should be administered over the flanks to avoid the brown fat present on the dorsum which may result in erratic absorption rates.

Pre-medication

Whether I administer a pre-medication is dependent on the stress levels of the patient and the surgical procedure being performed.

It is important to consider the stress of being handled and being injected. Rodents find injections very stressful and as they have small muscle masses IM injections are very painful and may need to be given in multiple sites depending on volume. I think very carefully before I administer pre-medicants in rodents as I have to consider the type of

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Table 3.5 Pre-medicant dose.

Drug	Dose and route	Reversal
Midazolam	1–2 mg/kg IM SC	Flumazenil 0.1 mg/kg SC
Midazolam + butorphanol	1–2 mg/kg IM SC + 0.2–0.5 mg/kg SC	Flumazenil 0.1 mg/kg SC
Medetomidine + butorphanol	0.15 mg/kg IM 0.5–1 mg/kg IM	Atipamezole 0.5–1.5 mg/kg IM SC

surgery (and which analgesics will be required) and decide whether the stress and pain of the pre-med is worse than the mask induction. In my experience mask induction while wrapped in a towel burrito results in a fast, seemingly minimal stress induction, whereas injections always result in vocalisations and obvious stress. The drugs used for this are the same drugs we use in more familiar mammals. The doses can be found in Table 3.5. Buprenorphine may be given OTM as a premed.

Local anaesthesia for muscle splash blocks or infiltration

Guinea pigs do not have an increased susceptibility to the toxic side effects of local anaesthesia, *it is simply their small size that makes it much easier to accidentally overdose them.*

Combining the drugs allows for greater effect with a lower recommended dose. Lidocaine has faster onset but a shorter duration of action (60–120 minutes) whereas bupivacaine has a slower onset of action but a longer duration (180–480 minutes). Mixing 4 mg/kg of lidocaine with 2 mg/kg of bupivacaine in a syringe then diluting the mixture with saline to increase the volume of the solution and allow easier splash blocking (exactly what it sounds like). Drip the solution onto the wound in the muscles or infiltrate an area. Pulling up the required dose and pre-diluting, prevents accidental overdose.

Induction

There are several anaesthetic protocols available for use in guinea pigs. There is an argument to be made that the safest anaesthetic is the one you are used to.

My preferred inhalational gaseous anaesthetic is isoflurane. Reportedly, most rodents react more favourably to the smell of sevoflurane but in my experience many dislike the smell just as much and in the UK isoflurane is the only licensed gaseous inhalational anaesthetic for rodents.

My preferred method of induction is to burrito the guinea pig and use a face mask. Have cotton buds (Q tips) to hand to swab the mouth clear of food or saliva post-induction.

Allow four breaths of 100% oxygen (this is all that is required to fully saturate the blood with oxygen), then turn the vaporiser straight to 5% isoflurane. Initially the guinea pig may wriggle but it will quickly become induced (most are less than 40 seconds) with minimal stress. In the vast majority of cases the guinea pig will not have even passed urine or faeces (which frequently occurs due to stress and commonly passed when induction chambers are utilised).

Turn the vaporiser to 2.5% and assess anaesthetic depth (using the same reflexes we are used to in more familiar mammals, a moistened cotton bud (Q tip) can be used to check the blink reflex), adjusting the setting on the vaporiser as necessary.

The large cornea should be protected by applying an ocular lubricant.

Intubation

Intubation is challenging due to the palatal ostium but can be done visually using an otoscope or endoscope. I do not routinely intubate guinea pigs.

Maintenance

I use a T-piece circuit, with an oxygen flow of 1.5 L/min. Isoflurane 2–4% as required.

Monitoring

Monitoring the anaesthetic is the same as for more familiar species. Multiparameter monitoring is possible. MAI Animal Health make the Vetcorder Portable Monitor, which is suitable for all species, including dogs and cats, for under £1,000. It can record: an electrocardiogram (ECG), heart rate, pulse oximetry (placed on tongue or paw), and rectal temperature. If recording an ECG, rather than use Alligator clips (which can traumatise the skin), insert small gauge needles through the skin (25 g) and attach the clips to the needles.

If that is outside the practice's budget, a soft, flexible tipped rectal thermometer can be used to monitor rectal temperature. Hypothermia is a major risk factor in anaesthetic deaths. A human foetal Doppler can be purchased online (I used eBay) for around £50, ensure you request the 8 mHz probe rather than the standard 2 mHz one. An infant stethoscope can be used to monitor heart rate if the Doppler is not an option. Respiratory rates can be counted and pulse oximeters can be used from other species but they may struggle to get an accurate reading due to the high heart rate (226–300 beats per minute in guinea pigs).

Theatre

Guinea pigs have a high surface area to volume ratio which means they lose heat rapidly. The resulting hypothermia can cause fatalities. Keeping the ambient temperature in theatre high, wrapping the extremities in bubble wrap, placing a towel on the operating table, and using a plastic drape (also allows visualisation of your patient!) are all easy ways to help mitigate heat loss. Minimise the number of times the door is opened as each time it happens the ambient temperature in the room will drop. Hypothermia leads to prolonged recovery and increased risk of death.

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Positioning

Guinea pigs are diaphragmatic breathers. Their small thoracic cavity in comparison to their large abdominal cavity can make breathing more difficult due to the weight of the abdominal organs sitting against the diaphragm. Placing a folded drape or face cloth under the thorax elevates the thorax and allows the abdominal organs to move away from the diaphragm. If the guinea pig is not intubated, ensure the head and neck are straightened to allow for easier breathing.

Surgical preparation

To conserve body temperature, minimise the surgical clip. Only one agent is used for surgical scrub, either chlorhexidine or an iodine-based skin disinfectant depending on the surgeon's preference. We do not use a surgical spirit-based spray as during evaporation this causes loss of heat. *Using warm water for the surgical scrub will help reduce heat loss*.

Transparent, plastic surgical drapes are ideal as they allow visualisation of the patient while conserving body heat. The required surgical window can be cut into the drape and a surgical spray adhesive is used in lieu of towel clips.

EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) can be applied to the wound postoperatively if no other local anaesthetic infiltration has been performed in the skin. This helps with the immediate post-operative skin pain as most rodents show the most overt signs of pain 12 h post-operatively. Since I have started using this technique, I have seen a faster return to eating on recovery.

Recovery

If medetomidine has been used it can be reversed with 0.1–0.5 ml/kg atipamezole (5 mg/ml) SC, reversing the medetomidine will also reverse the analgesia provided by medetomidine but that is not reason enough for not reversing.

Maintain the guinea pig on oxygen, and if intubated, extubate when the jaw tone increases. Shivering thermogenesis is a sign of recovery from anaesthesia. Continue to monitor while in an incubator or a warm room (ideally around 26°C (78.8F°)) until up and moving then the temperature of the room can be reduced to 20°C (68°F).

Syringe feed (10–20 ml /kg Oxbow Recovery, Science Recovery or EmerAid Herbivore) once the guinea pig is fully conscious and monitor faecal output. Advise the owner to monitor food intake and faecal output. If the owner is unable to syringe feed at home, then they must bring the guinea pig back to the practice as soon as possible for admission if they will not eat at home. In the vast majority of cases with good analgesia this will not be an issue (see Table 3.3).

Dental disease

Our current understanding of dental disease is that it is a *common, multifactorial condition*. Genetics, diet, and husbandry (lack of UVB) are all implicated in the development of dental disease. Not all owners are aware that guinea pigs' teeth grow continuously throughout life.

Diet

The mainstay of the guinea pigs' diet is hay. Lowland pasture hays contain higher energy, lower fibre, and lower phytolith (therefore less abrasive) contents compared to the mountain vegetation which forms the diet guinea pigs evolved to eat. I believe this is why captive guinea pigs almost always have slightly longer molars than their wild counterparts.

Diet is believed to contribute to dental disease by inappropriate wear of the teeth, a wider vertical movement leading to abnormal pressures (pellets/nuggets), spikes form in the mouth and over time abnormal pressures result in retrograde unerupted crown growth, being too high in phosphorus, and hypovitaminosis C.

Time taken to educate owners on correct diet and husbandry at their routine check-ups can help reduce the risk of dental disease. Since I implemented this 15 years ago, I have seen a decrease in the numbers of dental disease cases I am seeing as a first opinion vet, although this decrease is also due to improved pain assessment and management. A diet sheet is given to every new guinea pig client. Male guinea pigs are overrepresented with dental disease although currently we do not know why.

Examine incisors

Careful examination of the incisors to assess the length, shape, occlusal surface, and enamel quality is important. Incisor malocclusion is not as common in the guinea pig as it is in the rabbit. Subtle signs such as diagonal wear or horizontal ribbing of the enamel should increase our index of suspicion for dental issues. Malocclusion can be obvious to us but missed by the owners.

Open communication

An honest, open, and frank conversation is needed. *Not all owners will be emotionally, financially or time invested enough to proceed with treatment*. Owners need to be prepared to expect repeated dental procedures approximately every 8 to 12 weeks, with the second dental procedure occurring in a shorter time frame while the guinea pig becomes used to chewing in a more normal pattern. In some cases, euthanasia is the best option for that individual. *Euthanasia is not a welfare concern (a difficult concept for me to understand as a young vet), however existing in chronic pain and substandard husbandry is a massive welfare issue*.

In the vast majority of cases there is no cure, management of this lifelong condition with repeated dental surgeries, long-term analgesia, and husbandry reviews will be required. *This requires dedication and financial input from the owner. Some owners may not be able to cope with this*.

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Some early cases of dental disease without root changes may be curable after the first dental procedure, with dietary corrections so that they are mainly fed on fresh growing grass, hay and dried grass and addressing any underlying medical issues (e.g. pain). This type of diet encourages prolonged chewing in a natural chewing pattern. The high silicate phytoliths in the grass promotes normal wear of the teeth.

In my experience most owners are keen to do anything that is required – apart from feed their guinea pigs hay! There is a subset of dedicated owners who are utterly convinced their pets are the exception to the rule and do not like hay or grass. Explaining that their guinea pig may just have been too painful to eat hay and offer dried grass instead (which is softer to consume but still high in silica) may help. Full dietary review and guiding owners to trusted websites like the ones discussed previously in the diet section can help.

Once the guinea pig is stable enough (passing normal faeces but not necessarily eating on its own) I advise general anaesthesia, radiographs of the skull, full exploration of the oral cavity and dental surgery. Radiographing the skull is compulsory if the owner wishes me to perform a dental. *Radiographs are required to stage the disease*.

I radiograph the first time and how frequently I repeat the radiographs depend entirely on the stage of the disease, mild 'root' changes I will repeat the radiograph in 6 months (then in a year if still requiring dental procedures but minimal 'root' changes), severe 'root' changes I recommend repeating the radiographs at 3 months then 6 months. Ongoing frequency is dependent on if there is progression of 'root' changes and clinical judgement is utilised.

Without knowing the condition, length, and shape of the maxillary and mandibular 'roots' (technically they are actually unerupted crowns) we cannot give information on prognosis or know if long term analgesia is required. *Without this information, guinea pigs with moderate to severe 'root' disease will be in chronic pain, will not chew properly and will require much more frequent dentals*. With appropriate analgesia, guinea pigs will cope better with moderate 'root' disease, although generally speaking they do not cope as well with severe root disease, even with analgesia, as rabbits do.

Dental radiographs

Once anaesthetised (see section on General anaesthesia) I will take a right lateral and a dorsoventral skull radiograph as routine. *Good positioning is required for the radiographs to be of diagnostic use*. Personally, I do not often take oblique views unless I am worried about dental abscessation and need further assessment. If I am worried about dental disease being secondary to temporomandibular joint arthritis, I will take a cranio-caudal view of the skull (Figure 3.9).

Severe root disease is easy to see but mild or subtle changes can be harder to appreciate if you do not spend all day looking at guinea pig skull radiographs. I utilise four sets of lines on the lateral view and two sets on the dorsoventral view drawn onto the radiographs to help with assessing dental disease (Boehmer & Crossley, 2009).

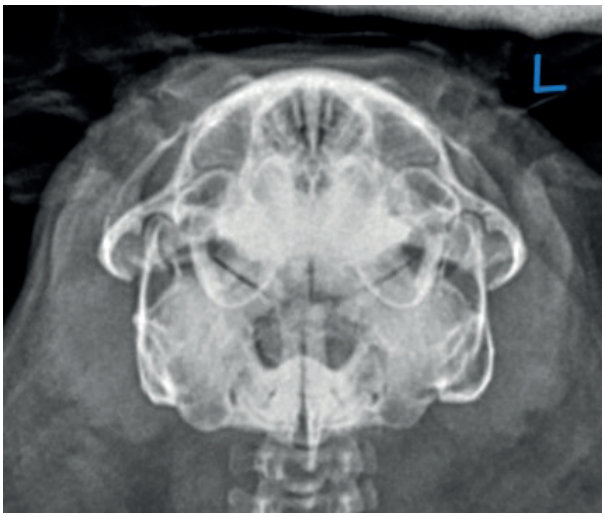


Figure 3.9 A cranio-caudal radiograph of the skull showing the temporomandibular joints and occlusal angle of the teeth.

Lateral skull radiograph

Correct positioning is vital, in a true lateral skull radiograph both mandibles are overlying each other as are the tympanic bullae. Rotation of the skull, meaning the bodies of both mandibles are visible is not a lateral view, it is an oblique and the lines cannot be drawn on the radiograph.

Two lines (drawn in white) are drawn along the palatine and mandibular bones rostral to the dental arcades. Unlike in rabbits, the guinea pig's palatal and mandibular cortices converge rostrally noticeably when the mouth is closed. The greater the degree of clinically erupted crown elongation the less obvious this becomes in guinea pigs. It is a nice visual indicator of the degree of overgrowth of clinically erupted crowns and is usually the first set of lines I will draw.

A line (in yellow) is drawn from the tip of the nasal bone to the dorsal notch of the tympanic bulla, about three-quarters of the height of the bulla. There should be *minimal to no sign of 'roots' above this line in the normal guinea pig*.

Another line (this time in red) is drawn from the point on the rostral surface of the mandibular incisor where it is crossed by a line projected cranially from the mandibular bone plate (white line), to the notch of the tympanic bulla (yellow line). This red line runs straight along the occlusal line of the mandibular cheek teeth.

Draw a line vertically (green lines) on the rostral border of the maxillary and mandibular arcade and they should be level with each other. *Mismatch of the cheek teeth arcades is a very reliable sign of significant dental disease in guinea pigs. There is often rostral displacement of the mandibular arcade in cases of dental disease*.

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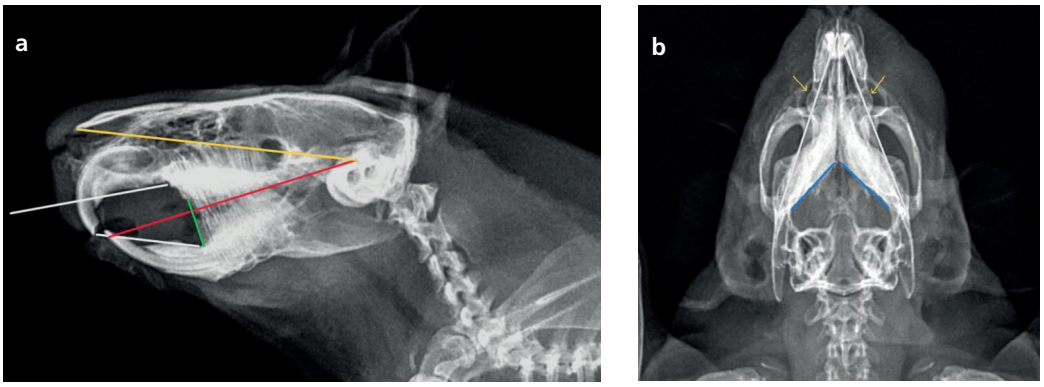
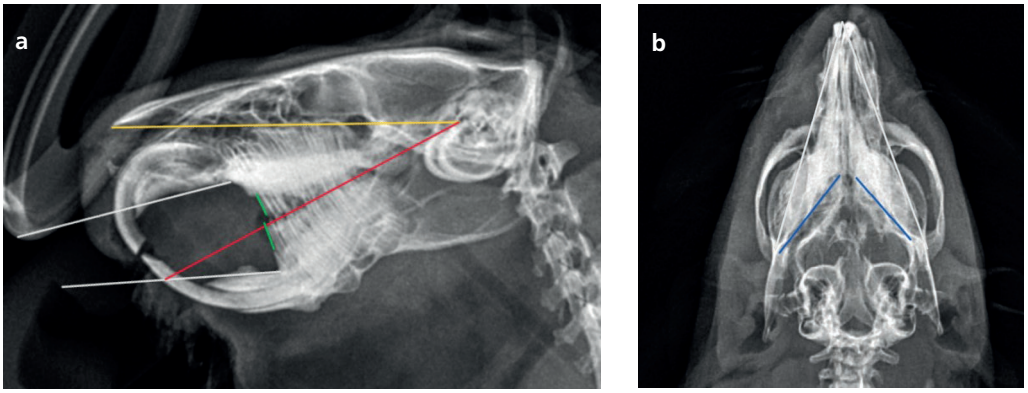


Figure 3.10
a Lateral skull radiograph of a guinea pig without dental disease with objective lines drawn.
b Dorsoventral skull radiograph of a guinea pig without dental disease with objective lines drawn.



Figures 3.11 show the same lines drawn on radiographs of guinea pigs with dental disease.
a Lateral skull radiograph of a guinea pig with dental disease with objective lines drawn. Note the rostral displacement of the mandibular arcade and the widening of the wide lines. There is mandibular overgrowth of the roots also.
b Dorsoventral skull radiograph of a guinea pig with dental disease with objective lines drawn.

Dorsoventral skull radiograph

The first line drawn (white lines), connects the mesial border of the maxillary incisor to the most caudolateral part of the ipsilateral mandible (at the level of the temporal zygomatic process). The second line is exactly the same but on the contralateral side. *No part of any tooth should be visible outside of these two lines apart from the radiolucent apical bulla of the maxillary first cheek tooth (the premolar) (yellow arrows).*

The blue lines indicate the medial cortical borders of the mandibles, there should be no penetration of this line in the normal guinea pig.

Oral examination

Once I have radiographed, I will perform a full oral examination. In the normal guinea pig:

- The dental formula is: (2x) I 1/1 C 0/0 P1/1 M3/3
- The maxillary incisors are 0.6 cm long and it is normal for the mandibular incisors to be at least twice as long, 1.2 cm). There should be a horizontal occlusal surface. Vertical ridges are normal.
- Cheek teeth: each mandibular tooth occludes with one maxillary tooth and works as a functional unit and the unerupted crown is curved in the jaw. The mandible is wider than the maxilla and at rest the cheek teeth are not in occlusion. Chewing is performed both in a lateral and rostrocaudal movements.
- All arcades should be 2–3 mm above gingival margin and occlusal plane is 30-degree oblique (the lower arcades are angled towards the tongue; the upper arcades are angled towards the cheeks) which means small changes in this plane can hamper chewing. The mouth is symmetrical (Figure 3.12).
- Guinea pigs with dental disease are likely to have asymmetrical dental arcades (Figure 3.13).

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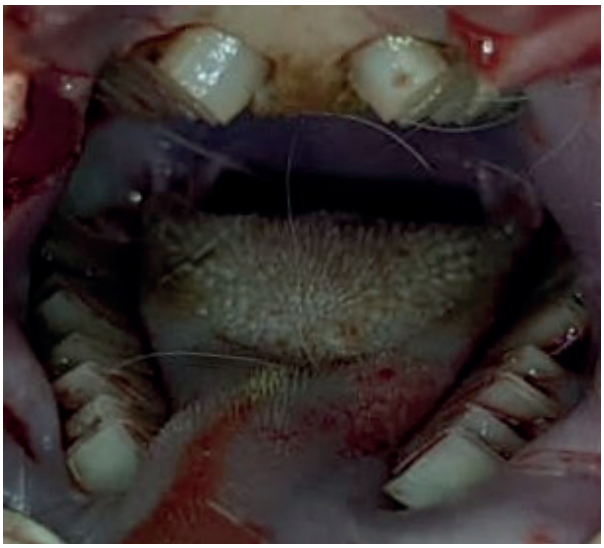


Figure 3.12 Normal dental arcades in a guinea pig. The blood is due to the fact the picture was taken during a post mortem examination.



Figure 3.14 The tongue is completely bridged. The mouth is wet because this animal can no longer effectively swallow due to tongue entrapment and guinea pigs continuously produce saliva.



Figure 3.13 Dental disease frequently results in asymmetrical dental arcades.

Generally speaking, the maxillary molars flare towards the cheek (although the spikes formed in my experience are more like shelves than the more dramatic spikes seen in rabbits) and damage the buccal mucosa (painful) while the lower arcades grow lingually across the tongue. Due to the angle of the teeth in the mandibular bone, bridging and potential entrapment of the tongue can occur. Cases as advanced as full entrapment generally carry a grave prognosis (Figure 3.14).

Guinea pigs seem to suffer from much more pain associated with retrograde root growth compared to rabbits. They are also much more likely to require several days of assisted feeding and longer courses of analgesia. Frequency between dentals could be as frequent as every eight weeks (although I usually am able to reduce the frequency to every 3 months) but owners should be warned that the first few dentals may be closer together as the guinea pigs masticatory muscles adjust to chewing normally and adjust from being stretched. If dental surgery is being required more frequently it is an indicator that our analgesia is insufficient.

The current belief is that dental disease in guinea pigs is caused by improper wear due to altered chewing and inappropriate diet, vitamin C deficiency, and temporomandibular joint disease.

> The aim of dental surgery is to restore the correct shape, length, and symmetry of all the dental arcades therefore allowing normal functioning of the teeth.

Incisor malocclusion can be managed with repeated trimming of the teeth (this can be performed conscious in many patients) using a diamond cutting disc (use of guard advised) or a dental drill bit with the water turned off, as guinea pigs become distressed by the water being sprayed into their face. The normal incisor eruption rate is approximately 2 mm a week but this can increase if they are unopposed in malocclusion. *It should be remembered that guinea pigs chew in a rostrocaudal motion as well as laterally; if the incisor malocclusion leads to the upper incisors sitting behind the lower incisors it prevents the normal rostrocaudal chewing movement.* Some guinea pigs will find frequent trimming stressful, therefore if the incisors are non-functional, or are preventing normal chewing, extraction is recommended.

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Incisors should never be clipped with molar cutters or nail cutters. This practice is outdated and barbaric. Due to the conformation of the enamel, clipping causes the enamel to shatter leading to vertical fractures, pulpitis and potentially apical abscessation. There is no excuse for clipping incisors.

Dental surgery

Extraction of the incisors

Owners worry that their pet cannot eat without their incisors. Counselling owners to consider incisors as scissors, that guinea pigs have prehensile lips and longer term cutting of grass and fresh foods into bite sized portions is all that is required. Incisors are also used during the grooming process and so owners will need to brush their guinea pig more especially during moult or in long coated breeds. *If incisor extraction is performed, all the incisors should be extracted.*

RISKS ASSOCIATED

Fracture of the incisor; if this happens continue to remove the rest of the incisors and reschedule removal of the remaining incisor once it has regrown. Post-operative infection in the socket can occur.

EQUIPMENT NEEDED

Small Crossley Incisor Luxator, size 15 scalpel blade, sterile saline flush, dilute chlorhexidine swab, curved root forceps or curved artery forceps, 4/0 Vicryl on a needle, needle holder.

I prefer to position guinea pigs in lateral recumbency for this procedure. If the coronal length of the incisors is excessively long, it is advisable to reduce the coronal length (via diamond cutting disc) prior to starting the extraction. Restrain the head in the same grip used for a conscious oral exam. Wipe the gingiva over the incisors with your chlorhexidine swab.

The Crossley Incisor Luxator has two ends: a shorter tighter curved blade and a longer gentler curved blade. These blades match the curve of the incisors: upper incisors = shorter tighter curved blade, lower incisors = longer gentler curved blade. Using the wrong blade makes the procedure much more difficult.

The 'root' of the incisor is often three times the length of the clinical crown. It is not 'pretty' surgery, there will be bleeding and occasionally a nose bleed.

I remove the upper incisors first. Using the 15 blade, incise the periodontal ligament distal to the main upper incisors.

Initially hold the luxator in a pen-like grip and using the shorter blade, place between the medial aspect of the right upper incisor and the soft tissue. With the luxator blade touching the tooth on one side, break down the periodontal ligament by pushing forwards and simultaneously using a 'see-saw' motion.

Once the luxator blade has slipped into the socket, change your grip to a standard scalpel grip. Continue pushing forwards and simultaneously using a 'see-saw' motion, and the entire luxator blade will follow the curve of the incisor and 'disappear' into the socket which is scary the first time we perform this surgery. Gently move the luxator using a laterally side to side motion to help loosen the tooth.

Remove the luxator blade and repeat the procedure on the other side of the same incisor. Use the size 15 blade under the tooth and rotate the blade to help loosen the tooth. Repeat all of the above steps until the incisor is able to be wiggled back and forth with ease by at least 1–2 cm.

Using curved forceps grasp the tooth and push it back and forth into the socket several times. This aims to destroy the apical bud the tooth grows from. Pulling along the curve (if you pull straight out the incisor is liable to fracture). Flush the socket with sterile saline to remove any bone fragments or debris. Repeat the procedure on the left upper incisor. Unless the tooth has fractured, close the gingiva over the socket (prevents food entering) using 4/0 Vicryl on a needle.

The lower incisors are removed in the same manner except the long blade of the luxator is used.

Cheek teeth

RISKS ASSOCIATED

Branches of the facial artery are located in the buccal mucosa, lateral to the last maxillary molars. Trauma to the overlying soft tissue by handheld instruments can lead to death if bleeding is not quickly stopped. Owners should be counselled that while these complications are rare, a fatal bleed can quickly occur. Collaclot is an avian collagen sponge which will quickly lead to haemostasis. It is inexpensive, has a long shelf and in these situations can be lifesaving.

EQUIPMENT NEEDED

Straight dental handpiece with rodent drill bit (with soft tissue guard), diagnostic pen torch with speculum, mouth gag, cheek pouch dilator (or Thudicum nasal speculum), moistened cotton buds.

Owing to the small amount of available space in the oral cavity, I do not intubate for dental procedures. I prefer to position guinea pigs in dorsal recumbency for this procedure with their thorax raised on a towel. Positioning like this allows me better access and visualisation of all four arcades. There are many different types of cheek pouch dilators on the market and in my experience, the springs of many of them are too strong, resulting in the

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instrument 'pinging' out of the mouth or overstretching the cheeks which will be painful. It is important when placing the oral gag over the incisors that it is opened slowly and stop opening as soon as there is any tension felt. *Over opening the mouth will cause damage to the temporomandibular joint*.

I use a diagnostic pen torch with Perspex speculum (Eickemeyer) as both my light source and my speculum/soft tissue guard. The tapered tip of the speculum provides good illumination even to the caudal teeth.

Guinea pigs, especially with clinical or subclinical hypovitaminosis C, are prone to periodontal disease. There will be pockets filled with food, hair, and debris around the teeth. It is important that all of the material is removed, I use small atraumatic forceps to grasp the hair/debris and pull it out. The pockets are then cleaned with dilute chlorhexidine.

Using the dental drill with a rodent drill bit and soft tissue guard, held in a pen grip (allows much greater control over the drill), to remove any spurs and to reduce the coronal length (if required) in all arcades. Any crowns that are very loose should be gently pulled out. Remove tooth dust with moistened cotton buds, ensure both upper arcades are symmetrical and that both lower arcades are symmetrical (Figures 3.15a & b).

Guinea pigs have a soft tissue attachment between their tongue and their cheek which can make coronal reduction of the mandibular premolars more challenging as the speculum needs to be used to push the soft tissue out of the way to allow drilling of the tooth.

If there is partial entrapment of the tongue it is difficult to use the drill. I will use molar clippers to cut one molar to allow the space for the drill, which I then use to reduce coronal length on the rest of the lower molars. The tooth is unlikely to shatter due to the folded-in nature of the enamel.

Full entrapment of the tongue, in my experience, is accompanied by severe root disease. Every case of full entrapment that I have tried to treat has been euthanised due to an inability of the guinea pig to eat normally despite multimodal analgesia. I recommend euthanasia in cases of full entrapment.

Hand rasping kits are still available. Only the smoother, diamond rasp should ever be used. Hand rasping takes much longer to perform and there is a higher risk of perforating the arteries. Caution should be exercised as any kind of rasping can potentially lead to damage of the periodontal ligament, loosen the molars, and exacerbate dental disease.

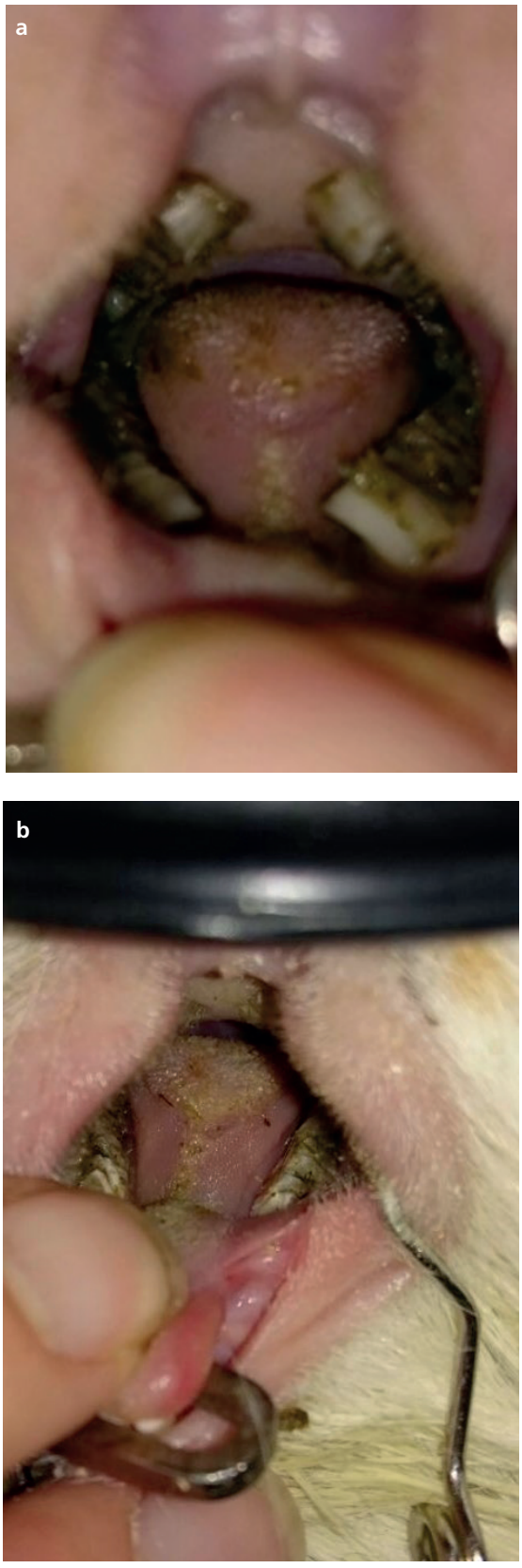


Figure 3.15
a Pre-dental.
b Post-dental.

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Post-dental care

Assist feed on recovery from anaesthesia. Assisted feeding may be required for a few days (often longer than would be needed in rabbits), allowing the jaw muscles to readapt to closing the mouth and to allow the occlusal surfaces of the cheek teeth to wear back into function. Monitor faecal output.

Continue analgesia for at least a week after the procedure although cases with 'root' disease will require long term analgesia.

Dental abscesses

Periapical infections and dental related abscesses are a complication of dental disease, which can be frustrating and difficult to treat, although in my experience, it is much less common in guinea pigs than in rabbits.

Not every facial abscess is related to dental disease, good quality radiographs of the skull and in an ideal world CT assessment would be utilised to gain the most information regarding the structures involved. Not every owner will be in the position to opt for CT scanning therefore I mainly work on radiographs.

> 'Gold standard' is a term I never use. It can cause shame in owners if they cannot afford to give their beloved pet the 'gold standard' treatment. I use the term 'textbook standard' as it removes the monetary perception. For many people their go-to emotion when they are shamed or fearful (many owners fear our negative perception) is anger. This will increase our stress levels, the owners' stress levels and ultimately everyone loses. Small changes in terminology can make a big difference to the entire outcome in many cases.

The site of the abscess will ultimately determine all the views taken but multiple views and oblique radiograph should be performed. Prognosis is variable depending on location and number of arcades involved. The guinea pig should be fully stabilised before surgery.

> To plan a surgical approach, we need to realise that there are three components to the abscess: a capsule, osteomyelitis, and the diseased tissue (dental, bony, and soft). All of this can act as a sequestrum and lead to recurrence if any is left behind.

The entire area needs to be opened up, remove all of the capsule, use rongeurs to nibble away reactive/infected bone and extract any fragments of teeth. Send some of the capsule for culture and sensitivity as the frank pus is often sterile. Any and all diseased tissue should be removed. Marsupialisation (see section on rabbit abscesses) of the area to the skin will allow continued debridement/flushing while it heals. Owners can find this distressing but advising it will increase the chances of success and reassuring them that guinea pigs heal extremely fast (sometimes too quickly) and usually the large wound will have completely healed within four weeks, will assuage many of their fears.

Medical management post-operatively consists of: multimodal analgesia, twice daily flushing of the wound with saline, using a cotton bud to remove any discharge in the wound, thick scabbing should be removed via soaking and gentle rubbing, topical agents such as: manuka honey, F10 barrier ointment, or Flamazine cream can be packed into the wound, antibiotics should be chosen on basis of culture and sensitivity (but sensible choices would include a combination of either trimethoprim sulfamethoxazole and metronidazole or enrofloxacin and metronidazole).

Scurvy (hypovitaminosis C)

Guinea pigs, capybaras, humans, most primates, and fruit bats are the only mammalian species I am aware of that cannot produce their own vitamin C therefore it is vital they are given a dietary source. *Vitamin C is a water-soluble vitamin which plays an important role in many of the body's functions such as: wound healing, healthy collagen production, neurotransmitter production and as an antioxidant.*

Healthy guinea pigs have a daily requirement of 10–30 mg/kg of vitamin C; illness, pregnancy or stress can result in almost a threefold increase in their daily requirement. Clinical signs can develop in as little as ten to fourteen days of no dietary vitamin C being provided. *Even marginal hypovitaminosis C can predispose to almost any disease state.*

Supplementation

Traditionally owners were advised to add vitamin C to drinking water, this is now discouraged. *Vitamin C is highly labile and will break down to negligible levels* when exposed to a few hours of light. Good quality guinea pig nuggets/pellets contain added vitamin C, however one-third of the vitamin C content will have broken down ninety days post-manufacture. Pet food manufacturers such as Oxbow Animal Health produce vitamin C treats for small herbivores but they are relatively expensive and anecdotally, palatability varies widely between individuals.

The best way to supplement the diet with vitamin C is via adding fresh vegetables high in vitamin C to the daily diet. Diet changes should be implemented slowly, as a sudden increase in the vegetable content of the diet may lead to digestive disturbances and subsequent diarrhoea. Suitable vegetables include broccoli, brussels sprouts, cauliflower, spinach, kale, parsley, and orange or red bell peppers.

Strawberries are also very high in vitamin C and can be offered as a treat. Citrus fruits are lower in vitamin C than strawberries but still contain a good amount. Most guinea pigs enjoy a small amount of citrus fruits, apples or tomatoes in their diet, but some guinea pigs cannot tolerate the

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acidic nature of these fruits and it will trigger painful bouts of cheilitis in these individuals.

Clinical signs

Subclinical hypovitaminosis C

Subclinical hypovitaminosis C may be accompanied by overt signs of an infectious disease such as diarrhoea, skin disease or respiratory disease (URT or bacterial pneumonia). Conjunctivitis, and/or swollen, reddened orbital margins should raise the index of suspicion for subclinical hypovitaminosis C.

Scurvy clinical signs

Primary vitamin C deficiency most obvious clinical signs are related to fragility of small blood vessels (poor-quality collagen) which rupture resulting in painful bruising of the tissues including the skin, subcutaneous and bleeding into the joints. The guinea pig will often have swollen joints (especially the stifle), lameness, demonstrate a reluctance to move (due to pain in the joints), and scream when restrained (due to painful bruises).

Haemorrhages around the temporomandibular joint (with resulting pain), and weakening of the periodontal ligament leads to secondary dental disease, reluctance to eat and drooling.

A gradual weight loss, wasting, a rough hair coat, delayed wound healing, tooth grinding (pain), paresis and skin disease may also be seen. There can be an increase in T4 levels.

Diagnosis

Diagnosis is usually based on clinical signs. Radiography may show enlargement of the long bone epiphyses and the costochondral junctions of the ribs. The respiratory tract should be examined on the radiograph for any evidence of secondary disease.

Serum levels of vitamin C can be assessed, the normal level is quoted as 114.13 umol/l (2.01 mg/dl) with clinical cases often being as low as <31.23 umol/l (0.55 mg/dl) (Shomer et al., 2015).

Treatment

Vitamin C supplementation should immediately be given. Recommended levels vary between 50–200 mg/kg in the literature and I recommend 100 mg/kg PO q24hrs. This level should continue until all the clinical signs have resolved. The diet should gradually include vegetables rich in vitamin C to prevent recurrence.

Owing to the higher level of vitamin C required I recommend using a human vitamin C tablet or powder which can be measured/cut to provide the required daily dose. Some guinea pigs will consume the dose as a treat whereas others will need the dose to be mixed with a small amount of water and immediately syringed into the mouth.

Supportive care, analgesia, and antibiotics (if concurrent bacterial infection) are just as important. If dental disease has occurred, dental surgery will be necessary once stabilised.

Prevention

Prevention is easy when providing a high-quality varied diet. *Scurvy is a captive-made condition and should not be occurring.*

Respiratory disease

Respiratory disease is an extremely common, frequently frustrating collection of conditions to treat. Ammonia build-up (poor hygiene), poor housing, poor diet (especially hypovitaminosis C) and co-housing with rabbits (*Bordetella bronchiseptica* and *Staphylococcus aureus* are both considered normal commensal bacteria in rabbits) will increase the susceptibility of overt disease. Many of the bacteria responsible are actually normal flora but act as opportunistic pathogens in the face of stress. Investigation of respiratory disease in guinea pigs is very similar to techniques used in more familiar species.

Species-specific anatomy

Guinea pigs have a small, narrow oral cavity and their soft palate extends to the base of their tongue. A small opening within the soft palate, called the palatal ostium, connects the oropharynx and the pharynx. The larynx sits dorsally within the oropharynx and is in close association with the nasopharynx. The thoracic cavity is relatively small. *All of these anatomical features predispose guinea pigs to respiratory disease and make intubation challenging* (Edis & Pellett, 2018).

It is important to advise owners that in cases of poor husbandry, failure to correct their home environment will lead to a failure of treatment. Owners should also be advised flare ups are common and, in some cases, e.g. *B. bronchiseptica* infection the guinea pig may remain a carrier for life. The normal respiratory rate for guinea pigs is 40–120 breaths per minute. The pattern of breathing should also be assessed, e.g. are they abdominal breathing?

Selected pathogens known to cause respiratory disease (not an exhaustive list).

- *Bordetella bronchiseptica*: is a significant cause of respiratory disease and subclinical infections may be encountered. Rhinitis, purulent bronchopneumonia (can involve consolidated lung lobes), fibrinosuppurative pleuritis, metritis, abortion, stillbirths, infertility, encephalitis, otitis media/internal and death may be seen. Septicaemic disease can progress rapidly resulting in death within 24–72 h.
- *Streptococcus pneumonia*: guinea pigs may be an asymptomatic carrier and it is believed that the infection can be transmitted from humans to

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guinea pigs (Percy & Barthold, 2001). Rhinitis, bronchopneumonia, fibrinopurulent pleuritis and pericarditis (leading to a pericardial effusion) are commonly seen. Nystagmus and torticollis can be seen if otitis media/interna occurs. Abortion may occur in pregnant sows.

- *Streptococcus equi subsp zooepidemicus*: is a normal component of nasal and conjunctival flora. Currently it is believed to be an opportunistic pathogen gaining entry via abrasions (caused by food or dental disease) in the oral cavity. Cervical lymphadenitis develops (see section on cervical lymphadenitis) and necrotising bronchopneumonia can occur if the infection becomes systemic. Less commonly, otitis media/interna, haematuria, mastitis, abortions, stillbirths, hepatitis, nephritis, and sudden death may be seen with septicaemic infection.
- *Chlamydophila caviae*: Conjunctivitis with or without rhinitis, is commonly seen. Less commonly seen is: bronchitis, pneumonia, and metritis.
- Adenovirus: uncommonly seen in the pet guinea pig. Causes mild respiratory disease, has an incubation period of 5–10 days and stress is a factor in the development of clinical disease.

Upper respiratory tract disease

Upper respiratory tract disease (URD) is commonly referred to as 'snuffles' in guinea pigs regardless of underlying cause. *As guinea pigs are obligate nasal breathers, URD tends to be more stressful/severe than in other species*.

The differential diagnosis list for URD is allergy, bacterial (e.g. *B. bronchiseptica*, *S. pneumoniae*, *Chlamydophila caviae*, *S. equi subsp. zooepidemicus*, adenovirus (uncommon in pet guinea pigs), *Klebsiella* spp., *Staphylococcus* spp., *Pseudomonas* spp., among others), dental disease ('root' overgrowth), foreign body, or neoplasia (uncommon).

Common clinical signs include nasal discharge (unilateral or bilateral), sneezing, conjunctivitis, reduced appetite, stary coat, and lethargy in severe cases (has to choose breathing over eating or grooming).

Less common clinical signs include pain on facial palpation, pawing at face or epistaxis, head tilt (if otitis media/interna is involved). Guinea pigs are frequently bright even with moderate discharge and URD may be much more advanced/chronic/serious than the owners appreciate due to guinea pigs frustrating tendency to hide how ill they are.

During a clinical examination it is important to check if there is any evidence of nasal discharge? As obligate nasal breathers, guinea pigs fastidiously groom their face and nose, so a minor nasal discharge may not be evident at the nares – check the medial aspect of their front legs by running your thumbs down the inside of the front legs. If there is a nasal discharge, we will often feel crusting on the fur. If there is discharge, what colour is it and is it bilateral? An air flow test can be performed using a wisp of cotton wool just as in more familiar species.

Are the incisors normal? Is there a clinical index of suspicion for dental disease?

Thoracic auscultation is performed on both sides and listening to several areas on each side. Take note of any adventitious noises but also of any areas of dullness. *If you are finding it difficult to differentiate between upper respiratory sounds and lower respiratory sounds – listen to the ventral neck*. We do this all the time in brachycephalic dog breeds. Unless there is concurrent lower respiratory disease, the thoracic auscultation is often normal. Check the thoracic compliance is normal. Lack of thoracic compliance indicates that there is pathology in the thorax such as abscessation, lymphoma, or dilated cardiomyopathy to name a few.

Bacterial URD infections can progress into rhinitis and although initially may present unilaterally will often progress onto bilateral involvement. Allergic rhinitis (shavings, hay dust, scented candles, air freshener, perfume are a few common allergens I have come across) may initially present as a clear discharge but progress onto a white pus like discharge with secondary bacterial involvement. URD may result in otitis media/interna (with or without overt clinical signs) due to migration of bacteria via the eustachian tubes, therefore it is important to assess the tympanic bullae. Secondary pneumonia can result due to bacteria translocation via the trachea.

Investigation of URD

If there is evidence of dental disease on the clinical exam, radiographs of the skull are strongly advised to assess for any retrograde 'root' growth and assess the nasal cavity. I advise for dorsoventral, craniocaudal, and lateral views to be taken. Check for symmetry, bony destruction, radiopacities (pus is thick), septal deviation – all the same changes we would look for in more familiar species. The only difference is that we also need to check for retrograde 'root' growth into the nasal cavity.

While the guinea pig is anaesthetised for the radiographs, a deep nasal swab should be taken to send for bacterial culture and sensitivity. Use a small bacterial swab and insert up the nostril (try not to touch the swab to the nares), aiming medially and slightly ventrally. Insert the swab 1–2 cm and gently rotate. If there are signs of conjunctivitis, conjunctival scrapings can be taken using a bacteriology swab, this can then be sent for bacteriology, PCR for *C. caviae* and/or a smear can be examined once stained with Giemsa may then reveal intracytoplasmic inclusions suggestive of *Chlamydophila* involvement.

Pure growths of one or two bacteria in a culture should be viewed as significant (i.e. they are contributing/causing the disease process) whereas a very mixed species culture is more suggestive of normal flora.

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Treatment and antibiotic therapy for URD

Supportive care of fluids and assisted feeding should be given if the guinea pig is anorexic.

Regular cleaning of their bedding to stop ammonia build-up is important as ammonia causes paralysis of the cilia in the respiratory tract, preventing mucociliary clearance.

If allergies are suspected then any suitable changes should be made to the home environment e.g. changing from shavings (especially if scented!) to paper-based bedding, removal of air fresheners. Trial therapy with antihistamines: chlorphenamine maleate 0.2–0.4 mg/kg q12hrs, diphenhydramine 2 mg/kg q8–12hrs, or hydroxyzine 2 mg/kg q8–12hrs, has proved beneficial in many cases.

Ideally antibiotic choice is guided by culture and sensitivity results; however, owners' finances do not always allow testing. Owing to fluoroquinolones being important antibiotics both in human and veterinary medicine, an argument can be made for starting mild cases on trimethoprim sulfamethoxazole 30 mg/kg q12hrs and using a fluoroquinolone in non-responsive cases. *Chlamydophila caviae* for example will not respond to trimethoprim.

> We can make an informed choice in severe cases to start the guinea pig on either chloramphenicol 50 mg/kg po q12 hours or enrofloxacin 10 mg/kg q12 hours while waiting on our bacterial culture sensitivity results.

Antibiotic courses can vary in length from 2 weeks for mild cases up to 8 weeks or longer for severe or chronic cases. Some owners will find that stressful but with suitable antibiotic choices the risk of dysbiosis is minimal. See Table 3.6 for commonly used antibiotics in guinea pigs.

Vitamin C supplementation should be given via an increase in fresh vegetables such as broccoli or red and orange bell peppers.

Tetracycline eye ointment can be used in cases with conjunctivitis.

Owners can nebulise their guinea pigs at home with saline which can make the guinea pig feel better by rehydrating the airways. Allowing the owner to nebulise antibiotics at home is a decision for each veterinary surgeon to make. I am uneasy about potential risks due to owners inadvertently inhaling the nebulised antibiotic. In the clinic, antibiotics can also be nebulised two to three times daily for 30 minutes (Table 3.7). See the section on LRD treatment for further information on nebulisation.

Mucosal linings of the nasal cavity may be swollen, therefore even in cases without erosion they will benefit from meloxicam twice daily.

A mucolytic such as bromhexine 0.5–1 mg/kg q12–24hrs can help reduce the viscosity of the nasal discharge enabling easier clearance by the guinea pig. *Warning the owner that initially this may cause an increase in sneezing or pawing (grooming) of the nose is advisable as owners tend to panic and think it is a sign their pet is getting worse.*

> Ensure that good husbandry is maintained as high levels of ammonia may result in recurrence as opportunistic commensal bacteria just need an immunocompromised host. Advise paper-based bedding and pre-'dunk' hay in water before feeding to reduce any dust.

Table 3.6 Commonly used antibiotics in guinea pigs.

Drug	Dose
Azithromycin	15–30 mg/kg PO q24hrs
Ciprofloxacin	10–25 mg/kg PO q12–24hrs 1 drop topical q8–12hrs
Chloramphenicol	30–50 mg/kg SC, IM, IV, PO q8–24hrs
Doxycycline	2.5–5 mg/kg PO q12hrs
Enrofloxacin	10–20mg/kg PO SC q12hrs (IM painful and may result in sterile abscess/muscle necrosis)
Marbofloxacin	5 mg/kg PO q12–24hrs
Metronidazole	15–20mg/kg PO q12–24hrs
Oxytetracycline	50 mg/kg PO q12hrs
Penicillin G Procaine form	*Do not give orally* 50,000 U/kg SC q3–5days
Silver sulfadiazine cream	Topical q12–24hrs
Trimethoprim sulfamethoxazole	20–30mg/kg PO q12hrs

Table 3.7 Nebulisation doses.

Drug	Dose (most nebulisers will hold 10 ml)
Saline	10 ml good for hydration of respiratory tract
Acetylcysteine (mucolytic)	10–20 mg per 1 ml saline (higher doses may be associated with bronchospasm)
Bromhexine (mucolytic)	0.15 mg per 1 ml saline
F10	1:250 or ready to use (already prediluted)
Aminophylline	3 mg per 1 ml saline
Amikacin	5 mg per 1 ml saline
Doxycycline	13 mg per 1 ml saline
Enrofloxacin	10 mg per 1 ml saline
Gentamicin	5 mg per 1 ml saline

Reducing stress in the home environment is advisable – check if there are enough hides, separate from predators, add in a Pet Remedy diffuser, or a nutraceutical such as Zylkene.

Lower respiratory tract disease

Guinea pigs may seem clinically normal even with advanced disease. Owners should be advised that *stress could cause a rapid decompensation and even death*. Providing oxygen prior to or during clinical exam may be required and for really stressed-out guinea pigs midazolam should be considered as this will also improve the depth of respiration.

The differential diagnosis list for lower respiratory tract disease (LRD) (which *includes diseases affecting the thoracic cavity as they may present as respiratory disease*) is pneumonia, pleuropneumonia, abscessation, pericarditis, pleural effusion, cardiomyopathy, lymphoma, pulmonary neoplasia, or pulmonary metastatic disease (uncommon). Bacterial causes for LRD are common.

Common clinical signs include coughing, increased expiratory effort, dyspnoea, anorexia, weight loss, depression, exercise intolerance (may be missed in sedentary guinea pigs, especially if their cage is too small), and fatigue.

Auscultation of the lungs should be performed as per more familiar species although using an infant diaphragm will make it much easier. The list of adventitious noises that may be heard are exactly the same as in more familiar species – it's just lungs after all. Of equal importance is lack of sound, i.e. no air movement where air movement would be expected. This would indicate areas of consolidation, e.g. abscessation, tumour or pleural effusion.

A reduction in thoracic compliance (although their thorax is not as compliant as in rabbits or rats) immediately alerts us to the presence of a space occupying lesion which could be tumour, abscess, or an enlarged heart.

Investigation of LRD

If the guinea pig is still passing faeces, then progress to sedation and radiographs. *Many guinea pigs will find it easier to breath once sedated as they are no longer stressed. Careful monitoring is required as breathing may be more difficult in certain positions. To reduce the risk of artefact from hypostatic congestion we always perform the dorsoventral thoracic radiograph first* before progressing onto right and possibly left lateral radiographs. Assess the radiographs for the exact same patterns as in more familiar species, paying particular attention for bronchopneumonia, lung lobe consolidation, pleural effusion, neoplasia, and tracheal elevation.

Neoplasia of the respiratory tract is seen and is frequently misdiagnosed as pneumonia (Greenacre, 2004). I have had the inverse experience of misdiagnosing pneumonia as pulmonary neoplasia once! Guinea pigs with pulmonary neoplasia will usually be over three years of age and bronchogenic papillary adenoma is the most frequently diagnosed. Bronchogenic papillary adenoma are slow growing, do not metastasise, and act to reduce functional lung volume by acting as a space occupying lesion (Greenacre, 2004).

Other respiratory neoplasms that have been reported in guinea pigs include bronchogenic and alveologenic adenocarcinoma, and a malignant mixed tumour of the mammary gland that had metastasised to the lungs (Greenacre, 2004). Malignant pulmonary neoplasms are uncommon. Uterine neoplasms rarely metastasise in guinea pigs.

> Lymphosarcoma and leukaemia may increase the size of the mediastinal lymph nodes causing dyspnoea, and should be included in the differential list for guinea pigs presenting with respiratory signs.

Ultrasonography can be utilised to differentiate between soft tissue opacities and fluid. *Owners can find the strangest things distressing, so it is important to tell owners that their guinea pig will be shaved for ultrasound*; many owners will not make the connection between ultrasound and the need for clipping.

Drainage of a pleural effusion will immediately provide relief while hopefully allowing a cytological diagnosis.

Bronchoalveolar lavage (BAL), can be performed, with 1–2 ml/kg of warm sterile saline being the recommended volume. Intubate with a sterile endotracheal tube (ET) which is challenging in guinea pigs but can be achieved using an otoscope, pass a suitably sized (depends on size of ET tube) dog urinary catheter that has been prefilled with sterile saline and already has the syringe attached. This prevents the saline for the BAL being lost in the dead space of the catheter. Instil the saline and then aspirate. Scant saline may be recovered but keeping the syringe attached to the catheter as it is withdrawn, allows the saline that has been aspirated into the catheter to be collected into a sterile tube for submission cytology and ideally culture.

Treatment of LRD

Ultimately the treatment required will be dictated by the actual diagnosis.

Ideally antibiotic choice is guided by culture and sensitivity results; however, owners' finances do not always allow testing. Owing to fluoroquinolones being important antibiotics both in human and veterinary medicine, an argument can be made for starting mild cases on trimethoprim sulfamethoxazole 30 mg/kg q12hrs and using a fluoroquinolone in non-responsive cases. *Chlamydophila caviae* for example will not respond to trimethoprim but may respond to enrofloxacin.

I continue antibiotics for one to two weeks after resolution of clinical signs to try and reduce the risk of relapse. Owners should be prepared that antibiotic courses can be two months long, some owners will find that stressful but with suitable antibiotic choices the risk of dysbiosis is minimal.

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We can make an informed choice in severe cases to start the guinea pig on either chloramphenicol 50 mg/kg PO q12hrs or enrofloxacin 10 mg/kg q12hrs while waiting on our bacterial culture sensitivity results.

Vitamin C supplementation should be given via an increase in fresh vegetables such as broccoli or red and orange bell peppers.

Meloxicam twice daily is recommended and nebulisation can benefit by delivering drugs locally, rehydrating the respiratory tract and may produce a 'feel good' sensation as a result.

Pulmonary oedema can be treated using furosemide 2–4 mg/kg q4–6hrs (there are liquid versions available, e.g. Frusol). The dose should be decreased to the lowest controlling dose.

Reduction of stress in the home environment will be beneficial.

Nebulisation therapy

Mesh nebulisers are much quieter, produce less vibration, and so are less stressful for the guinea pig. Thankfully they can now be bought inexpensively online. Droplet size produced should be 3 μm (Desprez *et al.*, 2019). Mucolytics and antibiotics can be delivered using nebulisation. F10 disinfectant (bactericidal, virucidal, fungicidal) can safely be nebulised using a 1:250 dilution of the concentrated product, for 15 minutes twice daily. Mild cases of respiratory disease may be successfully managed with the use of F10 alone.

Videos can be found on YouTube to direct owners on how to set up their own nebulisation cage at home.

Mucolytics can be nebulised enabling the guinea pig to clear any discharge more efficiently, which can result in an increase in sneezing or coughing in the short term. *Counselling owners that the guinea pig may develop a productive sneeze or cough when given mucolytics will manage their expectations and stress levels*.

Cervical lymphadenitis

Also known as caseous lymphadenitis, the most commonly seen presentation is an otherwise clinically healthy guinea pig with extremely large cervical lymph node/s.

Streptococcus equi subsp zooepidemicus is the main causative pathogen and is a normal component of nasal and conjunctival flora. Currently it is believed to be an opportunistic pathogen gaining entry via abrasions (caused by food or dental disease) in the oral cavity, resulting in infection and abscessation of the lymph nodes. Although transmission from guinea pigs to humans has not been reported, the zoonotic potential of this agent should be considered when working with infected guinea pigs.

Other pathogens including: *Streptococcus* spp, and *Yersinia pseudotuberculosis* (zoonotic) have also been documented to cause *caseous lymphadenitis*. *Lymphoma should be included in the differential list for enlarged lymph nodes especially in guinea pigs over three years of age*.

Diagnosis

Perform a fine needle aspirate (FNA) of the lymph node using a 21 g needle. I send the slides to an external laboratory for diagnosis.

Treatment of bacterial cervical lymphadenitis

The size of the lymph nodes can cause pain and difficulty prehending food and swallowing. Medical management alone will be unsuccessful due to poor penetration of antibiotics through the abscess capsule and the thick nature of the pus.

Surgical intervention is required. Either removal of the lymph nodes in toto or marsupialisation of the abscess (marsupialisation is described in the rabbit chapter). Anatomically the ventral neck is similar as in more familiar mammals and surgical texts such as *Small Animal Surgery* (currently the fifth edition) can be consulted for the anatomy. Surgical removal of the lymph nodes carries the risk of damage to nerves which may result in the inability of the guinea pig to swallow.

Cardiac disease

Cardiac disease is not uncommon and most likely underdiagnosed. The common clinical signs are nonspecific dyspnoea, lethargy, and anorexia, which adds to the difficulty of diagnosis. Dilated cardiomyopathy with secondary pericardial effusions is most commonly diagnosed, and a heart murmur is auscultated in the minority of cases (Curros Morenoa *et al.*, 2017).

There may be a reduction of thoracic compliance due to the cardiac enlargement and standard thoracic dorsoventral and lateral radiographs should be taken under sedation/general anaesthesia. Radiographic findings may include raised trachea, cardiomegaly, pleural effusions, and pulmonary oedema.

Assessing cardiac size radiographically

Normal cardiac size

The guinea pig has a short thorax, with the heart occupying a relatively large space within the thorax. The normal position of the heart is lying midline between the second to fifth ribs and should be 2–3.5 intercostal spaces (De Silva *et al.*, 2022).

Vertebral heart score

De Silva *et al.* (2022) established a vertebral heart score (VHS) for guinea pigs, albeit in a relatively small sample size, this can still be used as a tool to aid in the diagnosis and

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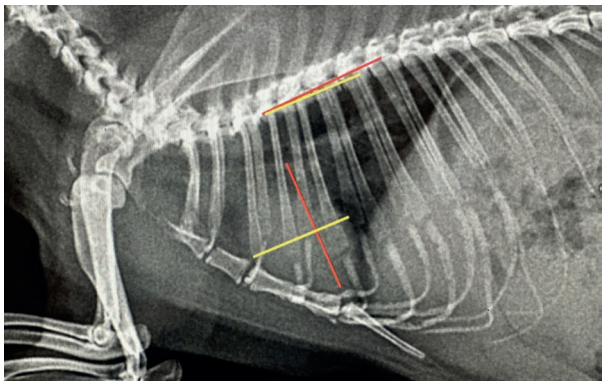


Figure 3.16 A guinea pig lateral radiograph with a normal VHS of 7.1.

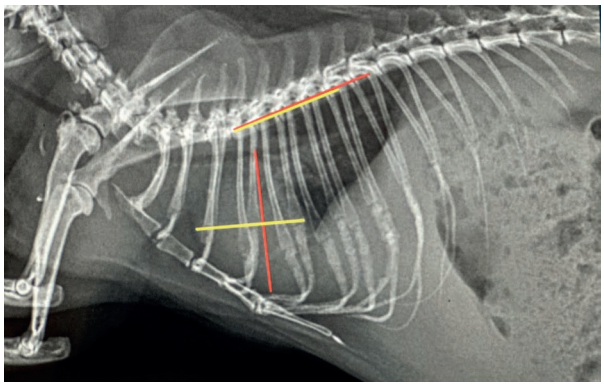


Figure 3.17 A guinea pig lateral radiograph with cardiomegaly and a VHS of 9.

monitoring of cardiomegaly. The method is very similar to what we perform in canine VHS using a right lateral radiograph. A line is drawn from the ventral border of the left mainstem bronchus to the cardiac apex to form the long axis (LA), the short axis (SA) line is drawn perpendicular to the LA in the middle third of the heart along the widest point. Both the LA and the SA are measured independently using the vertebrae, starting at the fourth thoracic vertebrae (T4), to nearest 0.1 of a vertebrae. Both measurements are added together to form the VHS. The normal VHS for guinea pigs was established to be: 7.4 (7.1–7.6) (Figure 3.16). *It should be remembered that a normal VHS does not necessarily rule out heart disease as, for example, concentric hypertrophic cardiomyopathy or specific cardiac chamber enlargement will not always result in an increased VHS*. Ultrasonography is recommended if there is a high index of suspicion of cardiac disease.

It is advisable to ultrasound the heart to check for the presence of a pericardial effusion.

Treatment of cardiac disease

If there is a pericardial effusion present, then drainage via ultrasound guided needle under a general anaesthesia is recommended.

Furosemide 2–5mg/kg PO or SC q12hrs can be given for pulmonary oedema. A human liquid preparation of furosemide called Frusol (Rosemount Pharmaceuticals Ltd) is available at variable strengths which some patients find palatable.

Pimobendan 0.6 mg/kg q8–12hrs. I dilute the contents of 5 mg capsule in a known quantity of water, e.g. 2 ml to make a 2.5 mg/ml solution. I advise we do not know how long the medication is stable for and advise owners how to make the solution and to make it fresh weekly. Enalapril can also be used 0.5 mg/kg q24hrs. My personal preference is to start on furosemide and pimobendan, then reassess in a week. Ideally taper furosemide to the lowest controlling dose and add in enalapril if needed. Prognosis is variable depending on severity of heart disease at first presentation, but I have seen guinea pigs get up to 1 year on medication.

Ophthalmology

The approach to the ocular exam is the same as with more familiar species, it is just eyes and associated structures! The notable differences in guinea pigs are more laterally situated eyes, a low blink rate, no sclera should be visible (if you can see the sclera that indicates exophthalmos), normal tears can have a slightly milk appearance (thought to be used to clean eyes and face), tear production is low normally, dacryocystitis is much less common than in the rabbit, ocular pain may be hard to appreciate (increased blink rate, epiphora, squinting are all indicators).

Fluorescein application should show at the nose within 2–5 minutes. Failure to appear or if the dye runs down the side of the face should increase suspicion of a blocked NLD.

Conjunctivitis

Conjunctivitis is seen very commonly in guinea pigs. Conjunctivitis with/without a flaky discharge can be an early sign of hypovitaminosis C (see Scurvy) and dietary assessment should be performed.

Eye ulcers

Guinea pigs will frequently present squinting in one eye or keeping it closed. Some cases may present without apparent ocular pain, but with excess lacrimation and chemosis of the conjunctiva. Corneal oedema associated with ulceration is variable and, in my experience, it can be subtle or absent in many cases. Neovascularisation is also highly variable between individuals. Fluorescein application will readily stain the ulcer (Figure 3.18a).

> Regardless of species, I advise owners that corneal ulcers do one of three things: heal within a week, take months to heal or lead to the loss of the eye. Managing the owners' expectations right at the start reduces stress for all involved.

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In my experience, *every single guinea pig I have diagnosed with a corneal ulcer has had an ocular foreign body*. Often this is a piece of hay (sometimes over 1 cm long) or a grass awn (Figure 3.18b). A careful assessment of the conjunctiva should be made and proxymetacaine can be applied to facilitate this.

If a foreign body is not identified, especially in cases of severe chemosis, I warn owners that it is highly likely that there is a foreign body within the conjunctiva and that the swelling will need to reduce before it is able to be identified. This prevents owners getting annoyed when your colleague pulls out the foreign body at the recheck 3 days later.

An uncommon cause of corneal ulceration is facial nerve paralysis secondary to otitis media. This will present with an inability or reduced ability to blink affected eye and one lip hanging lower than the other which can be subtle and easily missed if not looked for.

Corneal ulceration is an incredibly painful condition and therefore analgesia should be provided. Topical application of an ocular lubricant can help with ocular comfort. Topical antibiotic drops or gel, with a broad-spectrum drug such as chloramphenicol or chlortetracycline hydrochloride (Ophtocycline, Dechra) should be applied at least ten minutes before the lubricant drops to ensure they do not just run out of the eye.

Entropion

When entropion is present, the hairless lid margin is not visible due to the lid in-turning leading to haired skin resting against the corneal surface. Clinical signs are the same as in more familiar species: increased blink rate, squinting, and excess lacrimation. Keratitis, corneal oedema, or corneal ulceration may be present. Ocular surface pain may be sufficient on occasion to cause inappetence and lethargy.

Entropion may be due to a defect in the tarsal plate of the lid as seen in several dog breeds, it may also occur secondary to the irritation caused by conjunctivitis, keratoconjunctivitis sicca or trichiasis (where the eyelashes grow inwards towards the eye – more common in Rex or Texel breeds).

Before surgical intervention is performed it is advisable to treat any underlying conditions such as conjunctivitis first. Medical treatment first will reduce swelling and allow proper assessment of the degree of entropion.

The Hotz–Celsus surgical technique is my surgical treatment of choice for entropion regardless of species. Due to the small size of the eyelids, I use a sterile cotton bud (Q tip), soaked with sterile saline, inserted under the eyelid. A small boat or elliptical shaped segment of skin is removed with the straight edge close to the eyelid margin. The wound is then repaired using 0.7 m (6/0) Polyglactin 910 with a swaged-on needle (Vicryl, Ethicon). Analgesia should be given for a week post-operatively.

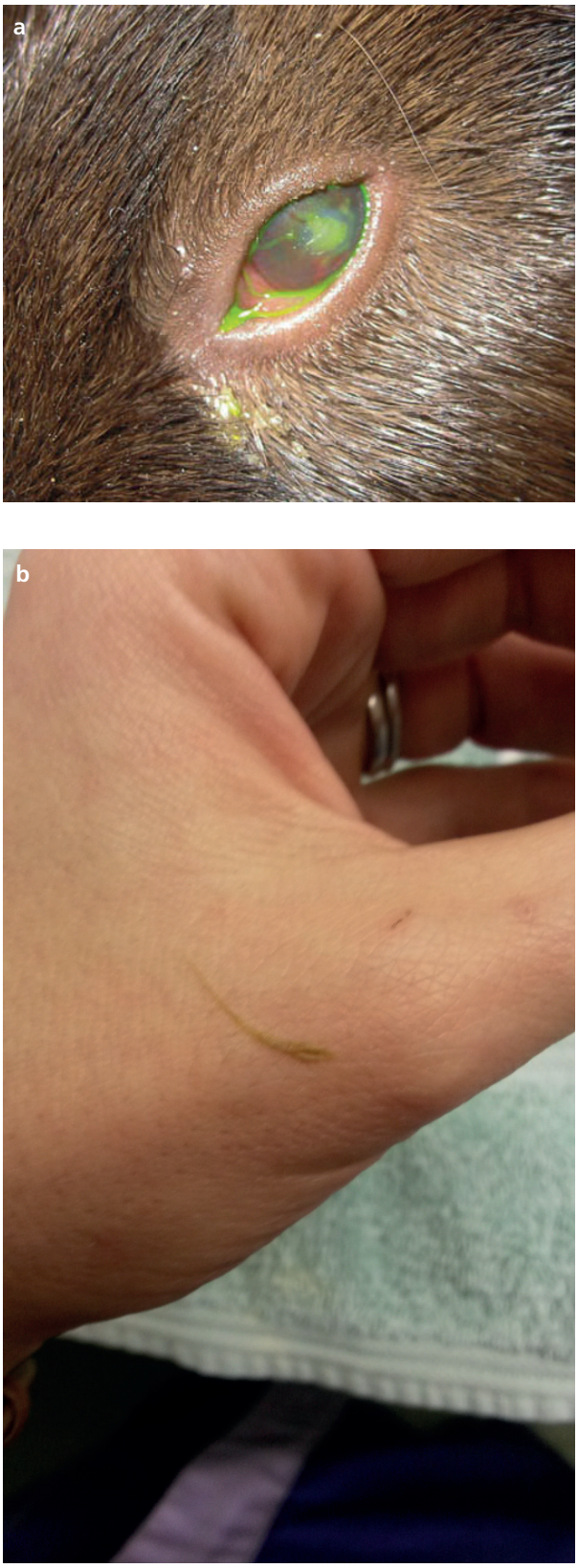


Figure 3.18
a Corneal ulcer staining fluorescein positive.
b A foreign body removed from the conjunctiva of a guinea pig with a corneal ulcer.

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The associated complications are similar to other species. If not enough skin is taken then the entropion will not be corrected and a repeat surgery will be necessary. If too much skin is taken, ectropion will result, predisposing the guinea pig to conjunctivitis.

Exophthalmos

Differentials for exophthalmos include retrobulbar abscess or tumour. Most of these will present unilaterally. As abscessation happens gradually it is common for the retropulsion attempt to be non-painful however this cannot be used to differentiate between abscess or tumour.

Owing to the presence of a space occupying lesion the globe will not easily retropulse, if at all. The presence of dental disease increases the index of suspicion for abscessation. The sclera will become visible which can be used to locate the area of the lesion (sclera is visible in same area the space occupying lesion is situated) (Figure 3.19).

A condition known as idiopathic retrobulbar adenitis is occasionally seen. It causes a non-painful, exophthalmos due to inflammation of retrobulbar glandular tissue of unknown aetiology. Ultrasonography will show multilobulated retrobulbar masses and may respond to meloxicam and broad-spectrum antibiotics for a seven-day course. If the exophthalmos results in failure to close the eyelids, there will be desiccation of the cornea and enucleation is required. The recommended technique for enucleation can be found in Chapter 1 on rabbits (see Transconjunctival enucleation), the notable difference is that *guinea pigs do not have the large retrobulbar venous plexus seen in rabbits*.

Conjunctival masses

Pea eye, fatty eye, and flesh eye are all common terms used by breeders and show owners for certain conjunctival mass appearances. Other than weight reduction in fatty eye cases, no treatment is necessary.

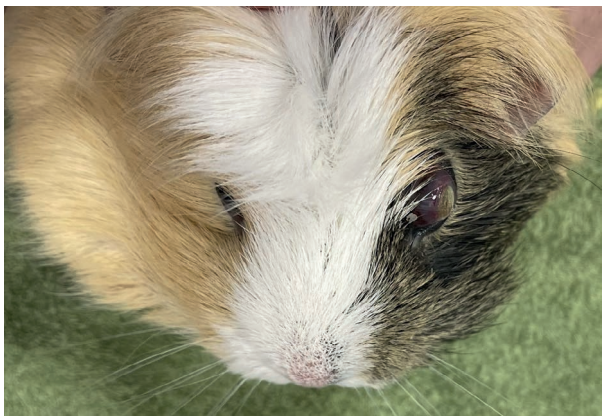


Figure 3.19 Left sided exophthalmos and exposure keratitis.

- Pea eye: has a pale or pink mass protruding from the lower conjunctival fornix. Pea eye is seen commonly and is due to protrusions of portions of the lacrimal or zygomatic glands (Figure 3.20a).
- Fatty eye: may resemble pea eye but on closer examination a layer of fat can be seen under the conjunctiva. The guinea pig is usually overweight and a reduction in calorie intake will resolve (Figure 3.20b).
- Flesh eye: pink to red protrusion of the conjunctiva near the medial canthus.

Although uncommonly seen in the eye, lymphosarcoma should be a differential for conjunctival masses in the guinea pig.

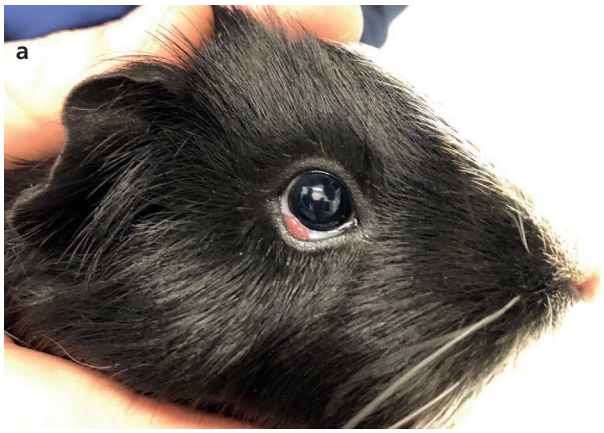




Figure 3.20
a Pea eye is seen commonly and is due to protrusions of portions of the lacrimal or zygomatic glands.
b Fatty eye in a guinea pig. The guinea pig is usually overweight and a reduction in calorie intake will resolve.

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Figure 3.21 Heterotrophic bone formation. White lesions form at the limbus and no treatment is required.

Corneal lipidosis/lipid dystrophy

As with more familiar species, lipid deposition in the eye is corneal lipidosis. A circular or oval white area in the central or paracentral cornea, and with higher magnification it can be seen that the lesion consists of a number of focal white deposits, either granular in appearance or more commonly spicular. The vision does not appear to be affected and no treatment is required.

Heterotrophic bone formation

This should not be confused with lipid deposition. White lesions form at the limbus, covering the iridocorneal angle. The lesions can be entirely circumferential or patchy. Despite concerns this may lead to glaucoma, clinically this is not found to be the case. There is no treatment required (Figure 3.21).

Dermoid

Exactly as seen in more familiar species – a congenital lesion where skin like tissue is seen in an abnormal position. Both corneal and conjunctival dermoids have been reported. Surgery is curative. Guinea pig's cornea is thinner than in dogs which may make removal of the dermoid without damaging the cornea more challenging.

Orthopaedics: fractures

Fracture repair principles used for dogs and cats are easily adapted to small mammals (it is just mammalian bone), and guinea pigs have similar skeletal anatomy that allows texts aimed at repair of selected fractures in dogs and cats to be consulted for surgical approach.

Most fractures in guinea pigs are due to trauma, either being dropped by the owner, trapping a leg in the cage or from a bite. Owing to their legs being so close to their body (which makes lameness harder to detect), their amazing ability to hide pain, and today's busy lifestyle of many owners, it is not uncommon for a considerable delay to be present between the trauma occurring and presentation. Occasionally, an old, healed fracture will be found as an incidental finding on radiographs with the owners being completely unaware of the previous trauma. The tibia is the most commonly fractured bone.

Pathological fractures can occur due to osteopenia (lack of exercise, lack of UVB, diet too high in phosphorus/too low in calcium), osteomyelitis, and neoplasia (I have seen two cases of osteosarcoma in guinea pigs, see Figure 3.22).

Various options exist for fractures in guinea pigs and many owners are keen to explore options. *Pain causes anorexia and gut stasis so prompt analgesia and supportive care is required.*

The main differences in guinea pig orthopaedics which we need to appreciate are:

- Guinea pigs rest and move with their limbs very close to their body. This makes external coaptation more challenging to effectively apply – but not impossible.
- Dogs and cats find it easy to lift a painful limb while ambulating normally on the remaining three limbs – guinea pigs find this very difficult. Guinea pigs also rely on their front legs for thorough grooming and hind legs for balance when grooming perianal region or consuming caecotrophs.

Supportive care (check for signs of shock and treat appropriately) should be given and owners should be advised that depending on the presentation, stabilisation may take several days, with the exception of spinal fractures, shock is more life threatening than a fracture is.

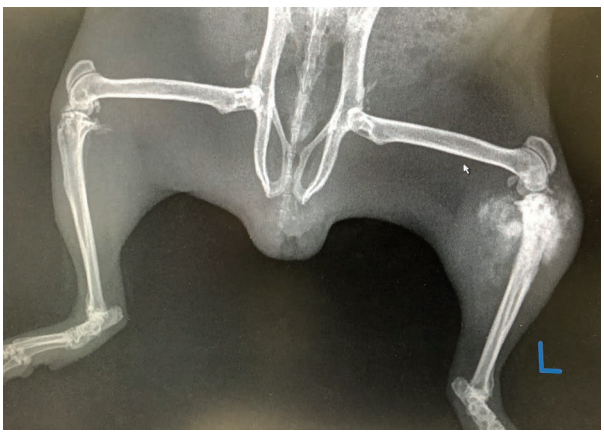


Figure 3.22 Radiograph of a confirmed osteosarcoma in the left proximal tibia.

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General anaesthesia is required to ensure diagnostic quality radiographs. Two orthogonal views should be taken, and I advise full body radiographs to assess for other skeletal injuries. Careful handling under general anaesthesia should be used if suspicious of a vertebral fracture as the epaxial muscles will not be supporting the vertebrae. If an open fracture is present, then a swab should be taken of the fracture to allow bacterial culture and sensitivity to be performed.

Regardless of fracture repair technique used, it is vitally important to adequately analgise (they will find this just as painful as you would), minimise stress, and restrict activity levels until healed.

External coaptation

The principles of external coaptation are the same regardless of species. Stabilisation of the joint above and below the injury is vitally important. Once anaesthetised, the fracture should be manually reduced while taking care not to damage the blood supply to the bone. The blood supply is necessary to allow healing of the bone. Ensure normal anatomical alignment of the joints proximal and distal to the fracture. As long as a minimum of 50% of cortical contact is achieved on both radiographic views, good healing should occur.

Standard materials used for splints and casts in dogs and cats can be applied although they will need cutting down to an appropriate size. Moldable thermoplastic cast material can be used to make gutter splints, layering of cut sections will create a robust splint and if comfortably applied most guinea pigs will not chew it. The gutter splint should be placed over a light, modified Robert Jones dressing, *but care should be taken as excessive padding allows excessive movement of the fracture.*

Padded aluminium finger splints from human suppliers can be useful too although not as robust, and they will prevent two radiographic views being taken to ensure good alignment.

Ensure the dressing is applied while the limb is in a normal, resting, anatomical position without rotation.

Owners should be advised to monitor the dressing for soiling, slippage, chewing of the split or change in their pet's demeanour/appetite. Chewing of the splint may be the first warning sign of pressure sores as if comfortable most guinea pigs will leave it alone. If the owners notice these signs they should be instructed to revisit immediately, as *bandage morbidity can be worse than the fracture in some cases*.

Schedule weekly check-ups (and changes) with splints and casts to prevent complications. Common complications (just as seen in more familiar species!) include: chewing at the splint, soiling, formation of pressure sores or local skin infections (inadequate padding), swelling (from excessive activity or from the splint being too tight), delayed union, non-union, and malunion. Joint laxity and stiffness from the coaptation may also be seen but usually resolve.

Dressing changes require deep sedation or full general anaesthesia and should be performed weekly until there is palpable stability of the fracture. Repeat radiographs are advised to ensure still have good cortical alignment.

Intramedullary pinning

Intramedullary pinning can be performed in guinea pigs. If intramedullary pins are used as a sole surgical technique, then 70% of the medullary canal should be filled. Intramedullary *pins stabilise against bending force only*. External coaptation, cerclage wire, hemi-cerclage wire, anti-rotational figure-of-eight wire, and simple external skeletal fixator (ESF) frames can be added to intramedullary pins to counteract all forces acting on the fracture, if ESF frames are to be used, ensure the intramedullary pin only fills 50% of the medullary cavity.

External skeletal fixator pinning

The basic principles of using an ESF are similar in all species. *Manual placement can result in wobble that is likely to lead to premature loosening of the pins therefore low rotational speed (150–300 rpm) insertion is advised*. That being said, in small guinea pigs, the drill may cause excessive force and a hand chuck may be preferable. A recent study of the rabbit femur indicated that the *pin diameter selected should not exceed 15% of the bone diameter* to reduce the risk of iatrogenic fractures. More work is needed to establish if the same holds true for the other bones in other small exotic mammals, but it is logical to assume it does until proven otherwise.

Positive profile threaded pins are preferred, utilising pre-drilled holes one size smaller than the pin to reduce the risk of iatrogenic fracture. If these are unavailable, smooth pins may be enough, provided they are inserted at an angle without predrilling.

Two to three pins spread evenly per segment is advised if no intramedullary pin is used, one pin per segment may be sufficient if an intramedullary tie in is used. *The intramedullary pin should be no more than 50% the diameter of the bone to allow room to place the transfixation pins*. The connecting bar should be placed as close to this skin as possible while taking into account the likely degree of post-operative swelling. A lightweight connecting bar can be fashioned from epoxy resin or epoxy putty. A postoperative radiograph to check placement is advised.

Hospitalisation for a few days postoperatively will allow daily cleaning and inspection of the pin/skin interface while allowing monitoring analgesia level for efficacy.

Skin wounds can be checked weekly then extended to every 2 weeks if stable. One study suggested the ideal time to remove ESF was 6 weeks post-operatively. Clinically it is advised to stage removal when confident the fracture is stable, has normal stiffness, but may not yet be at full strength. Staged removal is the same as for

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other species, starting with any loose pins first if only ESF. In cases where an intramedullary pin is used (and the ESF pins are stable), cut the intramedullary pin where it joins the connecting bar and tape in place to prevent migration. Remove the intramedullary pin a week later, and gradually remove the ESF over the next two weeks. If an intramedullary pin is used and the ESF pins are loose, leave the intramedullary pin and deconstruct the ESF first.

Complications are common and include fissures/fracture of bone during pin placement, infection of pin entry wounds, osteomyelitis, and non-union. A swab should be taken for culture and sensitivity from entry wounds and antibiotic selection made in conjunction with the results. Start on enrofloxacin while waiting on the results. Clean the entry wound and flush with sterile saline. In cases of osteomyelitis surgical debridement may be required due to the thick nature of the pus and any necrotic bone will act as a sequestrum.

Non-union can still result in a functional limb in guinea pigs. If ambulation is good and any pain is controlled, this may be preferable to repeated limb saving surgery or amputation.

Amputation

Guinea pigs often cope well with amputation. *Leaving a large stump behind increases the weight that the remaining leg will have to carry and has an increased risk of trauma, therefore it is not advisable to leave a long stump – it does not help with weight bearing.*

The forelimbs are of particular importance for routine grooming. If a forelimb amputation is required, I advise *the owners they may need to groom the guinea pig daily*. If a companion is present, they will often be allogrooming anyway. Guinea pigs particularly enjoy being groomed around the eyes, head, and ears as this is an evolutionary safe place to be touched – i.e. that is where companion guinea pigs groom each other. Therefore, *if the patient is a single guinea pig, the owner will need to take over that role*, washing around the guinea pig's eyes (especially the side of the amputation!).

Forelimb amputation

For forelimb amputations I favour removing the scapula in all species. This provides a fast and cosmetic repair. Owners find the skeletal appearance of the scapula (once atrophy of the remaining musculature occurs) distressing. As we are only transecting soft tissues, the pain associated with surgery should be less than bone involvement as there will be no orthopaedic pain.

Hindlimb amputation

I favour a coxofemoral joint disarticulation amputation or proximal diaphyseal amputation.

> Coxofemoral joint disarticulation amputation in entire males may increase the risk of trauma to the scrotum/testis on the affected side due to increased exposure. Consideration should be given to castration at the same time both to prevent this and reduce any mounting behaviour which will now take place on one hind leg.

Analgesia

Regardless of the technique utilised, it is important to discharge the guinea pig with multimodal analgesia for the owner to continue at home. For non-spinal fractures I advise meloxicam, paracetamol, and tramadol, I will add in gabapentin if there is a spinal fracture. *Personally, I advise continuing on with the analgesia until the leg is healed then gradually weaning off the tramadol or gabapentin before stepping down the paracetamol and meloxicam. I do not believe that the frequently quoted ten days of analgesia is sufficient as humans will take analgesia for much longer with comparable injuries – guinea pigs cannot tell us they are still sore unless it is severe.*

Dermatology

Skin disease is a common presentation due to the fact owners can easily see something is wrong. Skin disease may be a primary or secondary problem and is investigated utilising the same techniques used in more familiar species. *A skeletal exam should also be performed to ensure there is no evidence of pain reducing the ability of the guinea pig to adequately groom.*

Clinical or subclinical hypovitaminosis C should be considered, and the *very fact the guinea pig is requiring treatment should prompt us to advise an increase in vitamin C supplementation.*

Cheilitis

Inflammation and crusting on the lips are called cheilitis (Figure 3.23). It is mainly due to abrasive food stuffs or acidic foods such as tomatoes or apples. There is very much an individual susceptibility to this as in a group of guinea pigs fed the same diet there may be none or only one affected.

The crusting can cause fissures, sores and bleeding to occur. It is very painful, and anorexia may result. Gentle cleaning of the lips with warm water, application of silver sulfadiazine (Flamazine, Smith and Nephew) twice daily and analgesia, meloxicam 0.6–1.5 mg/kg q12hrs until healed, which is usually 7–10 days. Causative food items should be avoided in future.

Spurs

Keratin overgrowths are common on the lateral aspect of the feet and are commonly referred to as spurs (Figure 3.24). These should be trimmed to prevent trauma associated with catching and tearing on things. The actual growths contain no sensitive material or blood supply and can be trimmed with a sharp pair of straight nail clippers.

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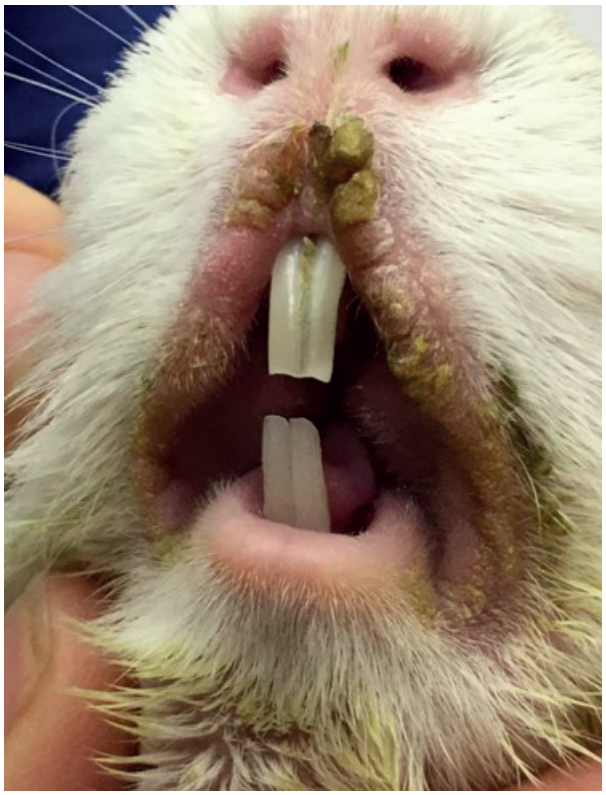


Figure 3.23 Cheilitis is very painful and may result in anorexia.



Figure 3.24 A palmar aspect photograph showing a lateral spur.

Grease/coccygeal gland

Approximately 1 cm dorsal to the anus, on the rump of the guinea pig, there is an accumulation of sebaceous glands that is more developed in male guinea pigs.

The secretion produced can cause matting of the overlying fur and give a greasy appearance to the area.



Figure 3.25 Grease gland which has become overactive leading to accumulation of sebum.

Excessive accumulation of the secretion can predispose the region to secondary bacterial infection (Figure 3.25).

Gentle trimming of the fur using scissors, cleaning of the area using a dilute solution of chlorhexidine is advised, and in cases of secondary bacterial infection: topical application of silver sulfadiazine or F10 wound ointment.

Alopecia

The pattern of alopecia and the presence or absence of pruritus may guide further investigations, which are performed using the same principles as in more familiar species. It should be noted that *symmetrical bald patches behind the ears is completely normal in guinea pigs*.

Chronic stress can result in generalised alopecia. Endocrine conditions such as Cushing's disease, alopecia of pregnancy or hormonally active cystic ovaries, all can result in symmetrical flank alopecia which may also extend ventrally (see Endocrine diseases section).

Alopecia associated with pruritus, flaking and scabbing may be associated with ectoparasites, fungal, bacterial, or nutritional disease such as scurvy. Localised, non-pruritic alopecia should prompt suspicion of fighting or barbering especially if the shafts of the hair are broken.

Ectoparasites

Mites

The most common mites seen in guinea pigs are the skin mite *Trixacarus caviae* and the fur mite *Chirodiscoides caviae*.

TRIXACARUS CAVIAE

Trixacarus caviae is a member of the *Sarcoptidae* family, this mite is arguably the most important ectoparasite of guinea pigs. This mite can be zoonotic, causing dermatitis in people, although transmission to humans is more likely to occur in immunocompromised owners.

Infestation is associated with severe pruritus, hair thinning, alopecia, erythema, hyperkeratosis, excoriation, weight loss, and secondary bacterial infections of the skin. Abortion or foetal resorption may occur in pregnant sows and the pruritus can be so intense that seizure-like episodes can occur.

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Diagnosis is achieved by clinical signs and deep skin scrapes. The mite is easily identifiable, having the typical round/oval sarcoptiform body with suckers on legs I and II.

Treatment involves killing the mites, controlling the itch, and treating any secondary infections. The environment should be thoroughly cleaned as the mite can live off the host for a long period of time.

Ivermectin (I use 1% spot on ordered from my wholesaler, which works out as one drop/500 g body weight repeated every 10 days for three treatments (200–400 ug/kg)) or selamectin 15–30 mg/kg has been shown to work against sarcoptic mites using two applications, 1 month apart.

The itch may be controlled using oral chlorpheniramine maleate 0.6 mg/kg q24hrs or a SC injection of 0.3 mg/kg dexamethasone. Topical treatment using silver sulfadiazine cream or F10 wound ointment may help secondary infections. In severe cases systemic antibiotics are warranted.

CHIRODISCOIDES CAVIAE

Chirodiscoides caviae are associated with the fur rather than the skin. The mites (visible to the naked eye) cling to the fur (often the proximal third of the shaft) and are elongated in shape with a triangular head, and *at first glance may resemble lice, however the presence of eight legs (lice only have six legs) should alert us to the fact we are dealing with a mite* (Figure 3.26).

Chirodiscoides caviae can be found anywhere on the body but seem to particularly aggregate in the axillae, groin, and the perineal region. The mite spends its entire life on the hair shaft, eggs are glued to the hairs, and all the developmental stages of the mite will be attached to the hair shaft.



Figure 3.26 Chirodiscoides caviae are elongated in shape with a triangular head, and at first glance may resemble lice, however the presence of eight legs (lice only have six legs) should alert us to the fact we are dealing with a mite.

Heavy infestations may be present without clinical signs in many cases. Other individuals will have clinical signs of varying severity, ranging from: pruritus (mild to severe), self-induced alopecia (usually on the caudal half of the body), excessive self-grooming leading to self-trauma, scaling, crusting and ulcerative dermatitis. The guinea pigs with the most severe clinical signs will usually have another underlying pathology.

Diagnosis is achieved by looking at hair plucks or tape tests under the microscope and identifying the mite. Severely affected individuals should be assessed for co-morbidities.

Treatment involves: ivermectin (I use 1% spot on ordered from my wholesaler which works out as one drop/500 g body weight repeated every 2 weeks for three treatments (200–400 ug/kg)) or selamectin 15 mg/kg has been shown to work when applied twice, 2 weeks apart.

CHEYLETIELLA PARASITOVORAX

Cheyletiella parasitovorax is uncommonly seen in guinea pigs and usually associated with guinea pigs that share an enclosure with a rabbit. Dandruff and the history of a rabbit cage mate should raise the index of suspicion and the mite is easily identified on a tape test. This mite can be zoonotic, causing dermatitis in people, although transmission to humans is more likely to occur in immunocompromised owners.

Treatment involves ivermectin (I use 1% spot on ordered from my wholesaler, which works out as one drop/500 g body weight repeated every 2 weeks for three treatments (200–400 ug/kg)) or selamectin 15 mg/kg has been shown to work against *Cheyletiella parasitovorax* using a single application.

Lice

There are two different biting lice that affect guinea pigs: *Gliricola porcelli* (the slender guinea pig louse); and *Gyropus ovalis* (the oval guinea pig louse). Both mites attach their eggs to the hairs. Heavy infestations are often a sign of immunocompromise, and other conditions should be sought rather than only treating the mites.

Clinical signs include a rough coat, pruritus, diffuse scale and alopecia, especially seen affecting around the ears and the dorsum.

Diagnosis is achieved by visualisation of the lice on the skin and examination of the lice under the microscope. *Gliricola porcelli* (the slender guinea pig louse) has six legs and a thin, elongated abdomen. *Gyropus ovalis* (the oval guinea pig louse) has six legs and an oval abdomen.

Treatment involves ivermectin (I use 1% spot on ordered from my wholesaler, which works out as one drop/500 g body weight repeated every 2 weeks for three treatments (200–400 ug/kg)) or selamectin 15 mg/kg has been shown to work using a single application.

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Barbering/ fur chewing

Barbering/fur chewing are associated with a lack of fibre in the diet, with stress, or overcrowding. Ear chewing may also be seen.

In single guinea pigs the lesions will be confined to areas the animal can reach, in other words the head and neck are spared. The affected areas have short, blunt shafts of hair due to being cut by the incisors, and the underlying skin is normal. In groups of animals the lesions may be anywhere due to other animals potentially performing the behaviour.

A full dietary and enclosure design history should be taken. Request owners take photos of the enclosure from multiple angles. Ensure access to long stem hay is provided as lack of it has been shown to induce the behaviour.

Enclosure design and placement should be assessed for number of hides, number of feeding/water stations, any bottlenecks, adequate floor space for number of individuals. Is the owner aware of any overt bullying?

Pet Remedy plug-ins can help reduce stress in the short term while changes to the husbandry are implemented. Holistic Exotic Veterinary Nutrition (HE'VN) produces a Wellbeing mix in conjunction with Hop to Forage. This herbal mix contains herbs and flowers which have been shown to decrease anxiety. It is palatable, only available to veterinary practices, and can be ordered by contacting either of these email addresses: hoptoforage@gmail.com, enquiries.hevn@gmail.com.

Pododermatitis

A common, painful, and sometimes frustrating (to treat) condition which results in chronic ulcerative granulomatous dermatitis on the plantar aspects of the feet. *It is mainly the hind feet that are affected but front feet should always be checked as they may develop as secondary lesions due to shifting of weight from hind-to fore quarters*.

Obesity, pregnancy (increases weight), poor husbandry (wet bedding or wire grid flooring causing irritation of the feet), inactivity (which may be due to pain), osteoarthritis or spondylosis (altering stance and therefore the normal weight bearing distribution), trauma, and hypovitaminosis C, are predisposing factors.

Painful guinea pigs are less active and may 'shuffle' when at rest. *This inactivity increases the amount of time the weight is borne on the area between the hock and the hind claws*, as at rest they are plantigrade, conversely digitigrade when moving. The increased pressure on the thin skin leads to ischaemia and necrosis. As the skin closely overlays tendon and bone, it is easy to understand why tendon displacement and osteomyelitis can readily become a problem.

The location of the lesions means it is easy to forget to check for foot lesions, especially in flighty or painful guinea pigs. Therefore, this condition is likely underdiagnosed in general practice. Without a doubt, *the under diagnosis of this painful condition is a welfare concern* and I have been just as guilty in the past of not checking the feet.

It is important to manage owners' expectations at the beginning. They need to understand that this will not be a 'quick fix', they will need to be willing to adjust their husbandry and their pet may be on analgesia for the rest of their lives. If the owner cannot commit to the time, financial, or emotional cost of the treatment then euthanasia should be discussed.

Sometimes owners are waiting for us to broach the topic of euthanasia, sometimes the mere fact we have broached the topic will make them understand how severe a condition is in a way no other explanation can.

Treating the lesions depends on the severity. Advising owners to provide deep bedding or providing several layers of fleece blankets/foam floor tiles under the bedding hay will help provide support to the feet.

As soon as lesions are observed, a full clinical exam with special attention paid to skeletal pain should be performed (see Pain section). Multimodal analgesia should be started, and owners should be counselled that analgesia may be required long term especially in cases of spinal pain or osteoarthritis. *Increasing vitamin C is required* also either via Oxbow Vit C treats or by increasing the amount of broccoli/peppers in the daily diet.

Mild lesions are seen as a small, circumscribed, circular area on the plantar aspect of the foot with redness/bruised appearance, and no infection or bleeding. It would be easy to miss mild lesions in my opinion. These may be resolved with weight loss (in cases requiring it), deep bedding for support and analgesia. Close monitoring is required to ensure there is no further progression.

Moderate lesions are red, extend along the foot, and can feel spongy to the touch (due to infection of the subcutaneous tissues). There may be a central superficial scab like lesion. These will be very painful (can you imagine if the soles of your feet looked like this).

Systemic antibiotics are indicated and ideally would be chosen on culture and sensitivity results. Broad-spectrum antibiotics such as trimethoprim-sulfamethoxazole (20–30 mg/kg q12hrs) or enrofloxacin (10–20 mg/kg q12hrs) are suitable choices. I apply bandaging to help pad and support the feet. Allevyn (Smith & Nephew) is my primary wound dressing of choice in cases without broken skin. It is spongy, gives support to the tissues while healing and I can easily trim to the shape of the foot and extend beyond the point of the hock. I use Micropore dressing tape to apply the dressing. I describe them as having to wear a pair of slippers until healed and advise owners it could be many weeks until the dressings are no longer required. I continue with Allevyn dressings until the redness is gone. Most guinea pigs leave the dressings completely alone in my experience. I change the dressings twice weekly although some owners are capable of doing this themselves.

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Severe lesions occur when there is full skin thickness loss. There is swelling of the foot, a thicker scab/corn, the tissue resembles a doughnut, and underlying tissue may bulge from the centre once it is cleaned. Infection is present, and the tissue may readily bleed when cleaned. The infection can extend into tendons and bone.

If concurrent bone involvement is suspected, then radiography is indicated. Osteomyelitis or tendon displacement carries a guarded to grave prognosis and we should consider whether it is in the best interests of the guinea pig to continue with treatment.

Necrotic or abscessed tissue should be surgically debrided under general anaesthesia and a swab taken from deep tissues for culture and sensitivity. Systemic antibiotics are indicated and ideally would be chosen on culture and sensitivity results. Broad-spectrum antibiotics with good tissue penetration such as trimethoprim-sulfamethoxazole (20–30 mg/kg q12hrs) or enrofloxacin are suitable choices. In cases of deep infections, antibiotics may be required for 8 weeks or more.

In open or infected wounds my primary dressing of choice is Granuflex. Granuflex is a hydrocolloid dressing and comes in a variety of thicknesses – I favour the thicker to provide more support. It absorbs exudate, supports the soft tissues, and keeps the wounds moist. It sticks to the wound (therefore should not be used in an area where stitches are present), and removing the Granuflex will help physically debride the wound. I warn owners that when the Granuflex is initially removed, the wound may look worse as it will be covered in exudate, once cleaned with sterile saline the area will look much better. My preference is to hold the Granuflex in place with Micropore dressing tape. The dressings should be replaced every two days in the first 2 to 3 weeks post-debridement, decreasing to every 5 days once the tissue looks healthier. *The granulation tissue that guinea pigs produce can bleed dramatically during cleaning which may distress both the unfamiliar vet and the owner*. I change to Allevyn dressings once the skin has healed but the area is still delicate.

End stage occurs when there is complete loss of function and euthanasia should be recommended on welfare grounds.

Dermatophytosis

Young guinea pigs will have not fully developed their immune system and have a lower concentration of fungistatic fatty acids in their sebum. Add the inevitable mixing of guinea pigs from different sources which may occur in pet shops and the resulting stress that occurs it is unsurprising that fungal skin infections are most commonly seen in young guinea pigs.

Trichophyton mentagrophytes or *Microsporum canis* are the most common isolates and it should be remembered that guinea pigs (like other species) can be asymptomatic carriers of this potentially zoonotic disease.

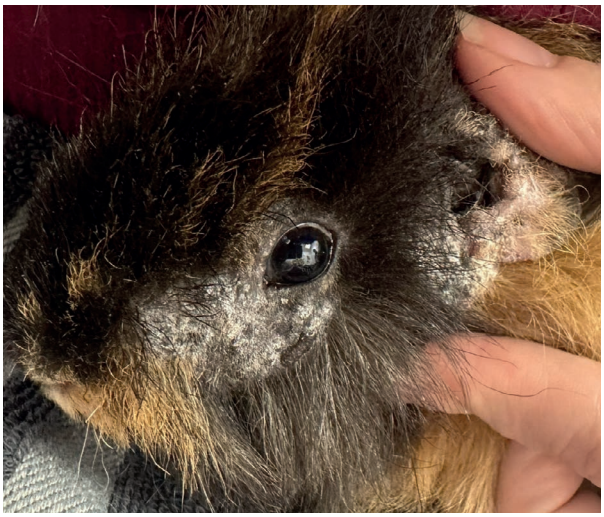


Figure 3.27 Fungal skin disease typically presents as hair loss, white scaling, and crusting lesions around the eyes, nose, and extremities. Gloves are advisable when handling affected animals.

Fungal skin disease typically presents as hair loss and crusting lesions around the eyes, nose, and extremities (although can present anywhere) (Figure 3.27). Lesions are often circular, have white scaling and are associated with more scabbing than in other species. Pruritus is seen occasionally and secondary bacterial infection is seen.

Diagnosis is via microscopic examination of the hair shaft and fungal culture. *Trichophyton mentagrophytes* does not fluoresce under a Wood's Lamp.

Treatment is always recommended due to the zoonotic risk for the owners. Topical treatment is not recommended due to guinea pigs finding dips and baths extremely stressful – this can lead to immunosuppression. Focal topical treatment is not recommended in isolation as dermatophytes will be present in areas not clinically affected.

Systemic therapy with either itraconazole 5–10 mg/kg q24hrs or terbinafine 20 mg/kg q24hrs should be started and continued until two negative fungal cultures are obtained 1 month apart. Not all owners will be able or willing to pay for repeated fungal cultures. Treatment should be for a minimum of three weeks and if owners cannot or will not repeat the cultures this should be recorded in the notes and owners should be made aware that without the tests we cannot guarantee full resolution just because the obvious clinical signs have resolved. Some sources report terbinafine is more effective. All in-contact guinea pigs should be treated whether they have clinical signs or not.

Disinfection of the environment with a suitable agent such as sodium hypochlorite or F10 is an important part of management. Wooden enclosures may be difficult to eradicate the dermatophytes from.

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Epitheliotropic T-cell lymphoma

Epitheliotropic T-cell lymphoma has been documented in several guinea pigs presenting as pruritic alopecia and scaling. Although uncommon, it should remain on the differential list in guinea pigs displaying these clinical signs, especially in those failing to respond to treatment. Diagnosis is achieved through biopsy and histopathology. Prognosis is grave, palliative care may be attempted with prednisolone 1–2 mg/kg.

Lumps and bumps

Investigation of masses is performed using the same tools already utilised in more familiar species.

Abscesses

Abscesses are common, present as subcutaneous firm, cool, and often non-painful masses. They are often missed in the early stages due to the initial small size, location, and the fur. Some abscesses can become very large, with resulting pressure necrosis of the overlying skin. Rupture of the abscess may occur, especially if there is necrosis of the skin.

> Guinea pigs lack the lysosomal enzyme required to digest dead cells which results in their pus being very thick. The abscess itself will often have a dense fibrous capsule with poor blood supply, meaning antibiotics struggle to penetrate the capsule. This is why medical management alone is usually unsuccessful.

Diagnosis can be achieved by aspirating the mass, although a large bore needle may be required due to the nature of the pus. Abscesses of the ventral neck are frequently not associated with dental disease and may be due to a 'hay poke' injury (Figures 3.28a–c).

Surgical intervention is recommended in virtually all cases. The exact approach will vary depending on location and underlying cause of the abscess formation. Ideally part of the capsule wall should be submitted for bacterial culture and sensitivity as the pus itself (often sterile) is unlikely to yield a result.

Broadly speaking, abscesses should be treated like tumours and if possible, the entire abscess should be removed in toto. This is often achievable in many areas. Any facial or mandibular abscesses should have radiography performed to determine if they are dental in origin. Dental associated abscesses are more challenging and are covered in the dental surgery section.

Marsupialisation surgery

In areas where full resection is not achievable, marsupialisation of the abscess achieves good results. Owners should be counselled that initially the cosmetic appearance of an open wound may be unpleasant, some owners may find this quite traumatising. There will be frequent check-ups

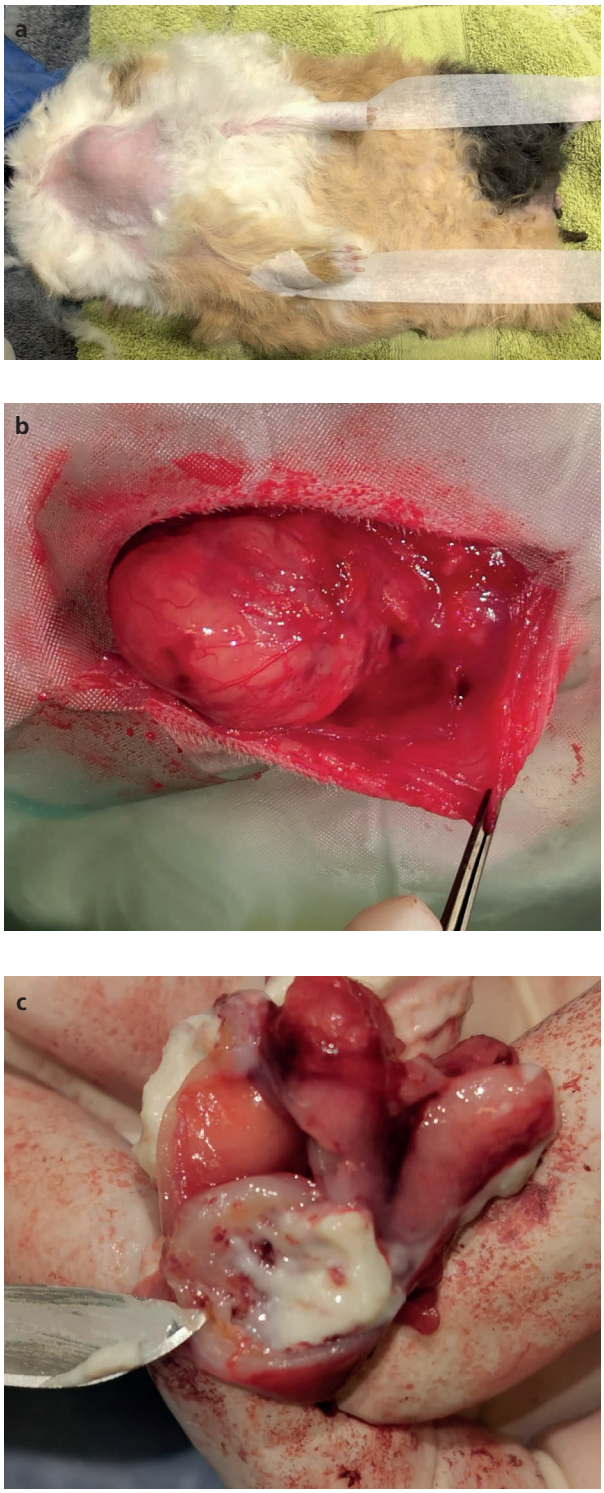


Figure 3.28
a A guinea pig with a ventral neck abscess being positioned for surgical removal.
b Surgical removal of the abscess pictured in 3.28a.
c The abscess was removed en bloc. The cut surface demonstrates how thick the capsule was which is why medical management usually fails.

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and it will take in the region of four weeks to fully heal. My preference is to hospitalise the guinea pig overnight to allow me to perform the first postoperative cleaning of the wound. Always warn owners there is a risk of recurrence. Pre-emptive analgesia and antibiotics should be given. A good first line choice while waiting for culture results would be a combination such as: trimethoprim sulfamethoxazole orally and oral metronidazole. The combination covers for aerobes and anaerobes.

Incise the skin over the abscess, and blunt dissect the tissue taking care not to penetrate the capsule of the abscess. Continue to use blunt dissection to separate as much of the abscess capsule from the healthy tissue as possible. If the abscess bursts, or you have reached the limit of what is resectable, it is important to remove all of the capsule of the abscess as *leaving any of the capsule behind will increase the risk of recurrence*. If necessary, the capsule can be stripped off underlying tissue. Debride any devitalised or necrotic tissue. Flush the area with warm saline. The skin is sutured to the open edges of the soft tissue using a fine monofilament, leaving a large deficit to heal by secondary intention. This allows daily flushing, debridement, and any topical therapeutics.

I advise owners to use saltwater (1 teaspoon full of salt to 500 ml cooled, boiled water) to flush the wound daily. Using a cotton bud (Q-Tip) to remove any discharge in the wound. Topical agents such as: manuka honey, F10 barrier ointment, or Flamazine cream can then be applied. Guinea pigs often heal too fast and the wound will contract dramatically in the first 7 days. Thick scabbing should be removed via soaking and gentle rubbing. The sutures can be removed at this stage.

Any granulation tissue present will bleed during cleaning which owners find frightening if not pre-warned – remember they do not know granulation tissue is wanted and a good thing. Managing owner's expectations by explaining what is likely to happen will help alleviate their (and your) stress. In most cases, by 4 weeks postoperatively the entire deficit will have closed.

Neoplasia

Trichofolliculomas

Trichofolliculomas are the most common skin neoplasia reported in guinea pigs (Figure 3.29). They occur most commonly in the dorsum as solitary masses with a central pore that discharges keratinous/sebaceous material. Surgery is indicated if size becomes an issue or the mass becomes traumatised. In many cases the mass does not cause an issue. Surgery is curative.

Lipoma

Lipomas are frequently seen and although they can occur anywhere, the inguinal region appears to be overrepresented. Diagnosis is by FNA and surgery is curative. Personally, I do not operate on them unless they are causing an issue. Differentials include lymphoma and mammary neoplasia.



Figure 3.29 Trichofolliculomas are the most common skin neoplasia reported in guinea pigs.

Mammary mass

Mammary neoplasia is common in male and female guinea pigs. Male guinea pigs are overrepresented for development of mammary neoplasia. Up to 75% of the masses are malignant in nature. Radiography of the chest is recommended to check for metastasis. Surgical removal with 1 cm margins and histopathology is recommended (Figures 3.30a & b).

Lymphoma

Lymphoma is common in guinea pigs and typically high-grade malignancy with a poor prognosis. Clinical signs include anorexia, lethargy, unkempt coat, peripheral lymphadenopathy, hepatomegaly, splenomegaly, and mediastinal masses (reduced thoracic compliance and potentially altered breathing).

Diagnosis is via biopsy or FNA and full biochemistry and haematology should be performed (see Venipuncture section) before treatment is attempted. Leukaemic animals may have a white blood cell count of 25,000–500,000 cells/ml.

Treatment is palliative, with prednisolone 1–2 mg/kg. Chemotherapy may provide longer survival times of 2–3 months using a lomustine prednisolone combination. Chemotherapy in this species is a decision for each individual veterinary surgeon, personally I have yet to administer chemotherapy other than single agent prednisolone in this species.

Thyroid mass

Hyperplasia, adenoma, and carcinoma of the thyroid have all been documented and it is usually seen in guinea pigs

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Figure 3.30
a The mammary mass is being held to highlight the extent.
b The mammary mass pictured in 3.30a was surgically removed with a margin.

over three years of age. There may be a palpable mass in the ventral neck. *Clinical signs are consistent with what we would expect in cats*: tachycardia, heart murmur may be present, weight loss, normal or increased appetite, polyuria/polydipsia, hyperactivity, nervousness, and soft faeces or diarrhoea (see section on endocrine disease).

Chronic cystitis

Female guinea pigs are overrepresented for developing chronic cystitis with acute flare ups. Wood (1981) suggested that female sex hormones may have a role in the development of cystitis. Azevedo *et al*. (2022) found that there was a much greater recurrence rate in cases of cystitis compared to urolithiasis.

Many guinea pigs with cystitis signs will have a full, painful bladder, and may spontaneously urinate on the consulting table during bladder palpation. Collection of the urine will allow a dipstick examination (to check for blood, glucose, protein, and pH just as with more familiar species), and potentially a microscopic examination of the sediment; normal guinea pig urine will contain calcium carbonate, calcium oxalate, and/or struvite crystals. Guinea pigs can also have porphyrin in their urine which may visually mimic blood. See Table 3.8 for normal urinalysis results.

Full clinical examination to check for skeletal pain and other comorbidities. Untreated pain or illness is a stressor in any species.

Treatment

The vast majority of guinea pigs have massive adrenal glands which may be due to chronic stress. Anecdotally, guinea pigs kept in herds, outdoors, with plenty of hides and thick layers of hay as bedding have smaller adrenal glands. As there may be a stress component to cystitis (think cats), thought should be given to using a product such as Feliway Cystease (N-acetyl glucosamine, hyaluronic acid, and L-tryptophan) or zylkene. Assessment of husbandry is recommended, both where in the house the enclosure is situated, and depth of bedding. Guinea pigs do not burrow but they do like to create hides/tunnels in deep hay which will reduce stress levels. Products such as Pet Remedy plug-ins are also beneficial at reducing stress.

Analgesia such as meloxicam should be given both as cystitis is painful, and to help control the inflammatory component. Increasing water consumption is advisable to help flush the urinary tract and may help reduce the risk of urolithiasis. Increasing the fresh vegetable component of the diet and feeding vegetables and leafy greens

Table 3.8 Urinalysis.

Urinalysis normal	
Colour	Highly variable, diet dependant Cloudy appearance common
Protein	Trace to +
pH	8–9
Specific gravity	1.030 (average) Range: 1.005–1.050
Sediment examination	calcium carbonate, struvite crystals, calcium oxalate

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wet automatically increases water consumption. Providing extra sources of water both by bowl and bottle with or without the addition of fruit juice may encourage further water drinking. Owners should be reminded that addition of fruit juice to the drinking water will encourage bacterial growth therefore not only does the water need changing daily, but the bottles and bowls require daily cleaning.

Urinary bacterial culture is advisable. Sediment examination may show bacteria and white blood cells. See Sterile collection of urine section. Trimethoprim sulfamethoxazole is a sensible first line choice of antibiotic while waiting on culture results. Chronic cases of cystitis are likely to require a long course of antibiotics and NSAID.

Potassium citrate 20 mg/kg q12hrs has been found to be beneficial. Potassium citrate binds calcium which may reduce calcium levels in the urine available for crystal or stone formation.

Any artificial supplementation of vitamin C should stop as vitamin C converts to oxalate which may result in calcium oxalate crystal formation. Guinea pigs require vitamin C as they cannot make their own but supplying it by feeding extra vegetables high in vitamin C levels (e.g. broccoli) increases water consumption and reduces the risk of excessive levels of vitamin C.

Urolithiasis

Guinea pigs absorb all calcium from ingested food and excess levels are excreted via the kidneys in the urine. Wild guinea pigs frequently ingest grass and plants that are soaked in dew or rain, thereby consuming more water.

In captivity the main source of food is hay which is a great source of fibre and nutrition but is extremely low in water content. Guinea pigs which eat a lot of fresh grass or greens will drink less than those who do not. However, neither group is likely to consume as much water as the wild guinea pigs. Maintenance water requirement is 100–200 ml/kg/day.

Inadequate water consumption can lead to calcium sand precipitating in the bladder leading to 'sludgy' urine, cystic calculi, renal calculi, and potentially life-threatening renal disease. *I believe that 'sludgy' urine etc. is not due to dietary intake of calcium but rather it is down to the fact that wild guinea pigs have a much higher water intake.*

Encouraging natural diuresis by providing cooled dandelion or nettle teas as drinking water will promote further fluid intake. Bottles of plain water should also be provided until owners are sure that their guinea pigs are readily consuming the flavoured water. Fresh dandelions can also be given daily to promote natural diuresis. H'EVN produces a urolithiasis herbal mix which can be added.

Uroliths are more common in guinea pigs over the age of 2 years, and many domestic guinea pigs have skeletal pain due to osteoarthritis, or hypovitaminosis C therefore are *reluctant to posture to pass urine as frequently* and may not be as mobile, therefore there is an increased risk of urine becoming concentrated and supersaturated, which will lead to further precipitation of sand and urolith formation.

Studies are conflicting as to whether there is a sex predilection. Male guinea pigs have a higher incidence of bladder uroliths compared to females who have a higher incidence of urethral uroliths (female guinea pigs have a wider urethra).

Clinical signs

Owners will often report vocalisation during toileting, and will frequently present their pet with the worry that there is a problem in the bladder. The pain caused by the posture required to urinate and defecate can result in vocalisation during these behaviours and so we need to also check for joint/skeletal pain during the clinical exam.

Less specific signs include hunched posture, tooth grinding, reduced appetite/anorexia, weight loss, lethargy, gut stasis, and continuous straining in cases of blocked urethra.

Blood in the urine and painful bladder palpation is highly suspicious of bladder calculi, but guinea pig uroliths (there are frequently more than one) can be present anywhere along the urinary tract including: kidneys, ureters, bladder, seminal vesicles (males), or the urethra. Female guinea pigs have a wide urethra and many stones may be passed naturally, but due to the narrow urethral papillae, females will sometimes struggle to pass the stone through the papillae and present with a blockage at this site.

Diagnosis

As uroliths are radiodense, radiography is diagnostic and allows identification of stones anywhere in the urinary tract (or seminal vesicles in males). Ultrasonography can also diagnose stones, but I find it more useful to check for secondary issues such as hydronephrosis or hydroureter.

Placing the guinea pig under an upside down plastic tub will allow a conscious radiograph to be taken (see Figures 3.7 and 3.31) which may be diagnostic but some stones may be obscured by positioning or skeleton.

Ideally the guinea pig should be sedated or anaesthetised to allow good positioning for radiography and urinary catheterisation (Figure 3.32a & b) Careful examination of radiographs for stones in the urethra, especially at the level of the os penis in males, is important to prevent missing uroliths. Skeletal examination for osteoarthritis or spinal disease should also be performed when looking at the radiographs.

Treatment

Exact treatment will depend on presentation. For example, anorexia will require a different level of intervention to haematuria alone. Some guinea pigs will require significant levels of stabilisation, IV fluids, and assisted feeding prior to surgery. Guinea pigs presenting with complete urinary obstruction will need swift emergency intervention and may have secondary renal failure (just as in more familiar species). Analgesia is important as any source of pain can

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Figure 3.31 A conscious DV radiograph showing two uroliths.

result in GI motility issues, negative energy balance, and hepatic lipidosis.

Dietary dissolution of uroliths is not possible in guinea pigs and surgical intervention is required if the stone is not small enough to pass naturally. Diazepam or midazolam can be given twice daily for three to five days to relieve urethrospasm which may aid natural passage of uroliths.

Potassium citrate 20 mg/kg q12hrs has been found to be beneficial. Potassium citrate binds calcium which may reduce calcium levels in the urine available for crystal or stone formation.

Any artificial supplementation of vitamin C should stop as vitamin C converts to oxalate which may result in calcium oxalate crystal formation. Guinea pigs require vitamin C as they cannot make their own but supplying it by feeding extra vegetables high in vitamin C levels (e.g. broccoli) increases water consumption and reduces the risk of excessive levels of vitamin C.

Recurrence after surgery is common but steps can be taken to minimise the risk (see prevention). Edell *et al*. (2022) performed a retrospective study of 158 cases and found 13.9% had recurrence of the uroliths on average 284 days later. Owners should be counselled that even with all the precautions we cannot guarantee recurrence will not occur.

Sterile collection of urine

The rough texture of the uroliths will cause trauma to the mucosa of the bladder (or other structures) and secondary infection is common.

Ideally perform a culture and sensitivity prior to antibiosis, and this can be achieved by passing a sterile urinary catheter while under sedation. For most guinea pigs this

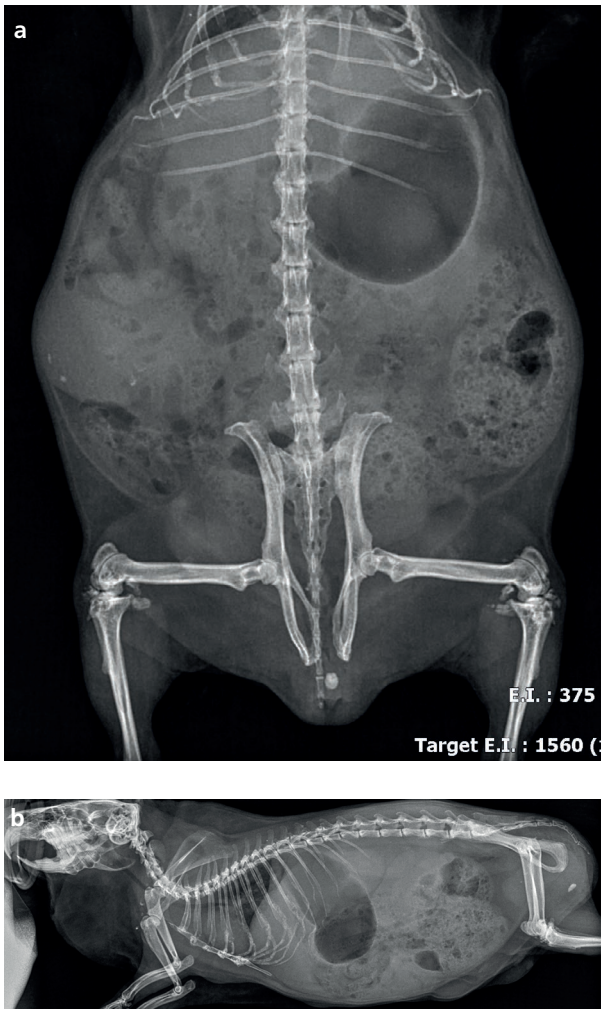


Figure 3.32
a VD radiograph showing a urethral urolith, severe bilateral stifle osteoarthritis, and narrowed disc spaces. b A lateral radiograph showing a tiny bladder stone, urethral urolith, severe bilateral stifle osteoarthritis, and narrowed disc spaces.

can be achieved using a 3–8 Fr urinary catheter, and fingertip pressure should be used to pass the catheter to avoid iatrogenic damage to the urethra. While sedated, a lateral radiograph can also be obtained to fully evaluate for stones as uroliths in guinea pigs are radio dense.

Trimethoprim sulfamethoxazole is a sensible first line choice of antibiotic while waiting on urinary culture results along with analgesia. I recommend starting medications for at least five days prior to surgery to minimise the secondary infection.

Urethral stones

Urethral catheterisation as above and retropulsion of the stone into the bladder can be attempted. The *stone may be embedded* into the urethral mucosa, which may prevent movement. Diluted lidocaine may be instilled via the catheter to alleviate urethro-spasm, which may facilitate

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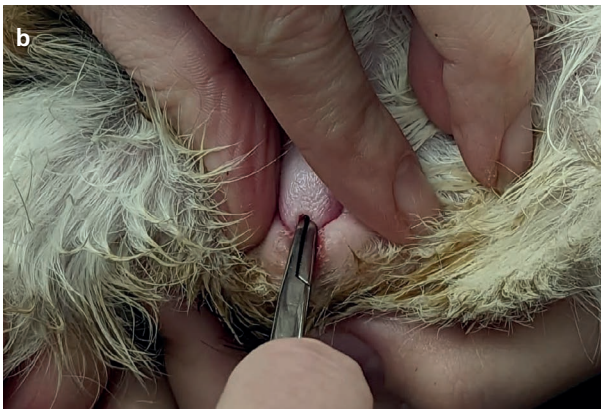


Figure 3.33
a Diluted lidocaine may be instilled via the catheter to alleviate urethro-spasm, which may facilitate retropulsion.
b After diluted lidocaine was instilled the stone was removed by gently widening the urethral papillae opening.

retropulsion (Figure 3.33a). Stones that are stuck at the urethral papillae in female guinea pigs may be able to be removed by gentle stretching of the urethra after relieving any urethro-spasm (Figure 3.33b) or may require an incision (similar to an episiotomy) to allow passage of the stone.

Cystotomy

Cystotomy in a guinea pig is very similar to in more familiar species, just on a much smaller scale. A urinary catheter can be placed as above (Figure 3.34).

My preference is to incise the skin directly over the bladder and extend the wound as needed.

Once the abdomen is open, taking care to fully tent the muscles and prevent iatrogenic damage to the caecum, I will exteriorise the bladder and pack around the bladder with moistened sterile swabs.

My preference is to ventro flex bladder and then stay sutures can be placed as per more familiar species.



Figure 3.34 A male guinea pig being catheterised.

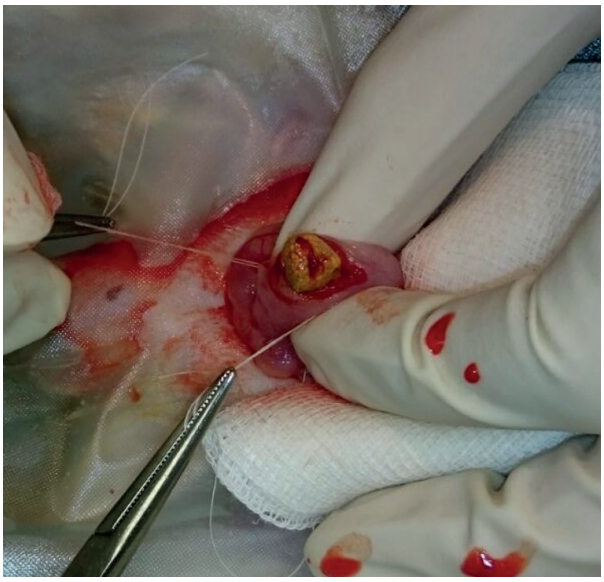


Figure 3.35 Removal of a bladder stone. Note the rough texture of the stone which can traumatise the bladder lining.

The bladder can then be incised near the cranial pole, in an avascular area. *Ensuring the bladder incision is nearer to the cranial pole of the bladder should prevent iatrogenic damage to the ureters (ureters enter the bladder in the trigone area of the neck, on the dorsal aspect)*. My rationale behind the dorsal incision is the hope that gravity will help healing, as the urine will initially make more contact with the ventral aspect of the bladder.

The texture of the stones can be extremely rough, this can cause the stone to adhere tightly to the mucosa of the bladder (Figure 3.35). Use sterile forceps to remove the stones, flushing the urethra and bladder neck with sterile saline will reduce the risk of leaving calculi behind. I also advise owners that I have experienced cases where calcified plaques have been present in the bladder mucosa rather than only discrete stones, therefore if you have flushed the

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bladder and yet cannot find all the calculi seen on the radiograph, examine the mucosa. If required, a small sliver of bladder mucosa can be removed for culture.

The bladder may be closed in one or two layers depending on the wall thickness. Most cases in my experience require two layers (avoiding penetrating the mucosa): 1.5 metric (4/0) Poliglecaprone 25 (Monocryl, Ethicon) with a swaged-on needle can be used to perform either two continuous inverting suture layers, or one inverting followed by a simple continuous, depending on individual surgeons' choice and experience. Remove the swabs and stay sutures, flush the abdomen outwith warmed saline and repair the muscle layer with 1.5 metric (4/0) Vicryl with a swaged-on needle use a cruciate pattern, repair the subcutaneous layer using a simple continuous and the skin layer may be repaired either with intradermal sutures or surgical staples. The stone can be sent for analysis. Analgesia should be continued for at least ten days, or if osteoarthritis is present, then the owner should be counselled the pet may be on lifelong analgesia.

Prevention

Increasing the water content of the diet by dipping hay in water, increasing the fresh grass component of the diet and feeding cucumber daily will help flush the urinary tract. Using daily dandelion or nettle tea will also help increase water intake and provide a natural diuresis.

Early diagnosis of osteoarthritis, prompt analgesia, and prevention of obesity will enable proper posturing during urination and allow fuller emptying of the bladder.

Endocrine disorders

Technically speaking follicular cysts may be considered an endocrine disorder as they are hormonally active, however they are covered in detail in the reproductive medicine section. Follicular cysts are the second most commonly diagnosed endocrine disorder in guinea pigs.

Hyperthyroidism

Hyperthyroidism is the most common endocrine disorder seen in guinea pigs and is likely underdiagnosed. Hyperplasia, adenoma, and carcinoma of the thyroid have all been documented and it is usually seen in guinea pigs over three years of age. One review found that approximately 55% of cases were due to an adenocarcinoma and there is a slight over representation of female guinea pigs, but more work needs to be done.

There may be a palpable mass in the ventral neck. *Clinical signs are consistent with what we would expect in cats*: tachycardia, heart murmur may be present, weight loss, normal or increased appetite, polyuria/polydipsia, hyperactivity, nervousness, and soft faeces or diarrhoea.

I had not heard of hyperthyroidism occurring in guinea pigs before I diagnosed my first case. The guinea pig was ravenously hungry, no sign of dental disease, was losing weight, and was hyperactive. I remember telling the owner that if her pet was a cat, I would have suggested hyperthyroidism and requested taking blood to check the thyroid, which confirmed the diagnosis and once on medication (I extrapolated from the available cat medications at the time) the guinea pig stabilised and went on to live a good life on medication. I used first principles, went back to basics and thought in body systems.

Diagnosis involves taking blood which is so much easier to do than most vets believe! (see Venipuncture section). A full biochemistry should also be run. The normal T4 range is: 29.6–45 nmol/l (2.3–3.5 μg/dl) (Castro *et al*., 1986). As with more familiar species, definitive diagnosis should not be based on a single blood test. A FNA can be taken from the mass to check for malignancy, the mass may be vascular as in more familiar species, so owners should be warned that there may be bleeding.

Stress can significantly decrease thyroid hormones so we should remember that hyperthyroidism may be masked by an unrelated illness inducing a euthyroid sick syndrome. Scurvy can result in an increase of circulating thyroid hormone, if scurvy or subclinical hypovitaminosis C is suspected then correct the diet and retest a month later.

Treatment

Treatment options are as variable as they are for cats.

Thyroidectomy is possible with all the same risks associated with the procedure in cats, but with a higher risk of destroying the parathyroid gland if a bilateral procedure is used. *Guinea pigs have islets of parathyroid throughout their thyroid as opposed to discrete separate glands*.

Radioactive iodine can be used but may be cost prohibitive in many cases.

Medication can be given to control the condition, which is my treatment of choice. Thiamazole (Thyronorm 5mg/ml, Norbrook) 1–5 mg/kg is my preferred treatment as it is a liquid and therefore can be more accurately dosed. In most cases medication is required once daily.

Methimazole can be used, 0.5 mg/kg q12–24hrs increasing to 2 mg/kg q12–24hrs as required. Carbimazole can also be used 1–2 mg/kg q24hrs.

Regular monitoring of T4 and renal parameters is recommended.

Hyperadrenocorticism (Cushings)

Hyperadrenocorticism is uncommon but is still the third most commonly diagnosed endocrine disorder in guinea pigs. Adrenal dependant and pituitary dependant are recognised, and can both be present in an individual guinea pig.

Clinical signs are the same as we would expect in more familiar species: bilateral, symmetrical, non-pruritic alopecia, polyuria/polydipsia, weight loss, thinning of the skin, and muscle weakness. Muscle weakness can cause multiple

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signs including apparent enlargement of the abdomen and exophthalmos (weakness of orbital muscle and abnormal periocular fat deposition).

Diagnosis is via a saliva swab for cortisol measurement and a modified ACTH stimulation test.

Saliva cortisol levels highly correlate with plasma cortisol levels, but with two important benefits. *Saliva is easy to obtain, and saliva levels of cortisol do not increase due to stress of handling*.

A saliva swab is taken for pre-ACTH levels of cortisol, 20 IU of ACTH is injected IM, then a second saliva swab is taken four hours later. The normal pre-ACTH saliva cortisol level is quoted as 5.5 nmol/l (2 ng/ml), with the expected 4 h post-ACTH saliva cortisol level being 468.9 nmol/l (170 ng/ml).

Treatment

Trilostane has been used titrating from 2–4 mg/kg q12hrs. *The drug will require compounding to allow treatment*. Regular ultrasound examination of the adrenal glands is recommended as in the reported case the guinea pig who had responded well to medical management relapsed due to an adrenal mass.

Insulinoma

Insulinoma is mainly seen in aged guinea pigs of 5 years or older. Clinical signs are the same as we would expect in more familiar species, secondary to the hypoglycaemia: acute onset neurological signs (head tilt, ataxia, twitching, seizures), weakness, weight loss, and lethargy.

A tentative diagnosis can be made using *Whipple's triad: symptoms of hypoglycaemia, documented hypoglycaemia, and response to treatment with glucose*. The normal blood glucose level of a guinea pig is: 3.3–6.9 mmol/l (60–125 mg/dl). *Repeated hypoglycaemia in the face of high insulin levels is highly suggestive of the diagnosis*, definitive diagnosis relying on a pancreatic biopsy which is not frequently performed.

Differentials of hypoglycaemia are sepsis, hepatopathy, and prolonged anorexia.

Treatment

Prognosis in guinea pigs is guarded to poor, treatment may be attempted using prednisolone 0.5–2 mg/kg or diazoxide 5–25 mg/kg q 12 hrs (or a combination of both). *The aim is not to restore euglycemia but to increase the blood glucose enough to prevent clinical signs*.

Reproductive medicine

Females

Ovarian cysts

Although five types of cysts are recognised in the literature (mainly laboratory guinea pigs), only three are commonly discussed as occurring in pet domestic guinea pigs: cystic rete ovarii, follicular cysts and paraovarian cysts. Histopathology is required to differentiate between the cysts. Ovarian cysts have been seen in guinea pigs as young as 10 weeks of age (cysts were only a few millimetres) but are most commonly diagnosed between 2 and 4 years of age. *There is an extremely high incidence of 100% in some studies but commonly quoted as 50–80% in others, leading some authors to query whether cystic rete is actually a normal process in guinea pigs*. Ovaries without cysts should measure 3–6 mm in length and 2–3 mm in diameter.

Cystic rete ovarii (sometimes referred to as serous cysts) are the most commonly seen form of ovarian cyst in the guinea pig. These cysts do not produce hormones and can reach large sizes. Follicular cysts are hormonally active and account for approximately 22% of the cysts diagnosed and also had cystic rete ovarii in the same ovary. Paraovarian cysts were actually rarely seen in pet guinea pigs. *Ovarian neoplasia is occasionally seen which may also produce hormones which could be confused as follicular cysts*.

The classical clinical signs of cystic ovaries are distended abdomen, symmetrical bilateral flank/abdomen alopecia, and crusting around the nipples. *These clinical signs are hormonal in nature therefore will more than likely be associated with follicular cysts*. However, we need to remember that these guinea pigs will have concurrent cystic rete ovarii (Figures 3.36a–c).

As prey species they have mastered hiding disease so in many cases clinical signs will not be pathognomonic and often very subtle. Nonspecific clinical signs include distended abdomen, reduced appetite (which may be missed by owners if several guinea pigs), weight loss, lethargy, vaginal bleeding, abdominal pain, and vocalisation when handled.

On a clinical exam, careful abdominal palpation will reveal large ovaries which may (or may not) be painful when palpated (abdominal tensing or vocalisation when palpated). They are situated where you would expect ovaries to be – caudal to the kidneys. *Continue the rest of your clinical exam to ensure that is the reason the guinea pig has a painful abdomen* – I have seen several cases where the reason the guinea pig was painful was not due to the large cystic ovaries but rather the large bladder calculi and post-cystotomy returned completely back to normal despite still having the ovaries in situ.

DIAGNOSIS

Ultrasonography of ovaries will confirm cysts as large fluid filled structures with thin walls. Neoplastic ovaries will show as solid masses of varying echogenicity.

TREATMENT

Only follicular cysts respond to medical management and even then, the response is variable and temporary. Medical management includes percutaneous drainage, human chorionic gonadotropin (hCG), or gonadotropin-releasing hormone

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Figure 3.36
a Crusting of the nipple associated with follicular cysts.
b Bilateral alopecia due to hormonal production of follicular cysts.
c Same guinea pig as in 3.36b with fur regrowth post-OVH.

(GnRH). Surgical intervention is the treatment of choice. *Guinea pigs who show no clinical signs associated with their cystic ovaries do not require treatment.*

MEDICAL MANAGEMENT

Percutaneous drainage

Ultrasonography should be performed first to ensure there is no sign of ovarian neoplasia. Percutaneous drainage is painful and should be performed under deep sedation or general anaesthesia. Aseptically prepare the area. The cystic ovary is stabilised against the abdominal wall and drained via a needle and syringe. *The effect is temporary, and the cysts will refill between 2 days to a few weeks later.*

Risks

Any time a needle is penetrated into the abdomen there is a risk of sterile or infectious peritonitis. Failure to ensure there is no ovarian neoplasia could result in seeding of neoplastic cells. Uncontrolled haemorrhage is a recognised risk although

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there is little in the literature to corroborate it. Cysts may rupture and there *have been multiple deaths in guinea pigs due to hormonal overload caused by the rupture of follicular cysts during percutaneous drainage*. The hormone levels contained in the cyst fluid are 3–10 times higher than seen in plasma/serum.

Given the risks and that the effect is temporary (a few days at the shortest), my opinion is that it is not clinically justifiable. If the cysts are follicular and the owner will not opt for surgical treatment, then medical management can be attempted. Owners should be counselled that this too will be temporary and also will not address the likely concurrent uterine disease.

Human chorionic gonadotropin (hCG)

hCG is given by IM injection at a dose of 1000 IU/guinea pig, repeated 7–10 days later. The *large volume required is painful* and may need to be injected into two sites to ensure IM. hCG causes an immune response, stimulating the production of antibodies which may make *subsequent doses less effective*. Owners should be counselled there is also a *risk of anaphylaxis with repeated injections*.

Gonadotropin-releasing hormone (GnRH)

GnRH is also given IM at a dose of 25 μg/guinea pig repeated 2 weeks later. The smaller volume is better tolerated by guinea pigs.

Deslorelin

GnRH agonists like deslorelin implants have been shown to have no effect on cyst size and in one study there was a higher incidence of uterine disease in implanted guinea pigs. It is not recommended to use these implants in guinea pigs. Male guinea pigs who received deslorelin implants remained fertile.

SURGICAL TREATMENT

Ovariectomy is not recommended due to the high incidence of concurrent uterine disease. The treatment of choice in *symptomatic* guinea pigs is ovariohysterectomy.

Ovariohysterectomy (OVH) procedure

My belief with spaying any species is that you make the wound as large as you need to. Wounds heal 'side to side' so a 10 cm wound will heal as fast as a 5 cm one. Most of the vets I see who run into problems perioperatively are trying to operate through a tiny wound.

First, learn to spay correctly. Second, learn to perform the surgery faster. Last, learn to perform the surgery through a smaller wound. It is completely unrealistic to expect a vet who is 2 years qualified to spay through a wound the same size as a 20-year qualified vet. These expectations (sometimes it is the vet putting pressure on themselves) lead to high stress levels, demoralisation, and increased morbidity/mortality.

Species-specific considerations

Apart from anaesthesia considerations, and the friability of the soft tissue, there are seven main differences between spaying guinea pigs and spaying more familiar species.

- Guinea pigs have a large caecum in close proximity to the abdominal wall, tenting the linea alba upwards is important on entry to the abdomen to prevent inadvertent piercing of the caecum.
- The primary fat storage site in sows is the mesometrium and mesovarium.
- The ovaries are near the end of the uterine horns, and the infundibulum (oviduct) runs cranial to the ovary and comes back on itself. It is important to remove the entire infundibulum as it can contain ovarian tissue which may result in further ovarian cysts growing post-operatively.
- The ovarian ligament is very short, which can make exteriorisation more difficult.
- Catgut should not be used. Guinea pigs struggle to break catgut down leading to increased risks of adhesions and granuloma formation which can cause chronic pain.
- Guinea pigs are very sensitive to pain caused by handling of their intestinal tract which can lead to significant postoperative pain.
- Guinea pigs form adhesions readily, gentle tissue handling will help reduce the risk. If swabs are required in the abdomen, then pre-soaking them with saline will reduce the risk of drying out viscera. If any of the viscera or fat becomes dry it will increase the risk of adhesions. Flushing the abdomen with warmed sterile saline post-operatively can also help reduce the risk and will hydrate your patient.

Performing an ovariohysterectomy

Holding your scalpel in a pen like grip, make a skin incision midline approximately 5 cm long starting mid abdomen.

Blunt dissect through the subcutaneous layer to expose the muscle. The thickness of the subcutaneous layer varies between individuals and in some it is almost absent. The linea alba is sometimes not as obvious as we are used to in dogs. There are often three white lines running roughly parallel – *the middle one is the linea alba*.

Grasp the linea alba in your rat tooth forceps (ensuring you have only grasped the muscle and not underlying viscera), tent the muscles up as high as you are comfortable with (the muscle layer is not overly friable) and perform a reverse stab incision. We tent the muscle to elevate away from the large caecum. Enlarge the opening in the muscles using scissors, ensuring the tips are pointing up (so we know what we are cutting).

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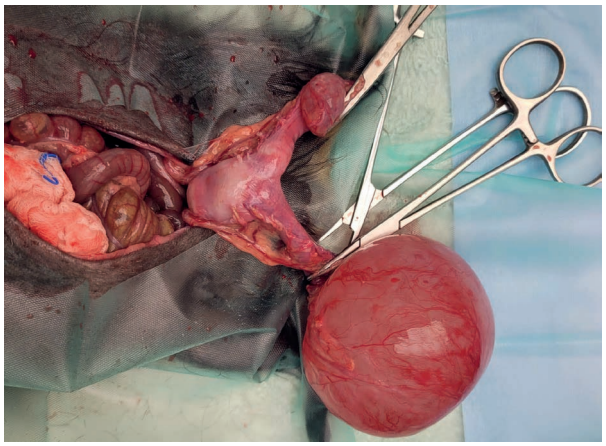


Figure 3.37 Owing to the size of the ovarian cyst the abdominal wound was extended cranially. Note the concurrent uterine mass, which is why the cervical region is so wide.

The uterus sits between the bladder and the descending colon. Locate the end of the uterine horn, the infundibulum (aka oviduct) will run cranially from the end of the horn and then circle back around the ovary. Sometimes the size of the ovarian cyst means we have to extend our abdominal incision cranially (Figure 3.37). If the ovary does not exteriorise easily, massage the fat distal to the ovary between your fingers which will often loosen the tissue. The ovarian artery runs perpendicular to the ovary. Visualisation of the artery is not always possible due to fat storage. An avascular window is present cranial and caudal to the ovary.

Holding the ovary in your non-dominant hand, break down the avascular windows to allow placement of artery forceps across the ovarian artery (or where you know it will be). *Ensure the entire oviduct is with the ovary as remnants of the oviduct left behind can cause cystic masses to form in the future*. Do not pre-crush the ligature site. Using 3 metric (2/0) Polyglactin 910 (Vicryl, Ethicon) or similar material place two ligatures. Place another artery forcep directly above the one already placed, so that the two are touching. Keeping the one closest to the ligatures still and steady, rotate and twist the other forcep away. This causes the tissues to stretch and tear, any remaining tissue can be cut.

Check for any bleeding before letting go of your ovarian stump. I leave approximately 1 cm tails on my ligatures, this allows me to find the site quickly if I am worried about a bleeding stump.

The uterine artery runs parallel to the uterine body. Approximately one-third of the way along the uterine horn it forms multiple branches. Ligating before it branches is much easier. Massage the mesometrium to find the avascular window on either side of the uterine artery. Place your artery forcep through these windows and across the artery. Do not pre-crush the ligature site. Using 3 m (2/0) Polyglactin 910 (Vicryl, Ethicon) or similar material place two ligatures. Place another artery forcep directly above the one already placed, so that the two are touching. Keeping the one closest to the ligatures still and steady, rotate and twist the other forcep away. This causes the tissues to stretch and tear, any remaining tissue can be cut. Check for any bleeding before letting go.

My preference is to remove the uterus through the cervix. Place one artery forcep across the cervix, using 3 m (2/0) Polyglactin 910 with a swaged-on needle (Vicryl, Ethicon), place a transfixing ligature. I then place a circumferential ligature.

Place another artery forcep directly above the one already placed, so that the two are touching. Keeping the one closest to the ligatures still and steady, rotate and twist the other forcep away. This causes the tissues to stretch and tear, any remaining tissue can be cut. Check for any bleeding before letting go.

Check all stumps for any signs of bleeding then flush the abdomen with warmed sterile saline.

Repair the muscle layer with cruciate sutures (3 m (2/0) Polyglactin 910 with a swaged-on needle (Vicryl, Ethicon)), lifting the muscle to see placement of the needle at all times (I have seen students stitch the muscle onto the caecum when operating on cadavers). Cruciate sutures counteract tension in every direction, they are faster to place and have few knots.

Further analgesia can be provided by performing local anaesthesia splash blocks on closure. A mixture of Lidocaine 2mg/kg and Bupivacaine 1mg/kg can be used. Doses higher than this may lead to toxicity.

If there is a subcutaneous layer, this can be closed (1.5m (4/0) Polyglactin 910 with a swaged on needle (Vicryl, Ethicon)) using a simple continuous pattern then an intradermal pattern is used for skin closure or use staples. *Guinea pigs have quite thick skin layer and I will often use surgical staples to close the skin wound*. EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) may be applied to the wound. Warn owners if surgical staples have been used as some owners find the appearance shocking.

Buster collars should not be used on guinea pigs – they create stress (which leads to anorexia) and prevents eating of caecotrophs. The majority of guinea pigs will not interfere with their wounds if they have adequate analgesia on board, which should be continued for at least ten days. Restrict exercise post-operatively for a week.

Ovariectomy (OV) technique

Bilateral flank ovariectomy has been advocated in young sows to prevent cystic ovaries and to prevent uterine disease as the ovaries are removed prior to sensitisation of the uterine tissue. This is a less invasive technique which has the added advantage of limiting the manipulation of any intestinal tissue. OV is not recommended as a treatment for symptomatic cystic ovaries as concurrent uterine

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disease is extremely common therefore an OVH would be the treatment of choice in symptomatic cases. *Yuan et al. (2017) reported that ovariectomy is linked with an increased likelihood of developing osteoarthritis*.

Approach to the ovary is similar to flank spay in cats but the flank area is prepped on both sides. Some surgeons prefer to perform this with the guinea pig in ventral recumbency.

A 2 cm skin incision is made starting an inch caudal to the last rib and an inch distal to the epaxial muscles of the spine. The abdominal muscles are incised and the ovary should be just below or very close to the abdominal wound. Ensure the entire oviduct is removed with the ovary as remnants of the oviduct left behind can cause cystic masses to form in the future. Place artery forceps across the ovarian artery, using 3 m (2/0) Polyglactin 910 (Vicryl, Ethicon) or similar material place two ligatures. Place another artery forcep directly above the one already placed, so that the two are touching. Keeping the one closest to the ligatures still and steady, rotate and twist the other forcep away. This causes the tissues to stretch and tear, any remaining tissue can be cut.

Check for any bleeding before letting go of your ovarian stump. I leave approximately 1 cm tails on my ligatures, this allows me to find the site quickly if I am worried about a bleeding stump. The muscle layers are repaired with 1.5 m (4/0) Polyglactin 910 with a swaged-on needle (Vicryl, Ethicon) using a cruciate pattern and intradermal skin sutures are used.

UTERINE DISEASE

CYSTIC ENDOMETRIAL HYPERPLASIA (CEH)

The most frequently diagnosed uterine lesion is cystic endometrial hyperplasia, which was seen in 66% of female guinea pigs in one retrospective study.

Endometritis/pyometra

As in more familiar species, this can occur in breeding or non-breeding sows. Clinical signs may include a bloody or purulent vaginal discharge (if an open pyometra is present), depression, anorexia and pyrexia. Definitive diagnosis utilises cytology, bacterial culture and sensitivity of the vaginal discharge and ultrasound of the uterus. Treatment of choice includes stabilisation, systemic antibiotics, analgesia and OVH (Figure 3.38).

If an owner wishes to breed from the sow again or does not want surgical intervention then medical management can be attempted with three SC injection of aglepristone (Alizin 30 mg/ml, Virbac Limited) 10 mg/kg given on day 1, day 2 and day 8, stabilisation, systemic antibiotics, analgesia. Owners should be advised that as in more familiar species the sow will be at an increased risk of recurrence at the next oestrus.



Figure 3.38 A vaginal discharge associated with a pyometra.

Uterine neoplasia

The vast majority of uterine neoplasia are benign in the female guinea pig with an increased incidence seen in animals over 3 years of age. The most commonly diagnosed neoplasms in descending order of frequency are leiomyomas, adenomas, and leiomyosarcomas (see Figure 3.37). Leiomyomas are frequently found in close association with the bladder, which may complicate surgical resection. Adenocarcinomas have also been documented. Laik-Schandelmaier *et al.* (2017) noted that in the cases of malignancy they saw, no evidence of metastasis was found. Owing to the fact that benign lesions outnumber the malignant and that metastasis may be uncommon, surgical OVH should be considered the treatment of choice.

Routine OV/OVH

If we consider cystic rete may be normal in guinea pigs (the vast majority of them are asymptomatic), that follicular cysts (which are symptomatic) account for only 22% of ovarian cysts and that uterine neoplasia is frequently benign and apparently slow to metastasise, I personally cannot see how routine ovariectomy or OVH can be ethically recommended. Especially as there may be a link with increased risk of osteoarthritis. *Rather than make a blanket recommendation on a surgical procedure I recommend OVH on a case-by-case basis guided by the clinical signs and diagnostic*

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tests. The vast majority of female guinea pigs I have treated have lived a long and apparently good quality life with their reproductive tract intact.

Dystocia

Parturition in the guinea pig is normally complete within 15–40 minutes. The pubic symphysis begins to spread approximately 10 days before parturition and the gap can be palpated externally. Once the gap has reached 1.5 cm parturition can be expected within the next 48 hours. *Owners should be educated that continuous straining for 20 minutes or unproductive contractions for over 2 hours is abnormal and veterinary intervention is required.* Bloody or green vaginal discharge may be seen in cases of dystocia. Pressure from the gravid uterus or a pup stuck in the birth canal may lead to temporary paresis/paralysis of the rear legs.

PREDISPOSING FACTORS

Reasons for dystocia occurring are as varied as in more familiar species. Historically, first time breeding after the closure of the pubic symphysis (breeding at 8–12 months' old) was described as a definite cause of dystocia. Currently, many exotics vets have seen older primiparous sows birth normally despite their age. So, while it may increase the risk of dystocia it should be remembered it is not a certainty that the sow will need a caesarean section based on age of first breeding alone. Sows bred for the first time over eight months of age should be examined a week before expected parturition to palpate for opening of the pubic symphysis.

Obesity, hypovitaminosis C, abnormally large foetuses, uterine inertia, mechanical obstruction, large litter sizes (female exhaustion) can all be contributing factors in dystocia.

TREATMENT OF DYSTOCIA

Palpate the pubic symphysis, if there is less than 2.5 cm separation then surgical intervention is required. Midazolam 1–2 mg/kg IM and butorphanol 0.2–0.5 mg/kg SC can be administered as a pre-medication if the guinea pig is particularly stressed, then mask induction. *Apart from surgical entry to the abdomen (see OVH technique), the surgical procedure for caesarean is the same as in more familiar species.* Guinea pig pups are precocious and may not suckle (but will eat solid food and drink water) during the first day of life which is completely normal.

If there is 2.5 cm separation or more, then conservative treatment can be attempted first. Radiograph the sow to assess foetal size and ensure there is no mechanical obstruction, e.g. healed fracture of the pelvis narrowing the pelvic canal, or uterine torsion. If mechanical obstruction is present or maternal foetal size mismatch then progress to caesarean section.

CONSERVATIVE TREATMENT

IV or IO fluids can be given at a rate of 5–10 ml/kg/h to maintain blood pressure and counter shock. Provide heat (ideally ambient temperature to prevent burns), and maintain as stress free an environment as possible (lower lights, no loud sounds, or sudden movements, keep away from predators). Analgesia can be given via buprenorphine.

A dose of 5–10 ml of a 10% calcium gluconate (Calcivet or Zolcal D) can be given orally alongside oral glucose. If no contractions are noted then administration of oxytocin can be considered. Oxytocin administration is controversial as it can cause prolonged, violent uterine contractions. *Administering oxytocin SC rather than IM will help reduce this.* The recommended dose is 0.2–3 IU/kg. Pups should be passed within 15 minutes of oxytocin being administered.

If conservative treatment fails, then a caesarean is indicated.

Pregnancy toxaemia

Pregnancy toxaemia is a condition seen mainly 2 weeks before or up to 2 weeks post-parturition. Obesity, stress, and a large number of pups are all predisposing factors. Owing to their fastidious nature, novel foods should not be given nor should their method of water delivery be changed. Sudden changes to diet and water can result in anorexia and dehydration. Anorexia or reduced food intake (e.g. due to large uterus acting as space occupying mass) will lead to a negative energy balance and the development of ketosis. Incidence of pregnancy toxaemia is higher during hot weather therefore care should be taken to keep ambient temperature between 20–22°C (68–71.6°F).

Clinical signs include anorexia, depression, hunched posture, salivation, dyspnoea, incoordination which may progress to seizures and death. Ketones may be smelt on the breath and urinalysis will show aciduria (should be alkaline in herbivores) and ketones. Biochemistry will show hypoglycaemia, hyperlipidaemia, and hyperkalaemia. Liver enzymes may also be elevated due to hepatic lipidosis.

Prognosis is poor even with aggressive treatment. IV fluids with added glucose should be given. Oral glucose, calcium gluconate and assisted feeding (q2–3hrs) should be started as soon as the guinea pig is able to swallow. A short acting steroid (e.g. Dexadreson 2 mg/ml, MSD Animal Health UK Limited) should be administered IM at a dose of 0.2 mg/kg. Owners should be advised that steroid administration late in gestation can induce parturition or abortion.

Prevention is preferable, ensuring that stress is minimised, that temperatures do not exceed 22°C (71.6°F), adequate space is given for exercise and that familiar food is readily available at all times.

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Mammary disease

MASTITIS

Mastitis is uncommon in guinea pigs, can be caused by trauma or poor cage hygiene and clinical signs are the same as in more familiar species (Figure 3.39), antibiotic choice can be guided by culture and sensitivity of the milk. Treatment includes analgesia, systemic antibiotics, warm compresses and stripping the milk from the affected gland. Ideally pups should be weaned.

MAMMARY NEOPLASIA

Mammary neoplasia is common in male and female guinea pigs. Up to 75% of the masses are malignant in nature. Radiography of the chest is recommended to check for metastasis. Surgical removal with 1 cm margins and histopathology is recommended (see Figures 3.30a & b).

Males

Deslorelin implants have no contraceptive effect on male guinea pigs due to their unique GnRH and therefore should not be used in this species.

Mammary neoplasia

Male guinea pigs are overrepresented for development of mammary neoplasia, see above.

Smegma

Build-up of smegma is very common, often a greyish white malodorous thick layer is seen coating the penis. Owners should be taught to regularly examine the penis (once weekly) to prevent large build-up. The smegma also increases the risk of foreign bodies such as hay getting stuck inside the prepuce. Smegma itself may dry out and become hard and crumbly. Gently extruding the penis by putting pressure above the prepuce and washing the penis with warm water to remove most but not all of the smegma is recommended.

In the boar the anus develops a sac-like appearance known as the anal sac. In young boars this will not cause a problem and smegma may be found in the folds of the sac. Aged entire boars (occasionally also seen in neutered males) often lose the ability to fully expel faecal pellets, especially the softer caecotrophs. This leads to accumulation of the caecotrophs within the anal sac, stretching and flaccidity of the anal sac and local irritation and infection (Figure 3.40). The sac will need to be emptied out and gently cleaned which is very unpleasant smelling (see Video 3.1). Flamazine or F10 wound ointment can be applied to the area as required. Analgesics should also be given and concurrent osteoarthritis will make prehension of caecotrophs extremely difficult.

> Some boars will need the owners to empty this area daily, others weekly will suffice (warn owners this is a smelly job!) and ideally, they should offer the caecotrophs to the boar to allow consumption.

Testicular neoplasia

Testicular neoplasia is documented but rare in guinea pigs. Swelling of one testicle with or without atrophy of the contralateral testicle should increase the index of suspicion for neoplasia. Castration is the treatment of choice.



Figure 3.39 Mastitis in a guinea pig with rupture of the gland.



Figure 3.40 Faecal accumulation within the anal sac leading to distention and flaccidity.

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Trauma

Fighting with cage mates can lead to bites in the region of the scrotum and prepuce. Bacterial orchitis and epididymitis can result. Treatment with antibiotics and analgesics is required and severe cases may require castration.

Castration technique

Castrating guinea pigs is essentially the same procedure we do regularly in other species – we remove the testicles and want a live patient at the end of it.

Species-specific considerations

Apart from the anaesthetic considerations and avoiding the use of catgut, the only other difference is that *guinea pigs have open inguinal canals, therefore if we do not close the tunic our patient has a real risk of abdominal organs herniating into the vaginal tunic/scrotum with potentially devastating consequences*. The open inguinal canal can also make castration challenging if we do not stabilise the testicle as the slightest pressure exerted on the testicle will push it back into the abdomen. If this happens, gentle pressure just cranial to the pelvis will usually result in the testis slipping back into the scrotum.

The two techniques I teach are 'open/closed' or 'closed' castrations. When I first started castrating guinea pigs the open/closed technique gave me the confidence that I had indeed taken all of the testicle. Now *I am more confident, the closed technique is faster. I hold my scalpel in a pen-like grip* as this gives me much better control of direction and depth of my incision.

Basic technique

Hold the base of the testicle in your non-dominant hand, between your middle finger and thumb. Stabilise the testicle between your thumb and middle finger. Use your index/pointer finger to stretch the scrotal skin. Do not let go of the base of the testicle. The scrotal skin is thicker than in rabbits. Excess pressure will result in going through the skin and straight through the vaginal tunic and potentially into the testicle.

Holding the scalpel in your dominant hand, incise the *cranial aspect* of the scrotal sac, extending roughly a third of the length of the testis. Using blunt/blunt small scissors, push between the scrotal skin and vaginal tunic then open the scissors. This utilises blunt dissection to break down the attachments between the tunic and the scrotum. Repeat in all four quadrants. Using blunt/blunt scissors ensures we do not accidentally pierce the testicle.

Use your index finger and thumb of your non-dominant hand to gently squeeze the testicle caudally while your index finger and thumb of your dominant squeeze the caudal pole (epididymis) of the testicle cranially. This results in the testicle (which is still within the vaginal tunic) popping up through your incision. Once the epididymis is visualised through the tunic, we place an artery forcep across the epididymis. Now the testicle is stabilised by the artery forcep, we can let go with our non-dominant hand. Hold the artery forcep in your non-dominant hand (do not pull upward) and use a swab to help strip the rest of the scrotum down off the tunic. The tissue can be quite tough in mature boars.

Open/closed technique

Mature boars often have very thick tunics and the testicle is not always easily visualised. Guinea pigs also have a large fat pad cranial to the testicle. This means it can be difficult for inexperienced guinea pig surgeons to be confident they have the entire testicle. Making a small incision into the vaginal tunic directly over the testicle and milking the testicle through the incision allows direct visualisation and confirmation that the whole testicle will be taken. This is the 'open' part of the technique.

Place artery forceps across the tunic 1 cm cranial to the base of the testis. My personal ligature of choice is polyglactin 910 (Vicryl, Ethicon) or a similar material. I do not pre-crush ligature sites in guinea pigs as the tissue can be very friable. For a transfixing ligature use 2 m (3/0) Polyglactin 910 with a swaged-on needle (Vicryl, Ethicon), which includes the tunic, vascular bundle of the testis and the vas deferens. I also place a circumferential ligature (my preference in all species is to place two ligatures if I have space). This converts the technique back into a 'closed' technique. No viscera can herniate as the tunic is now closed.

Place another artery forcep directly above the one already placed, so that the two are touching. Keeping the one closest to the ligatures still and steady, rotate and twist the other forcep away. This causes the tissues to stretch and tear, any remaining tissue can be cut. Check for any bleeding and tuck the stump back into the scrotal sac.

Wound glue is used to close the scrotal sac.

Closed technique

The only difference is that we skip the step of opening into the tunic to check we have the full testicle.

Syringe feed on recovery and send home with a week of meloxicam.

There are some clinicians advocating the abdominal castration technique. Incising into the abdomen will increase the pain associated with castration and should be considered when planning the analgesia. *Personally, I have never had an issue with scrotal castrations and as I recommend incising the cranial aspect of the scrotal sac there does not seem to be an issue with trailing the wound on the ground*. I have seen a single case where a guinea pig who was castrated via an abdominal approach had had the urethra included in one of the ligatures of the testicular vessels with a fatal outcome.

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Gastrointestinal (GI) disease

Normal faeces are dark and elongated. They are firm and when fresh are slightly shiny/moist. Reduced appetite will lead to less frequent, smaller, darker, and drier faecal pellets being produced (Figure 3.41).

Diarrhoea

Bacterial (*Salmonella*, *Yersinia*, *Clostridia* and *Escherichia coli*) or parasitic diarrhoea is uncommon in guinea pigs, and when seen it will usually be in recently weaned pups, pregnant sows, or chronically stressed individuals.

Most cases of diarrhoea are dietary in origin, either from changing the diet too quickly, excessive amounts of fresh food stuffs, giving chilled food straight from the fridge or feeding lawn mower clippings (which should never be done). A diet low in fibre and high in carbohydrates also predisposes to diarrhoea. Some endocrine diseases may cause softer faecal pellets (see section on endocrine diseases).

Providing the guinea pig is bright, alert, and active, dietary modification alone should suffice. Withhold concentrates and salad leaves/vegetables and ensure that good quality hay and fresh clean water is available.

Some plants have an astringent quality which may help dry up the diarrhoea. Blackberry leaves, shepherd's purse, yarrow, and plantains all have this quality and are often readily consumed by guinea pigs. Owners should be advised to double check they know what they are foraging before feeding to their pets. I find that I have to advise owners not to pick vegetation on road verges (due to potential toxins) and to wash the vegetation.



Figure 3.41 Two normal faecal pellets beside a slightly smaller, darker faecal pellet from the cage mate. *The smaller faecal pellet was from a guinea pig with a reduced appetite.*

Holistic Exotic Veterinary Nutrition (HE'VN) produces a Gastrointestinal Mix in conjunction with Hop to Forage. This herbal mix contains herbs and flowers which have been shown to improve gut function.

A probiotic may be helpful to restore the gut flora, such as Avipro (Vetark). The vast majority of cases will resolve within 48 hours and the normal diet can be slowly reintroduced. Failure to resolve should alert us that there may be another underlying issue and culture and sensitivity should be considered. Some bacterial causes of diarrhoea are potentially zoonotic.

Gastrointestinal hypomotility

Gastrointestinal hypomotility, commonly referred to as gut stasis, can be a primary condition or secondary to virtually any other disease process. Pain of any origin can cause gastrointestinal hypomotility. Dehydration can also result in gastrointestinal hypomotility. Gastrointestinal hypomotility can result in bloat.

Bloat/colic

Bloat and or colic may occur secondary to GI hypomotility or as a primary issue post-ingestion of fermented food or an excess of leaves from the brassica family, e.g. cabbage or kale. Although rare, intestinal obstruction due to intussusception, omental torsion, neoplasia, or foreign body should be included on the differential list for guinea pigs with bloat.

The abdomen will be distended due to gas build-up in the stomach, the guinea pig will be reluctant to move due to visceral pain. *Guinea pigs cannot eructate the gas therefore it has to pass through the GI tract.*

Gentle abdominal massage and encouraging the guinea pig to move around will allow the gas to move through the intestinal tract faster. Injections SC of meloxicam and or maropitant may help with the visceral pain.

Gastric dilatation and volvulus

Gastric dilatation and volvulus (GDV) is not uncommon in guinea pigs but is highly likely underdiagnosed due to guinea pigs dying prior to presentation. It is mainly seen in guinea pigs over 2 years of age, and is *an acute onset, generally fatal condition*. One retrospective study at a specialist exotics referral clinic demonstrated a 75% mortality rate (Nógrádi *et al.*, 2017). Treatment is the same as required in the dog.

Clinical presentation is one of acute onset severe depression, reluctance to move, abdominal distention, hypersalivation, retching, dyspnoea, anorexia, or sudden death.

Clinical exam will often demonstrate a gas filled tympanic cranial abdomen with pain elicited on abdominal palpation. They may be non-responsive, and display signs of hypovolaemic shock: tachycardia, weak pulses, pale or cyanotic mucous membranes, and hypothermia.

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Diagnosis is via clinical exam and radiography will demonstrate a large gas filled stomach which is positioned on the right side of the abdomen. Even with surgery the prognosis is poor with only approximately 25% of guinea pigs surviving. Surgical intervention involves shock fluid therapy (normal maintenance is 4 ml/kg/h), multimodal analgesia, thermal support and repositioning the stomach (including a pexy) as per the dog.

Awareness of this as a differential, prompt diagnosis, and prompt intervention may well increase survival rates in the future.

Euthanasia

End of life conversations can be a difficult area to broach with some clients. Occasionally owners will wait for us to broach the topic as they are 'worried' if they do so we will think they are not dedicated enough. Reassuring clients that sometimes we need to love our pets enough to let them go when the time is right can be invaluable.

Many owners will want to stay with their guinea pig during this procedure and the process should be fully explained including possible events such as sudden jumping, the need to change veins or gasping. I administer a pre-med (e.g. medetomidine/butorphanol or midazolam/ketamine (maintains venous pressure) injected SC or IM), once the guinea pig has fully relaxed, I will use the lateral saphenous vein by choice, to inject the pentobarbitone, to allow the owners to stroke the guinea pigs head.

I advise allowing the bonded cage mate to see its companion after euthanasia. I advise clients that I believe it allows the cage mate to accept that its companion will not return. Over the years, many owners have reported to me they felt the cage mate was looking for its companion constantly after losing them and certainly anecdotally this is reduced when they get the opportunity to 'say goodbye'. Whether this is purely anthropomorphic or not is up for debate, however it demonstrates to your owners that you care and you listen to their concerns.

Many owners will opt for private cremation with return of their pets' ashes and all options that your practice offers for dogs and cats should also be made available to your guinea pig clients. I offer a lock of fur, footprints (coloured ink pad from craft shop and a card). I send sympathy cards if I know the patient and owner well.

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4

Rats

Pet rats, also known as the brown rat (*Rattus norvegicus*) are an underestimated species. They are incredibly *intelligent, highly social, bond tightly to their owners, display affection and have been proven to have empathy*. In my experience owners of pet rats are often adults and are highly invested in providing the best possible care for their pets.

They are myomorph rodents, their incisors continuously grow, and the labial surface of the incisors have iron deposition within the enamel resulting in an orange brown colouration. Conversely, their cheek teeth do not grow throughout life and are close rooted. The gestation period is 21 days and they birth altricial young.

The average lifespan is commonly quoted as 2–3 years, although in my experience 1.5–2 years is much more commonly seen. As a prey species, they have evolved to hide disease and pain to prevent predation. Despite being kept in captivity for various reasons (lab, sport, pets), rats still maintain many of their wild ancestors' behaviours.

Educating owners on husbandry and how to improve the welfare of their pet (e.g. environmental enrichment) should be part of a standard veterinary visit. New pet owners may be more likely to seek advice but every day is a school day and we need to be aware that owners may confuse love and dedication to their pet with appropriate welfare, this is where husbandry registration forms are invaluable to allow us to have a non-confrontational overview of how the pet is being kept.

The ethology of rats in the wild can provide important insight on husbandry practices that meet the rats' evolutionary requirements. Rats originated in South East Asia and evolved to tolerate higher temperatures and humidity than is often achieved in captivity. They are omnivores and as such evolved to have animal protein in their diet. Wild rats live in burrows, can have large home ranges (>1500 m^2) and have a complex social structure. Colonies of rats are made up of family groups containing 2–6 individuals. The whole colony shares a 'mutual scent', which allows colony members to recognise each other. Family groups generally will consist of one or two related males and a harem of females.

Companionship

Although rats do bond very tightly with their owners and will elicit play with people, there is an innate requirement for conspecific company. Even the most dedicated owner cannot fulfil the behavioural repertoire of another rat. Single sex groups, or mixed sex, if neutered, can be kept. Entire males will live peaceably together, provided they are raised together from a young age and have a large enough enclosure.

However, *it is important to realise that rats have a complicated societal hierarchy*, and in the wild, if individuals do not get on, they will defer to non-confrontational displays of dominance/submission to prevent injury (which would weaken both individuals).

They also will have the ability to remove themselves from the situation as their home range is far larger than most people realise. If a family unit is not getting on, quite simply they will move and start their own unit – this cannot happen with the pet rat. The intricate nature of the burrow system will also prevent 'bottlenecks'. 'Bottlenecks' could restrict access to important resources for the more submissive rat, and careful consideration of this potential problem during enclosure design is vital to reduce stress.

Play

Owners can sometimes misinterpret play as fighting. Young rats especially indulge in rough and tumble play. Educating owners on specific behaviours to examine can give owners more confidence in correctly interpreting the behaviours on display.

- During play the coat is smooth, they will alternate roles, they will pin each other on or nibble the nape of the neck/over the shoulders, and there will be no injuries.
- During fights the coat will be standing on end, they do not alternate roles, they bite the rump region, there may be bite wounds and the weaker rat will try to hide away.

Rats like to tickle each other, and they laugh! Unfortunately for us, without special microphones (a bat detector will work) their laugh is outwith the frequency of our hearing (most of rats vocalisations are in the

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ultrasound frequency, the chirrup interpreted as laughing is produced at 50 kHz).

Rat tickling is a technique developed by researchers to improve emotional wellbeing of research rats by mimicking the normal rough and tumble play of young rats. *This rewards the rats for interacting and causes a positive emotional response to be associated with handling.*

Allowing the rat to approach the handlers hand, then allowing the rat to following the handlers hand back and forth (mimicking a playful chase) progresses onto the handler grasping the rat over the shoulder/nape of neck region in a pinning motion (mimics the rats play pin) for ten seconds then rolling the rat over and the handler uses their fingers to tickle the rats abdomen in the same way they would a human child. Each phase of the play should be limited to short interactions of 15 seconds and should be stopped immediately if the rat becomes distressed – it is supposed to be fun!

Enclosure and enrichment

Poor enclosure design can result in chronic stress, and immunosuppression, which can result in the rats own respiratory microbial flora becoming opportunistic pathogens. Over the last 20 years I have seen a steady decline in respiratory disease cases in rats and I believe that a large part of the decline in cases is due to improvement in the rats' enclosures, especially the rising popularity of the large, multi level cages. Neville *et al*. (2022) recommended as an absolute minimum an enclosure of 90–120 cm × 90–120 cm and stressed that a much large enclosure is advised. There is no such thing as too large a cage, the larger the better.

Positioning of the enclosure within the house is important. Rats hear into the ultrasonic range of frequencies and can find ultrasonic sounds emitted by televisions, stereo systems, or refrigerators distressing. Likewise, vibrations caused by washing machines or tumble driers may also result in chronic stress.

Rats are social and like to be part of the action of the house. The ideal location would be in a busy area of the house while not next to the above appliances, and not in an area of intense light (see Lighting section). If the cage is not a tall multi-levelled cage, then placement of the cage upon a table will ensure the rats are more at human eye level. *Having the cage prominent is a visual reminder to owners on cleanliness but also allows the rats to interact more with the owners while in their cage*.

Multi-level cages need multiple hides, feed stations, and access water. It is important to provide enough resources so that all rats can have free access rather than having limited resources which can allow dominant rats to prevent the more submissive rats from accessing them.

Nest boxes with soft nesting material should be provided. *Cotton wool type nesting material should be avoided as it can become tangled around toes leading to avascular necrosis*. Using kitchen or toilet roll paper as nesting material gives the added fun of allowing the rats to shred the paper to their preferred length and width for nesting.

Fleece hammocks are popular, are easily washed and despite having several within an enclosure, it is not uncommon to have the entire mischief of rats piled up asleep in the same hammock.

Ensuring different materials and textures are used for toys and play obstacles improves environmental enrichment. Plastic tunnels can be used either as floor hides, or suspended from the ceiling of the cage by chains thereby creating a swinging hide. Providing multiple hides also increases the rats' ability to control its own exposure to light (see Lighting section).

Commercially there is a huge range of toys aimed at rodents, but some of their favourite toys can be the ones owners make themselves. It is important to provide safe wooden toys to provide ample gnawing opportunity, which is important for maintaining the length of their incisors while also satisfying their innate requirement to gnaw. Using household items e.g. a scarf hanger as a 'cargo net', provides climbing opportunities and also can be utilised as another pathway between levels.

Providing a deeper area of substrate to allow the natural and normal requirement of digging/burrowing behaviour is advisable. If the cage size or design does not permit a dig area, an adjoining structure could be made allowing a tunnel into a dig box. Clothes peg tubs can be easily slotted onto the side of the cage and used as a resting place or a foraging toy. Egg boxes or small paper bags can be used to hide food, creating foraging toys.

Various substrates are available. The ideal one is absorbent, dust free and free from perfumes. Shavings may be suitable for some rats, whereas others may develop allergic rhinitis. Paper-based bedding with dust extracted is available. Allowing the rat to shred cardboard and or toilet paper to mix through the substrate provides environmental enrichment while potentially making the substrate more suitable for building tunnels.

Solid wheels can be provided for running. When rodent wheels have been placed outdoors, various wild rodent species have chosen to utilise it, spending time running! This demonstrates that it is indeed a pleasurable or at least a valued resource. *To prevent painful spinal conditions, it is important to provide a large enough wheel that they do not have to bend their back while running*. They should be able to run with a straight back and without having to lift their head. Most commercially available wheels are far too small, as for most rats the wheel should have a diameter of at least 40 cm (16 inches). There are various YouTube videos educating owners on how to make their own safe rat wheels.

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Diet

Some texts refer to rats as opportunistic omnivores, which I believe strongly is a misnomer. To my mind an opportunistic omnivore is a species that eats carrion, or kills a prey item it stumbles across rather than actively hunting. Rats hunt, some colonies of wild rats specialise in the type of prey hunt, e.g. fish, nestlings etc. They are omnivores.

Commercial rat nuggets should form the main part of their daily diet, supplemented with small amounts of vegetables, fresh fruit, and daily treats. Small amounts of animal protein should be offered twice a week. Suitable daily treats include breakfast cereal, mealworms, egg, lentils, beans, peas, and chickpeas.

Rather than provide their daily rations in a bowl, scatter feeding in snuffle mats or providing foraging toys/puzzle feeders increases time taken to eat and helps mimic natural foraging.

> Sick rats often do not show the same level of interest in their daily treat, so owners can use this opportunity to gauge the health status of their rat, it may help alert an owner earlier in the course of the disease.

Owners should be counselled that diet has a massive impact on life span. Studies have shown that providing *a diet with 20% fat (rather than the recommended 5–10%) is life shortening and a diet too high in protein (>22%) is associated with a higher incidence of chronic nephritis*.

Rats perform coprophagy much to some owner's disgust. They will eat approximately 10% of their own faeces from the floor of the enclosure, and *they will also consume cage mates' faeces – this helps contribute to the colony scent*.

Water

Clean water should be provided by sipper bottles at multiple stations. The average water consumption is: 8–12 ml/100 g body weight/day.

Lighting

As rats are primarily nocturnal/crepuscular they evolved to be awake during periods of low light intensity. *They have poor eyesight and when exposed to levels of light comfortable for humans (often our homes will be 150+ lx), retinal atrophy and cataract formation can rapidly occur*.

Albino rats are particularly sensitive to light levels (they lack the protection of melanin in the iris, so even when the pupil is constricted, light will shine through the iris itself) and should not be exposed to light levels of 130 lx or greater.

It should be remembered that in our clinics the light intensity will be much higher than 150 lx, and particularly in theatre. *Anaesthetised rats cannot blink or protect their eyes from light; therefore it is important that not only do we lubricate their eyes but that we protect them from prolonged exposure to light. This can be as simple as taping their eyes closed during surgical procedures*.

Sexing

Sexing rats is performed by looking at the genital–anal distance.

Male

Male rats have a much longer distance between the genital opening and the anus compared to the female. Mature male rats also have very large, long testicles that are situated caudally to the prepuce and often obscure the anus, unless of course he has been castrated (Figure 4.1). *Male rats do not have nipples*.

Female

Female rats have six pairs of nipples and develop a small hairless ring around each nipple. The female rat has three urogenital openings close together. The urethral papillae, the vulvar opening (covered with a membrane unless in oestrus) and then the anus (Figure 4.2).

However, *if dealing with a wriggling adult rat, I will often look for the presence of nipples as a row of bald dots on the ventral abdomen as a fast way to sex them*.

Preventative health care

There are no vaccinations available for rats and routine worming or ectoparasite control is unnecessary.

Restriction of calories to 70% of ad lib feeding has been shown to greatly reduce the incidence of mammary tumours, which is a great incentive for owners to prevent obesity.

Neutering

Ovariectomy at 90 days of age reduced the incidence of pituitary adenomas from 66% to 4%. Several studies have



Figure 4.1 Male rat.

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Figure 4.2 Female rat.

also shown that early ovariectomy or ovariohysterectomy (between 5–7 months of age), dramatically reduces the incidence of mammary tumours, from 73.8% to 5.3% in one study (Planas-Silva *et al.*, 2008).

Ovariohysterectomy

Ovariohysterectomy is performed using a standard midline approach with the skin incision starting midline, between the umbilicus and pubis. The uterus is identified between the bladder and the descending colon and the uterine horns are followed cranially to identify the ovaries which are exteriorised. Perform an ovariohysterectomy as in other species, using 1.5 m (4/0) Polyglactin 910 as ligature material. Close the muscle layer using cruciate sutures using swaged-on 1.5 m (4/0) Polyglactin 910 and close the skin wound using an intradermal pattern with the same suture material. EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) may be applied post-operatively to supply further analgesia. Good analgesia will prevent wound interference by the animal.

Castration

Male rats have open inguinal canals. Failure to appreciate this and take steps to close the canal during castration can lead to inguinal herniation of abdominal organs. Castration is performed using a similar technique to rabbits, which ensures the vaginal tunic is closed off effectively closing the inguinal canal.

Hospitalisation

As a prey species, extremely sensitive to stress caused by the sight, sound, and smell of predators, there are several factors we need to take into consideration.

In an ideal world we would all have prey species wards. For many practices that is not the reality. Other options include setting up hospital cages, which are just small pet rat cages, in a quieter area of the clinic.

The ward should be kept warm, studies have shown that rats preferred temperature during the day is 27°C (80.6°F) falling to 24°C (75.2°F) at night (Ray et al., 2004). This is often much higher than owners will expose them to. A digital max/min thermometer and hygrometer (purchased inexpensively from a well-known online retailer) allows me to monitor the temperature and the humidity within the room.

Prey species do not like absolute silence. In the wilderness there is always birdsong or other animal noises unless there is a predator around. For this reason, playing music at a low level is recommended. Interestingly there is a variation on music preferences between species and *multiple studies have shown that Mozart has a positive effect on stress, anxiety, learning ability, and hypertension in rats, yet was aversive for hens*. I use Pet Remedy (Pet Remedy Ltd) plug-ins in each of my wards to try and help mitigate stress while hospitalised.

How the cage is set up is also important. Slippery surfaces are stressful and can result in injuries. Lining the floor with paper and providing hide with nesting paper (toilet tissue) or a fleece blanket and/or snug. The hide can be a cardboard box as they are easily disposed of and of ready supply in practice.

Check with the owner how they normally provide water. Most owners will use a sipper bottle as rats tend to readily contaminate bowls of water with bedding.

For elective procedures vets or front of house staff can request the owners bring their rat a 'packed lunch' of favourite and familiar foods. Owners often love this phrase and I have experienced some well packed and personalised lunch boxes! If the rat is being admitted as an emergency, then I will ask the owner to bring in some of their normal food if it is not a food I already have. Familiar food is important even if it is not ideal, for example, muesli. Once the pet has recovered, that is the time to gradually change the diet to a more advisable one. *Rats can be highly suspicious of unfamiliar foods*.

Failure to admit the bonded cage mate can result in stress and anxiety, the breakage of the bond, fighting, injuries, upset owners, and it may be difficult to re-bond the rats. *I always advise admitting the bonded cage mate at the same time*. Owners are, in my experience, happy to bring the cage mate in once they understand why.

In cases where there are more than three rats in the mischief (three cage mates is my limit), I will ask the owner to bring in the one it spends the most time with. Scent swapping (rubbing each group with a clean towel then

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using the towel to rub the other group transferring the scent) to ensure both groups are marked with the communal scent can help ensure a smooth reintroduction at home.

Hospitalisation sheets should be used and the animals weighed twice daily.

Venipuncture sites

Using EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) makes venipuncture much easier. Ensure the room temperature is approximately 25°C (77°F), and / or use hot hands (latex or rubber gloves filled with warm water) to warm the tail.

My veins of choice are the lateral tail vein (Figures 4.3a & b) for intravenous (IV) catheter placement and lateral saphenous or gingival vein for blood sampling. The lateral tail vein is often deeper than it appears, 2–3 mm below the skin. I regularly obtain 2 ml samples from this site although I often use only a 23 g (occasionally a 25 g) needle and allow the blood to drip directly into the blood collection tube. *Having your nurse 'pump' the vein by very slowly raising and releasing while you flex and unflex the tail allows good flow of the blood, but excessive pressure should not be applied as squeezing of the tail can result in a lymphocytosis.*

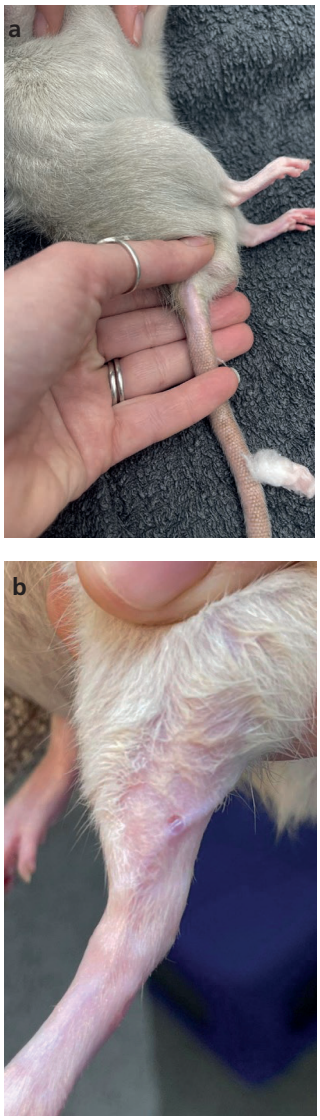


Figure 4.3
a Lateral tail vein.
b Lateral saphenous vein.

Rats have a very sensitive tail and may interpret the restraint and raising of the vein as painful. It is for this reason and that they find restraint for venipuncture incredibly stressful that I anaesthetise rats with isoflurane for venipuncture.

As with more familiar species one vein is often bigger than the other, therefore I clip fur over the vein at the base of the tail. I apply approximately 1 mm thick layer of EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) enough so there is a noticeably white patch, then I get the rest of my equipment organised. In my experience, EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) takes around 5 minutes to cause venodilation, some local anaesthesia and does not require a dressing to work. *Pick your catheter size judged on the size of the vein, not the size of your patient.* For most rats I will end up using a 24 g (yellow in the UK) catheter, but occasionally I have had to use a 26 g (purple in the UK) catheter.

Placing IV catheters in rats is not that difficult. Winged catheters are much easier to tape in as you can bend the wings around the tail. Dressing tape such as Micropore (3M) is applied proximal and distal to the cannula in exactly the same manner we would in our canine patients.

Elizabethan collars ideally should not be used in rats. Elizabethan collars make eating food more difficult, the eating of caecotrophs impossible and is an unnecessary source of severe stress. Rats also like to play with Elizabethan collars, spinning them which can lead to lacerations of the neck.

Alternative sites for venipuncture are the gingival vein, ventral tail artery, dorsal tail vein or the jugular vein. I rarely utilise the jugular vein.

Gingival vein

Use of this vein results in no haematoma, minimal bruising, and no impact on food prehension. Once anaesthetised, the rat is placed in dorsal recumbency. A 25 g needle and a 1 ml syringe is required. The vein is located midline, immediately below the gingival – incisor edge, between the mandibular incisors (Figure 4.4a). Pull the lower lip caudally. The vein is entered in a caudal direction using a 30–60 degree angle and penetrating the mucosa 2–4 mm deep. Once there is blood in the hub of the needle the lower lip can be released (Figure 4.4b).

Many exotics texts describe anaesthetising the rat and performing a cranial vena cava stick to draw blood. This carries the risk of death due to uncontrollable haemorrhage. Laboratory vets and technicians have perfected this technique with very few bad outcomes, but they are performing this very regularly whereas clinical veterinary staff do not.

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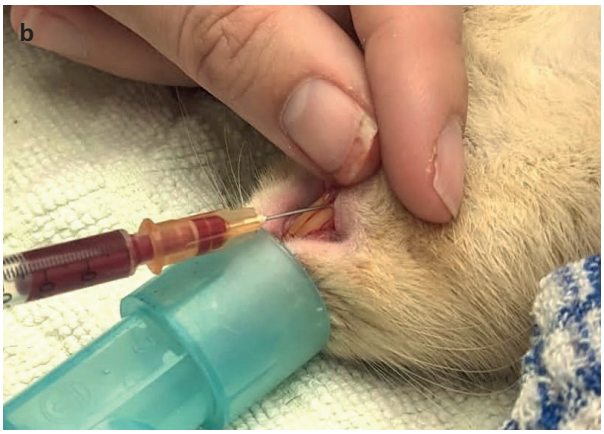


Figure 4.4
a The site of the gingival vein highlighted by the yellow dot.
b Gingival vein sampling.

Personally, I would not do this to one of my own pets therefore I will not do it to a client's.

The maximum volume of blood that can be safely taken is 0.7 % of their body weight. Therefore, in a 350 g rat we could safely take 2.45 ml which is more than we need to. Most biochemistry machines can run a profile on 0.3 ml of blood.

Biochemistry and haematology profiles can be run 'in-house' using our usual mammalian profiles or sent to an external laboratory. Both options have pros and cons. The main pro for running in-house is that you obtain your results quickly whereas the main con is that you have to interpret whereas many labs will provide interpretation upon request.

The main leucocyte in rats is the lymphocyte which accounts for approximately 75% of the leukocytes in the peripheral blood. See Tables 4.1 and 4.2 for reference ranges for biochemistry and haematology. *Acute stress does not affect the neutrophil/lymphocyte ratio but chronic stress results in an increased ratio due to a relative neutrophilia and a lymphopenia, each still being within recognised reference ranges*.

Dehydration

Hydration status in rats can be challenging to ascertain. Skin tent, eye position, tacky mucous membranes, cool limbs, altered mentation, and weight loss can all be utilised but, in my experience, may only be obvious in severely dehydrated patients. This may be referred to as interstitial deficits.

Severe blood loss or sepsis will result in hypovolaemic shock (perfusion deficits) necessitating fluid resuscitation rather than simple fluid therapy.

> Rodents in hypovolaemic shock tend to be bradycardic (heart rate <200 bpm), hypotensive (systolic blood pressure <90 mmHg (normal 90–120 mmHg)) and hypothermic (<36°C (<96.8°F)(normal 38–39°C (100.4–102.2°F))).

I tend to *assume that any ill rat is at least 5% dehydrated, more if there is diarrhoea. 10% dehydration will be accompanied by severe skin tenting, very dry mucous membranes, and potentially dry looking eyes, greater than 10% dehydration will be accompanied by signs of hypovolaemic shock*.

Blood pressure measurement

Indirect blood pressure measurement is the most used method in my experience in general practice. Many practices already have the required equipment: a Doppler ultrasound probe to detect the arterial flow, a pressure cuff to occlude the arterial flow, and a sphygmomanometer to measure the pressure. Just as in more familiar species, the size of the *cuff used should approximate 40% of the circumference of the limb used*. If a cuff is used which is too large it will give falsely decreased pressures. Small cuffs that are used in human new-borns (especially ones for premature babies) can be used in rodents. These can often be sourced from human medical websites or eBay.

Sites for cuff placement are the carpus, tarsus, or base of the tail. The Doppler should be placed distal to cuff and shaving the fur on the contact point may be necessary. The normal systolic blood pressure in a conscious rat is 90–120 mmHg, diastolic is 85 mmHg and the mean arterial pressure is 95 mmHg.

Treating hypothermia

I do not routinely take rectal temperatures in healthy rats due to the stress the animal experiences and the higher risk of iatrogenic damage occurring. It is also recognised that in healthy rats, rectal temperature measurement actually induced hyperthermia (Dangarembizi *et al.*, 2017). In ill, dehydrated patients, a soft rubber tipped thermometer can be used to check the rectal temperature to allow any hypothermia to be identified, corrected, and monitored. *Failure to correct the hypothermia can result in failure to stabilise and an increased risk of pulmonary oedema forming*.

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Table 4.1 Rat biochemistry.

BIOCHEMISTRY (Heatley & Russell, 2020)	REFERENCE RANGE (UK units)	REFERENCE RANGE (Us Units)
ALKP	40.3–58.8 IU/l	40.3–58.8 U/l
ALT	41–89 IU/l	41–89 U/l
AST	44.5–153.5 IU/l	44.5–153.5 U/l
AMYL	128–313 IU/l	128–313 U/l
GGT	0–3.8 IU/l	0–3.8 U/l
LDH	575–725 IU/l	575–725 U/l
CK	350–450 IU/l	350–450 U/l
GLU	5.69–7.08 mmol/l	102.5–127.5 mg/dl
CHOL	2.13–3.63 mmol/l	82.3–140.0 mg/dl
TRIGYLCERIDES	1.3–3.7 mmol/l	51.25–143.25 mg/dl
BA	20–40 µmol/l	0.79–1.57 mg/dl
TBIL	3.42–6.84 µmol/l	0.2–0.4 mg/dl
UREA	4.46–6.25 mmol/l	12.5–17.5 mg/dl
CREA	8.84–114.92 µmol/l	0.1–1.3 mg/dl
TP	65.8–75.2 g/l	6.58–7.52 g/dl
ALB	38.8–46.3 g/l	3.88–4.63 g/dl
GLOB	13–36 g/l	1.3–3.6 g/dl
CA	2.50–2.27 mmol/l	10–11 mg/dl
PHOS	1.44–2.84 mmol/l	4.7–8.8 mg/dl
K	4.45–5.15 mmol/l	4.45–5.15 mEq/l
Na	146–152 mmol/l	146–152 mEq/l

Table 4.2 Rat haematology.

HAEMATOLOGY (Heatley & Russell ,2020)	UNITS	REFERENCE RANGE
Red blood cells	10^{12}/l	6.6–9.7
Packed cell volume	%	44–62
Haemoglobin	mmol/l (g/l)	80.68–117.91 (130–190)
Mean corpuscular volume	Fl	60–70
Mean corpuscular haemoglobin concentration	pg/cell	17–22
Mean corpuscular haemoglobin	mmol/l (g/l)	16.14–21.2 (260–340)
Reticulocytes	%	0–2
Platelets	10^{9}/l	700–1600
White blood cells	10^{9}/l	1.9–12
Differential blood count (relative values)		
Monocytes	%	0.1–1.0
Lymphocytes	%	10–75
Neutrophils	%	19–53
Eosinophils	%	0–5
Basophils	%	0–1.3

Rewarming should take place relatively fast over 1–2 hours using a mixture of direct and indirect heat sources. 'Hot hands' (latex gloves filled with warm water) and snuggle safe microwavable heat pads are good if the rat can move away from the source of heat should it become uncomfortable. Incubators can be cheaply made using plant propagators, a reptile heat pad (approximately half to two-thirds the length of the propagator) and a thermostat with a probe, e.g. HabiStat. Regular monitoring of the rectal temperature is critical to prevent hyperthermia developing.

Fluid therapy

We should not be frightened of fluid therapy in small rodents as *the principles are basically the same as more familiar species*, albeit more closely aligned with the principles we use in the cat rather than dogs. The main differences are that changes can occur rapidly, necessitating close monitoring and *in virtually all cases the fluids should be warmed to 38–39 °C (100.4–102.2°F) prior to administration*. Use of a bottle warmer can assist in this for keeping the fluids warm.

Oral rehydration with assisted feeding should not be underestimated but given that maintenance is believed to be 75–100 ml/kg/day (3–4 ml/kg/h) intravenous fluids should be used in any rat that is over 5% dehydrated.

The rate of and type of the fluids given depends on the rate of ongoing fluid losses and the clinical status of our patient.

Fluid resuscitation

If there is evidence of perfusion deficits (hypovolaemia), then fluid resuscitation is required. Fluid resuscitation in small mammals is performed similarly to the cat, using a combination of crystalloids (for interstitial dehydration) and colloids (as stays in the vascular space these fluids help with hypovolaemia).

Ideally, we would check the systolic blood pressure of our patient, but I acknowledge this is not always feasible. Checking the systolic blood pressure allows us to tailor our fluid resuscitation to the required volume to restore normal blood pressure.

The fluids should be warmed to 38–39°C (100.4–102.2°F) prior to administration. Give a bolus of an isotonic crystalloid (I use saline), 10–15 ml/kg, followed by 5 ml/kg of a colloid administered slowly over 5–10 minutes. The blood pressure should then be rechecked, and once 40 mmHg has been reached, maintenance fluids only should be given until the patient has been rewarmed to 36.6°C (98°F). Several rounds of bolus crystalloids and slow IV colloids may be required every 10–15 minutes to achieve the required increase in blood pressure.

Once the core temperature has reached 36.6°C (98°F), 5 ml/kg of a colloid given slowly over 15 minutes should be administered, followed by blood pressure measurement, repeated until the systolic blood pressure is over 90 mmHg. The heart rate should normalise also.

> Once stable, i.e. normal heart rate, normal core temperature, normal systolic blood pressure, normal capillary refill time, dehydration level can be assessed and the rehydration phase of the fluid therapy can begin.

Rehydration

If there is evidence of interstitial dehydration but the patient is cardiovascularly stable, then 80% of the fluid deficits can be replaced over 12–24 h. If the interstitial volume is rapidly lost then it should be rapidly replaced over 4–6 h. *As 80% of administered crystalloids enter the interstitial tissue less than one hour after administration, crystalloids, e.g. saline, are perfect for this type of dehydration.*

The fluid volume required is calculated:

deficit (body weight in g × % dehydration) + estimated ongoing losses (ml) + maintenance (ml)

For example, a 346 g rat with respiratory disease presents approximately 5% dehydrated with no blood loss or diarrhoea: there are no ongoing losses in this case, maintenance is believed to be 75–100 ml/kg/day.

Deficit (346 × 5%) = 17.3 ml
Maintenance (0.346 × 100) = 34.6 ml
Total fluid volume: 17.3 + 34.6 = 51.9 ml
13.84 ml (17.3 × 80%) + 34.6 ml = 48.44 ml can be given over 12–24 h

How to give the fluids

Oral rehydration is often forgotten about. In patients that are less than 5% dehydrated, normothermic and have a functioning digestive tract, palatable isotonic fluids can be syringed orally.

I do not agree with subcutaneous fluid administration. It is *incredibly painful* and dehydrated patients will not absorb fluids via this route as efficiently. Rats hide pain so relying on the fact your patient 'allows' you to perform subcutaneous administration is not rationale for it not being painful. IV access is easier than you think in rats using either the lateral tail veins or the lateral saphenous (Figure 4.5) (see also Venipuncture section).

Peritoneal injection of fluids carries risks but a bolus of 10–15 ml/kg can be given in the lower left quadrant, with the patient restrained with its head lower than the hind quarters utilising gravity to shift the abdominal viscera cranially. A peritoneal bolus may help facilitate intravenous access via catheter in severely dehydrated patients.

If IV access cannot be achieved, then sedation (if not moribund), local anaesthesia and intraosseous (IO) administration of fluids should be considered as in more familiar

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Figure 4.5 24 g IV catheter in lateral tail vein.

species. In rats my preferred IO site is the proximal tibia although it is rare for me to need to place an IO catheter. After surgical preparation of the site, a 23–25 g needle (surgical steel suture material (sterile) as a stylet) is placed in a normograde fashion, a radiograph should be taken to ensure correct placement as inadvertent puncture of both cortices is a common complication in the tiny species (Figure 4.6). *IO placement is contraindicated in any patients that have sepsis, osteomyelitis, or metabolic bone disease.*

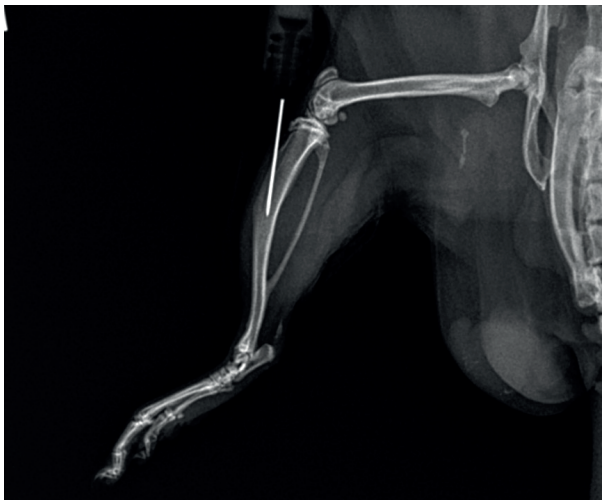


Figure 4.6 Radiograph checking IO catheter placement.

IV or IO catheters can be connected to syringe drivers or boluses can be administered every 30–60 minutes. The bag of fluids can be maintained at the correct temperature using a baby bottle warmer. *Ongoing analgesia is important, IO catheter flushing and administration of fluids is painful in humans therefore should be considered painful in other species.*

Hydration status should be assessed throughout the day, which enables you to alter the rate and volume of fluids therapy as required. My intravenous fluid of choice in most cases is saline.

Stress (environmental, inappropriate handling or severe pain) will further immunocompromise an already ill patient. In fact, there is good evidence that *stress increases the intensity of pain experienced*. Ensuring the rat is kept away from predators, and that good quality analgesia is given, utilising the same decision-making skills we employ in analgesia selection for dehydrated dogs and cats. I also utilise Pet Remedy plug ins (Pet Remedy Ltd) which are clinically proven to reduce stress and I have found them particularly useful in rats both in the clinic and for owners to use at home.

Assisted feeding

The term assisted feeding is preferable to force feeding when discussing with owners. There are high-quality, purpose made foods available such as: Harrison's recovery or EmerAid Omnivore. EmerAid and Harrisons both give information on recommended daily volumes. If your clinic does not stock these products, then thankfully many rats will readily take gently warmed human baby food from a spoon or a syringe.

Some rats will require wrapping in a towel (burrito technique) for assisted feeding. Some individuals will tolerate smaller, more frequent feeds better.

Rats can be highly suspicious of new foods (neophobia) therefore the rats' normal diet should always be readily available to encourage eating on its own.

Drugs contraindicated in rats

If in doubt, always check the formulary as it will often tell you whether it is safe to use a drug in any given species. My favourite formulary is James Carpenter's *Exotic Animal Formulary* now on its sixth edition (Carpenter & Marion, 2022).

There are very few drugs contraindicated in rats. As they are such popular laboratory animals there is often a lot of information available albeit there may not be efficacy studies. Rats are not as susceptible to antibiotic induced gut dysbiosis. Clavulanated amoxicillin for example, is safe to administer orally to rats.

The main class of drugs I rarely use, and if I do use it, it is with care are immunosuppressives. Steroids such as dexamethasone and prednisolone have their place in rat medicine, but I always caution the owners on the risk of immunosuppression potentially further complicating the

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condition. I will usually only use a steroid if a non-steroidal anti-inflammatory has failed to control the clinical signs.

Species-specific anatomy/behaviour

Rats are myomorphs with elodont – hypsodont incisors, (elodont: continuously growing throughout life and hypsodont: long anatomical crown compared to the root). They have two incisors in the maxilla and two in the mandible. The lower incisors are much longer than the upper incisors and are not as rigidly fixed in position as the upper incisors. This is due to the mandibular symphysis being cartilaginous to allow shock absorption during gnawing. The incisors usually have a yellow orange colouration due to the iron deposition within the enamel.

The cheek teeth are anelodont (anelodont: limited growth period therefore truly rooted).

Dental formula: 2 × (I 1/1; C 0/0; P 0/0; M 3/3) = 16. The tight cardiac stomach sphincter prohibits vomiting or eructation. They have short gestation periods (21 days) and birth altricial young.

There is no gall bladder in rats.

Male rats have renal tubular production of alpha globulins therefore, proteinuria (<0.3 g/l) is a normal finding in male rats.

Using a towel or a blanket on the table to reduce the slippery nature of the surface makes rats feel safer and they are less likely to struggle, although I tend to perform most of my clinical exams with them either in my arms or on my knee. Prey species have evolved to be prey, therefore have an innate requirement to escape predation at a split-second notice. This is why they find a slippery surface so frightening, if they cannot find purchase, they cannot escape predation. Using the blanket or towel reduces the chance of them injuring themselves or you as their handler.

Male rats have open inguinal canals. Failure to appreciate this and take steps to close the canal during castration can lead to inguinal herniation of abdominal organs.

Rats hide illness and pain. Again, from an evolutionary point of view this makes complete sense because if you show illness or weakness, you get eaten! So, by the time a rat looks overtly ill or painful, they literally no longer care if they live or die. Many owners underestimate this and unfortunately many of us, the veterinary profession also underestimate it.

Any SC injections should be given over the flanks to avoid the large area of brown fat storage rats have dorsally. Fat, in particular brown fat, results in unpredictable absorption of drugs.

Pain in rats

One of our jobs as veterinary professionals is to *actively look for pain*. In Chapter 1 we discussed that all vertebrates have the same anatomical and physiological pain pathways.

> If we accept that animals and people share similar mechanisms of pain and analgesic action then we should realise that the variation of analgesic efficacy in different individual people means that it is highly likely that a similar variation will be seen in animals – further reinforcing the need for regular pain assessments.

Rats show very subtle behaviour changes associated with post-operative pain and studies have shown that in rodents *the most significant signs of pain occur within the first 12–24 h post-operatively, therefore application of a local anaesthetic at the site of the incision could greatly reduce post-operative pain*. Abdominal contraction, back arching, twitching, and weight shifting at rest are all signs associated with post-operative pain. Rats will often freeze when in the presence of an observer so these signs can easily be missed if video surveillance is not an option or be mistaken for resting. Choosing to sleep on their side rather than tucked in is also a sign of pain.

Acute and surgical pain is easier to appreciate but what about chronic pain especially arthritic pain?

Chronic pain negatively impacts quality of life severely. It causes chronic maladaptive stress, activation of stress hormones, sleep disruption, immunosuppression, and reduces social interactions (which for an innately social species further exacerbates stress levels).

A grimace scale has been developed for rats and can be found at: https://www.nc3rs.org.uk/3rs-resources/grimace-scales/grimace-scale-rat.

One of the challenges associated with assessing pain in rats is their defence mechanism of self-induced analgesia. In the prolonged presence of a predator (which includes you!) they induce endogenous opiates which result in approximately 45 minutes of analgesia. Brief exposure induces less potent analgesia. *This can make accurate assessment in pain levels extremely difficult unless the rat is a well socialised individual who likes being handled even by strangers*.

The first signs of pain commonly seen in rats are: rough coat (from reduced grooming), a hunched body posture (slight arch of the back often thoracolumbar), a slower possible shuffling gait, pacing/restless when normally would be sleeping, abnormal sleeping position (stretched out instead of tucked), self-isolation from the group, personality change (previously friendly now aggressive or vice versa), drooling (if oral pain), anorexia and associated weight loss. In today's busy world these signs are commonly missed by owners until they become very obvious.

Ensuring that during *our history taking we target our questions* to include mobility can help. We already do that with cats as they tend to show more behavioural adaptations to osteoarthritic pain compared to dogs but we often forget to do that with rats (stop hitting your panic button). Recognition and alleviation of pain are an essential component of every animal's care.

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How many veterinary professionals genuinely perform the exact same clinical exam in a rat that you do in a dog? How many do a full skeletal exam, spinal palpation, and limb range of motion in every rat patient? If you do not do this – why not? *Osteoarthritis and spinal pain is massively under diagnosed in rats simply because we do not always think to look for it.*

Placed on a towel, or your knee for security, the neck flexion tests are performed exactly the same as in more familiar species. Extending the forelimbs one at a time allows you to differentiate if more pressure was required to extend one limb therefore identifying pain. View the animal from above, does the rat lean more to one side (taking pain off the sore side) or is a limb abducted?

Spinal palpation requires the exact same level of pressure that you would use in a cat. Failure to perform correctly will lead to failure to spot pain. Remember that the thoracic spine runs between the scapula and needs palpating too!

Rats may well vocalise if you palpate a painful area especially of the spine, but more frequently, I will feel vibrations as the epaxial muscles spasm. Back pain can sometimes be confused with abdominal pain and vice versa (same as more familiar species).

Self-injurious behaviour (e.g. chewing through skin) can be seen especially with chronic and or neuropathic pain.

Manipulation of the tail by winding it through your fingers gives a good indication of flexibility. *The rat tail is partially prehensile and will normally curl and grasp your fingers as it moves through them. I find that one of the first signs of joint pain can be found in the tail as a loss in the flexibility and gripping ability of the tail.* This improves with analgesia which leads me to conclude it is a reliable indicator of osteoarthritic pain in the tail (see Video 4.1 clinical exam for skeletal pain).

Osteoarthritis in rats

Owners can be shocked or initially disbelieving of a diagnosis of osteoarthritis in a rat of one year of age (which is halfway through the lifespan of many rats), yet I frequently diagnose osteoarthritis in that age group. How I explain this seemingly strange occurrence is discussed below.

Osteoarthritis is a degenerative condition affecting the cartilage and bones of a joint. It is commonly referred to as the wear and tear arthritis. Wild animals do not live as long as captive animals (many die before even reaching reproductive age) due to a combination of pressures such as: predation, disease, or trauma (cars, etc.).

In captivity (protected from such pressures) wild animals live longer. Therefore, the evolutionary pressures on prey species have selected for fast moving individuals that breed at a young age, and produce many young.

From an evolutionary point of view, by the time a rat is 10 months to 1 year of age it is already skeletally old. To me it makes complete sense why 'young' domesticated rats develop wear and tear arthritis, as evolutionary pressure did not select for a robust skeleton designed for longevity. We need to remember that if your average lifespan is 2 years, by 1 year old the animal is already middle aged!

The added pressures that domestic rats face such as enclosures that limit proper movement, inappropriate substrate (which can result in increased concussive forces on joints), and obesity, further compound the damage and pain caused by the wear and tear arthritis.

Once this is explained to owners, I find they completely understand why this has occurred in their beloved pet and they are much more willing to work with me.

Radiography can be utilised to investigate osteoarthritis or spinal pain in exactly the same way as more familiar species (Figures 4.7a–e).

Joint supplements

There are a lot of studies in rats on the effectiveness of chondroitin sulphate in controlling the pain associated with osteoarthritis and promoting cartilage regeneration. Nosivets *et al.* (2021) showed that it is high-quality, pharmaceutical grade chondroitin rather than the actual chondroitin characteristics that is the most important consideration. *It is important that we stress to clients that very high-quality chondroitin is required which not all sources will be.* Recommended doses vary between 150–450 mg/kg/day.

Guan *et al.* (2022) *found the addition of curcumin (20 mg/kg) to chondroitin (100 mg/kg/day) was significantly more effective than chondroitin alone.* The oral co-administration of curcumin and chondroitin sulphate not only better attenuated cartilage damage but also alleviated the inflammatory response, delayed the progression of osteoarthritis, improved the thickness of the synovium, and allowed a quicker return to joint usage and improved range of movement. Due to these results curcumin should also be advised as a supplement.

Pain

Chronic pain associated with osteoarthritis is a major welfare concern. It is underdiagnosed in practice mainly due to the rats' astounding ability to hide pain. I often diagnose osteoarthritis both during clinical exams and radiographically from 1 year of age.

Pain associated with osteoarthritis can be found when a full skeletal exam is performed (see clinical Exam section). *Owners may note their rat is climbing less frequently and may interpret that change in behaviour as just old age as opposed to pain* because of wear and tear.

Analgesia

Multimodal analgesia should be started (see Pain management section). Supplementation with curcumin and high pharmaceutical grade chondroitin will also provide analgesic effects (see Joint supplements section).

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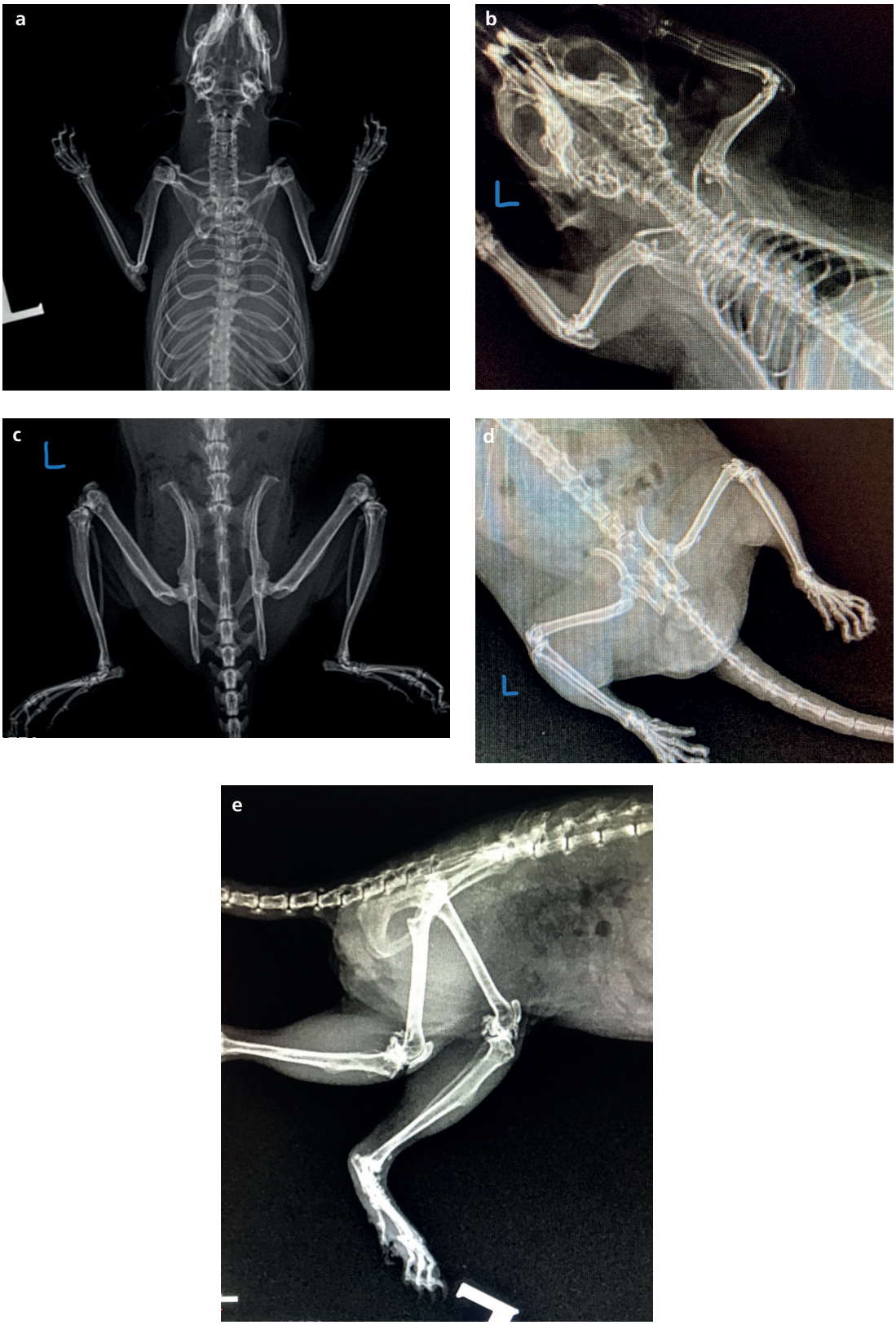


Figure 4.7
a Radiograph of normal shoulders and elbows (abnormal lung fields).
b Radiograph of OA in the shoulders and elbows.
c Radiograph of stifles with mild OA.
d Radiograph of severe OA in the stifles.
e Radiograph of severe OA in the stifles.

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Husbandry changes for rats with osteoarthritis

Lifestyle changes can make a big difference to quality of life. Ensuring the floor of the enclosure and exercise areas are non-slip and padded will reduce concussive injuries.

Reduce weight slowly in obese individuals and ensure regular short periods of exercise to prevent stiffness of the joints and gradually increase muscle mass. Scatter feeding concentrates through snuffle mats will gently increase exercise. Ensuring deep layers of substrate will help cushion the joints and providing heat discs, e.g. Snuggle Safes, during cold spells will reduce pain associated with the cold.

Neurology in rats

Hind limb weakness

Aged rats will commonly present with weakness in the hind legs. *This may be pain related due to undiagnosed osteoarthritis, spinal disease, or radiculoneuropathy*.

However, aged rats are very prone to a condition that reminds me of chronic degenerative radiculomyelopathy (CDRM) in the German Shepherd dog. In rats the condition has been termed Radiculoneuropathy and may be seen in as many *as 90% of rats over the age of two* years of age (Haines, 2010).

Clinical signs

Radiculoneuropathy occurs spontaneously and like CDRM, has variable speed of progression. The first sign noted by the owners may be paresis or a 'drunk' hind limb gait or scuffing of the hind feet (which may also be the first sign of osteoarthritic pain in hips/spine). This can progress onto complete hind limb paralysis. The ability of the tail to be partially prehensile is lost and this may progress to complete loss of tail function. Urine and faecal incontinence may also occur as well as muscle wastage of the hind legs and lumbar spine.

Treatment of radiculoneuropathy

The condition is not curable and treatment is palliative with close monitoring of quality of life. A non-steroidal anti-inflammatory drug (NSAID) (my drug of choice is usually meloxicam) should be given as there will be concurrent co-morbidities such as osteoarthritic pain. Vitamin B oral and/or parental administration has been shown to attenuate inflammatory and neuropathic pain in multiple species including the rat and can be incorporated into the treatment plan. Environmental changes such as fleece bedding (to reduce soft tissue trauma from paretic gait), lowering resources such as: food, water, and a sleeping hammock, to the lowest level, ensures easier access.

Pituitary adenoma

Pituitary adenomas are very common in aged rats with female rats being overrepresented. Vergneau-Grosset (2016) found that *75% of female rats that develop mammary fibroadenomas and 59% of female rats that develop other mammary gland tumours also have concurrent pituitary tumours*. It should also be borne in mind that pituitary tumours are common in rats and that although a link has been suggested between pituitary tumours and the development of mammary fibroadenomas it has not yet been proven. Obesity and high protein, high calorie diets have also been linked with development of pituitary adenomas.

Clinical signs

Clinical signs associated with pituitary adenomas include: head tilt, ataxia with normal muscle strength (owners may describe rat as weak due to the ataxia), circling, hind leg paresis, wide based stance, reduced appetite or anorexia, weight loss, difficulty holding food with front paws (proprioceptive defects), depression, and seizures.

Treatment

The condition is not curable and treatment is palliative with close monitoring of quality of life. Cabergoline 0.6 mg/kg q72hrs PO may temporarily reduce the size of the pituitary tumour and some rats get up to 8 months of good quality life on treatment (Eguchi *et al*., 1995; Mayer *et al*., 2011). I will also give analgesia as they will be experiencing headaches as well as dealing with the pain of comorbidities such as osteoarthritis.

Head tilt

Otitis media/interna (often secondary to mycoplasma) can cause a head tilt, ataxia, torticollis, and circling, see dermatology section for further information.

Pain management in rats

Consideration of the type of pain we are looking to control will lead to logical choices of the type of analgesics we select (Table 4.3). *Multimodal analgesia is recommended* just as in more familiar species as it has been shown to provide better relief from pain (Oliver *et al*., 2017).

> We should remember that affective and cognitive processing of pain occurs as much in rats as in primates and dogs, meaning that mice and rats are not somehow 'less sentient' species. Spinieli *et al*. (2022) demonstrated that persistent inflammatory pain is linked with anxiety-like behaviour and increased levels of blood corticosterone in the rat.

Rats have a much higher metabolic rate than our more familiar species and as such their required doses will be much higher. I *manage owners' expectations* by explaining that 'pain winddown' can take 8–12 weeks in chronic pain cases but that I reassess after 2 weeks. Asking owners to focus on three normal behaviours for their pet

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Table 4.3 Rat Analgesia.

Analgesic	Dose
Meloxicam	0.6–2 mg/kg q12hrs PO SC
Paracetamol (acetaminophen)	25–50 mg/kg q8–12hrs PO
Buprenorphine	0.5 mg/kg q8–12hrs OTM 0.03mg/kg q6hrs SC (these doses may not be effective in female rats)
Tramadol	20–30 mg/kg q6–12hrs in female rats, up to 40 mg/kg in males PO
Gabapentin	10–30 mg/kg q8–12hrs PO
Maropitant	2 mg/kg SC
Methadone	1–2 mg/kg SC q4hrs dependant on pain scoring
Amantadine	25 mg/kg PO q24hrs
Amitriptyline	5 mg/kg PO q24hrs
Lidocaine	2 mg/kg (maximum 4 mg/kg)
Bupivacaine	1 mg/kg (maximum 2 mg/kg)
Acupuncture	

can also help their perception of whether their pet has improved.

> There is a lot of data suggesting that the currently used dosing intervals for many analgesics are inadequate, further underlining the absolute requirement for regular assessment of pain and analgesic effect.

Oral or parenteral vitamin B has been shown to reduce inflammatory and neuropathic pain in many species and can be used as part of the multimodal analgesic approach.

Many of the studies on post-operative pain in rodents have demonstrated that the most significant signs of pain (i.e. overwhelms their ability to hide pain) occur in the first 12–24 h post-operatively. Applying EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) immediately postoperatively to the wound will help mitigate this and add to the multimodal approach.

My preferred NSAID is meloxicam. I have been using meloxicam at 0.6–2 mg/kg q 12 hrs for around 20 years now, although many cases I start at the 0.6 mg/kg initially for chronic pain but evidence suggests that at least 2 mg/kg is required post-operatively to achieve sufficient analgesia. Some data exits suggesting that some individuals require up to 5mg/kg but I have never personally gone above 3mg/kg in rats. Personally, I evaluate response to analgesia and adjust as required. Just as with more familiar species I utilise meloxicam in many types of pain. *As rats frequently eat, I am not concerned about administering directly with food in this species*.

Paracetamol (acetaminophen) works well if given in conjunction with meloxicam. I order mine from my usual wholesaler and use 25–50 mg/kg q8–12hrs. *Chen et al. (2018) demonstrated that 50 mg/kg dosing of paracetamol not only reduced pain but had an anti-anxiety effect whereas higher doses of 300 mg/kg induced anxiety and reduced or removed the rats ability to experience pleasure*.

In my experience tramadol works extremely well for various types of moderate pain. In humans, around 20% of the population do not get any analgesic effects from tramadol and I am highly suspicious the same will be true in some animals, most likely due to the lack of a particular enzyme. Liquid tramadol is often more cost effective when obtained from a compounding company. However, many rats will take the contents of 50 mg capsules dissolved in a known quantity of water (therefore providing you with a mg/ml solution), which the owners can make up daily for oral administration. Interestingly, Taylor *et al.* (2016) *found that the dose for analgesia varied dramatically between the sexes of the rats*. Doses 20–40 mg/kg provided analgesia for female rats, but male rats needed 40 mg/kg before any analgesic effect was noted. I use 20–30 mg/kg q6–12 hrs for female rats and 40 mg/kg q6–12 hrs in males. Opiates can cause pica in rats which may be associated with feelings of nausea or gastrointestinal discomfort. It is usually bedding material they will ingest. Maropitant may offset this side effect.

Gabapentin works well for neurogenic pain and is my go-to analgesic if I am suspicious of disc disease. The added benefit is that it helps with anxiety which can be part of chronic pain issues in all species – even human! The dose may need to be adjusted up if the patient has been taking the medication for a period of time. *This further highlights the importance of regular pain scoring*. I start my gabapentin at 10 mg/kg q 12 hrs and will go up to 30 mg/kg q8hrs. *Some rats, much like some humans, lose the analgesic effectiveness of gabapentin within a few weeks* but Kimura *et al.* (2016) demonstrated that sodium valproate, a drug used in epilepsy, (200 mg/kg q12hrs PO) could be used as a rescue drug to establish the analgesic effectiveness of gabapentin. Raza *et al.* (1996) showed that sodium valproate also has anti-inflammatory properties in rats and mice, therefore not only providing analgesia through rescuing the effectiveness of gabapentin but also in its own right.

Maropitant can be useful for visceral and skeletal pain, administered by subcutaneous (SC) injection at a dose of 2 mg/kg. The exact mechanism is unknown, and more work needs to be done on how much analgesia is provided but I have certainly found it to be useful in some cases. Also, just because rats cannot vomit does not mean they cannot feel nauseous.

Buprenorphine 0.5 mg/kg q8–12hrs can be given by the oral transmucosal (OTM) route which will provide similar analgesia levels as 0.03 mg/kg SC. Only the single use vials can be used in this manner as the multidose bottle contains

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preservative which is very bitter tasting. *If given by the SC route lower dose of 0.03 mg/kg should be used*. Taylor *et al.* (2016) found that the dose for analgesia varied dramatically between the sexes of the rats. Doses 0.5–0.6 mg/kg orally provided significant analgesia for male rats, but female rats did not show a significant analgesic response at any of the tested doses. Buprenorphine can cause pica in rats which may be associated with feelings of nausea or gastrointestinal discomfort. It is usually bedding material they will ingest.

Methadone has a short half-life in rats, but it is useful in cases of extreme pain. I use 1–2 mg/kg SC, this will last 4 hours and further dosing is dependent on pain levels. Opioids can cause problems with gut motility but so can pain. Pica has been observed in rodents that have been administered opioids.

Amantadine is an antiviral which works on the NMDA receptors and provides analgesia as part of a multimodal approach. Yu *et al.* (2016) found that 25 mg/kg PO q24hrs *also had an antidepressant like effect on chronic stress in rats* and improved cognitive ability.

Amitriptyline 5 mg/kg PO q24hrs can be used to help break the cycle of self-injurious behaviour as part of a multimodal approach (Green, 2021). *It has also been shown to reduce anxiety, improve learning and have an anti-inflammatory effect*. Care should be taken if it is currently dosed with tramadol to avoid serotonin storm, although in practice this seems to be rare.

Acupuncture has shown great success in alleviating chronic pain as part of a multi-modal approach and is frequently used in my practice. It has been shown to work by inducing endogenous opiates and naloxone will reverse analgesic effects of acupuncture. Acupuncture also stimulates fast pain fibres, essentially 'quietening' slow pain fibres.

Clinical exam

I always place a towel or blanket on the table to reduce the patient's stress. If possible, take the top off the carrier to enable easier, less stressful removal of the rat from the carrier. See Table 4.4 for biological data.

I will look at the rat face-on to check for facial symmetry and the presence of porphyrin (aka red tears) which may look black if they have dried (Figure 4.8). Owners should be reassured that the presence of red tears is a *nonspecific sign of stress* in rats and although is commonly present in cases of disease, it can also be that they are stressed for a more innocent reason including they did not like the way they were caught! (See Respiratory disease section for more information on red tears).

Table 4.4 Biological data.

Parameter	Reference range
Average longevity	1.5–2 years
Respiratory rate	70–100 bpm
Heart rate	280–500 bpm
Body temperature	38–39.5°C (100.4–103.1°F)
Maintenance fluid therapy	75–100 ml/kg/day
Sexual maturity	6–8 weeks
Gestation	21 days



Figure 4.8 Dried appearance of porphyrin at the eyes and nose. The fact it has had time to dry shows a decrease in grooming activity of the rat.

Personally, I prefer to perform the rest of the examination with the rat either on my knee or on my arm. I perform the same clinical exam, in the same order that I do for any other species.

I start by massaging the rat's ear as rats love their ears being rubbed! This helps reduce their stress and reassures the owner that you like their rat (owners are emotionally involved with their pets just as we are with ours).

Is there any evidence of nasal discharge? As obligate nasal breathers, rats fastidiously groom their face and nose, so a minor nasal discharge may not be evident at the nares – check the medial aspect of their front legs. Some owners may be concerned their rat is experiencing a nose bleed but invariably when examined, the 'nosebleed' will turn out to be excessive porphyrin (red tears) coming down the nasolacrimal ducts mimicking a nose bleed.

Palpate the mandible for any masses or peripheral lymphadenopathy.

I check for ocular symmetry from above the head, is there any evidence of exophthalmos? Are the eyelids normal? Take note of any ocular discharges. Is there any

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inflammation? Is there any sign of ectropion? Rex coated rats are overrepresented. Are any cataracts present? Fluorescein can be applied to check for corneal ulcers.

Palpate the base of the pinnae where they join the skull and visually inspect the pinnae.

A skeletal examination performed the same way as more familiar species and as previously discussed in the Pain section is my next step. I also check for thoracic compliance by compressing the thorax laterally mid thorax. Lack of thoracic compliance indicates that there is pathology in the thorax, such as thymoma, lymphoma, abscessation or dilated cardiomyopathy to name a few. Sometimes this subtle finding is the only clinical sign indicating radiography.

Thoracic auscultation is performed on both sides and listening to several areas on each side. Take note of any adventitious noises but also of any areas of dullness. If you are finding it difficult to differentiate between upper respiratory sounds and lower respiratory sounds – listen to the ventral neck! We do this all the time in brachycephalic dog breeds. I do not count heart rates in rats as I find it impossible with heart rates between 280–500 beats per minute. If it is beating, I cannot count it (if I can count it, the rat is bradycardic), and the rhythm sounds normal, I move on.

Check through the coat and examine the skin. We are looking for the same issues we would see in any examination of fur and skin: lumps, scabs, alopecia, dandruff, pustules, sores, wounds, and fleas (plus any other ectoparasites). Examine the genital region. Is there any evidence of urinary scald or faecal build-up? If the patient is an entire male palpate the testis and check for sperm plugs at the prepuce. Examine the anus. Examine the palmar and plantar aspects of the feet for any sores.

Gently palpate the abdomen. Take note of any areas producing repeatable flinches (pain) and any abnormal masses. Check for tympany or gas build-up. Are there faecal pellets palpable? It really is just a gentler version of the same examination we do in other species.

I often examine the incisors last as most rats will find this a little stressful. It is only the incisors that continuously grow in the rat and malocclusion is not common. When it occurs, it will normally be due to trauma, infection, or neoplasia.

The easiest way I find to examine the incisors is by holding the rat to my chest using my dominant hand, my non-dominant hand restrains the head from above and the thumb of my dominant hand pulls down the soft tissue of the chin.

It is completely normal for the upper incisors to be short (approximately 4 mm) and the lower incisors should normally be at least twice the length of the upper incisors (approximately 8 mm). The enamel contains iron which causes the orange/yellow pigmentation (Figure 4.9). *The mandibular symphysis remains cartilaginous throughout life in the rat which enables increased flexibility of the mandibular incisors*. This is important since rats (and the other myomorph rodents) are the 'true' gnawers, having the increased flexibility reduces concussive forces experienced during gnawing of hard materials. This flexibility is also why in cases of malocclusion, the mandibular incisors may twist and flare laterally in relation to the upper incisors.

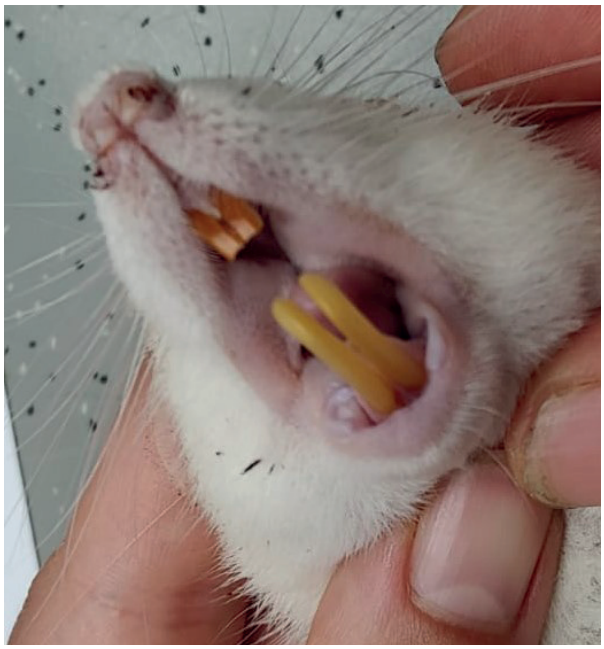


Figure 4.9 Normal incisors in the rat.

The upper incisors will grow along the tight curve of the reserve crown and circle back into the oral cavity. Assess for the presence of an oronasal fistula in cases of malocclusion as the lower incisors will often cause soft tissue trauma caudal to the upper incisors and in severe cases the upper incisors can grow around into the hard palate (see Dental disease section for treatment options).

The molars of rats are close rooted and rats can develop periodontal disease, dental caries, tooth decay and abscessation especially if fed on an inappropriate, high sugar diet. Extraction may be required but in practice conditions affecting the molars of rats are uncommonly seen.

Accurate assessment of the molars can only be performed properly under a general anaesthesia although a conscious oral assessment can be attempted using an otoscope. Personally, I rarely do this in conscious rats as if I am worried about dental issues, I will be advising an anaesthetic plus radiographs and full assessment anyway.

General anaesthesia

Rats cannot vomit and therefore they do not need to be starved before an anaesthetic. Owners are used to starving themselves or their dogs and cats prior to surgery, so we need to be clear when we book rats in for surgery that they should not be starved. Rats also have a limited glycogen reserve,

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so starving them may induce a hypoglycaemia with potentially fatal consequences. Advise owners to bring a 'packed lunch' of favourite and familiar foods.

> Bonded cage mates should always be admitted, not only will this reduce stress during the hospitalisation period, help protect the bond between conspecifics, it also has been shown to reduce healing times and increase coping ability for aversive or painful experiences (Denommé and Mason, 2022).

Allow rats at least an hour to acclimatise to the ward after travelling to the clinic to reduce stress levels prior to induction.

Pre-anaesthetic blood tests

The need for these is debated among vets. Personally, I advise they are an option but acknowledge in the young healthy individual the vast majority will be within reference ranges. The benefit is that it will give you a personalised reference range for this individual patient and as rats are so good at hiding disease, occasionally we get unexpected results which may alter our plans. Another consideration is that rats find restraint for venipuncture incredibly stressful and that isoflurane is recommended to perform the procedure.

Blood can be taken from the lateral saphenous vein, gingival vein, or the lateral tail vein. There is nothing wrong with aseptically dripping the blood from a needle into the collection tube when using the tail or leg vein. Biochemistry can be run 'in-house' using the same pre-op/comprehensive profiles used for dogs and cats (see Table 4.1 and 4.2 for reference ranges).

Pre-emptive analgesia

Once admitted (and if not already on medications) administer meloxicam 0.6–2 mg/kg and/or another analgesic if appropriate depending on the surgical procedure. These can either be given orally or by SC injection.

Pre-medication

Whether I administer a pre-medication is dependent on the stress levels of patient and the surgical procedure being performed. It is important to consider the stress of being handled and being injected. Rodents find injections very stressful and as they have small muscle masses IM injections are very painful and may need to be given in multiple sites depending on volume.

I think very carefully before I administer pre-medicants in rodents as I have to consider the type of surgery (and which analgesics will be required) and decide whether the stress and pain of the pre-med is worse than the mask induction. In my experience mask induction while wrapped in a towel burrito results in a fast, seemingly minimal stress induction, whereas injections always result in vocalisations and obvious stress. The drugs used for this are the same drugs we use in more familiar mammals. The doses can be found in Table 4.5. SC injections should be given over the flanks to avoid the high levels of brown fat found on the dorsum. Fat leads to erratic drug absorption.

Table 4.5 Premedicant dose.

Drug	Dose and route	Reversal
Midazolam	1–2 mg/kg IM SC	Flumazenil 0.1 mg/kg SC
Midazolam + butorphanol	1–2 mg/kg IM SC + 0.2–0.5 mg/kg SC	Flumazenil 0.1 mg/kg SC
Medetomidine + butorphanol	0.1 mg/kg IM + 2 mg/kg IM	Atipamezole 0.5–1.5 mg/kg IM SC

Local anaesthesia for muscle splash blocks or infiltration

Rats do not have an increased susceptibility to the toxic side effects of local anaesthesia, it is simply their small size that makes it much easier to accidentally overdose them.

Combining the drugs allows for greater effect with a lower recommended dose. Lidocaine has faster onset but a shorter duration of action (60–120 minutes) whereas bupivacaine has a slower onset of action but a longer duration (180–480 minutes). Mixing a maximum of 4 mg/kg of lidocaine with 2 mg/kg of bupivacaine in a syringe then diluting the mixture with saline to increase the volume of the solution and allow easier splash blocking (exactly what it sounds like). Drip the solution onto the wound in the muscles, or infiltrate the area. *Pulling up the required dose and pre-diluting, prevents accidental overdose*.

Induction

There are several anaesthetic protocols available for use in rats. There is an argument to be made that the safest anaesthetic is the one you are used to.

There may be a difference between how male and female rats react to anaesthetic agents. It is also known that in rats and humans, ageing decreases the dose of anaesthetic required to produce immobility in response to pain (32% reduction in dose required (Chemali *et al.*, 2015)). This is due to an increased sensitivity in the aged brain to anaesthetics. *It is important to reduce doses in aged rats to prevent accidental overdose and delays from emergence from general anaesthesia*.

My preferred inhalational gaseous anaesthetic is isoflurane. Reportedly, most rodents react more favourably to the smell of sevoflurane but in my experience, many dislike the smell just as much and in the UK, isoflurane is the only licensed gaseous inhalational anaesthetic for rodents.

My preferred method of induction is to burrito the rat and use a face mask (Figure 4.10). Have cotton buds to



Figure 4.10 Induction of rat via facemask while in a burrito.

hand to swab the mouth clear of saliva post-induction, although this often will not be necessary.

Allow four breaths of 100% oxygen (this is all that is required to fully saturate the blood with oxygen), then turn the vaporiser straight to 5% isoflurane. Initially the rat may wriggle but it will quickly become induced (most are less than 40 seconds) with minimal stress. *In the vast majority of cases the rat will not have even passed urine or faeces (which frequently occurs due to stress and commonly passed when induction chambers are utilised).*

Turn the vaporiser to 2.5% and assess anaesthetic depth (using the same reflexes we are used to in more familiar mammals, a moistened cotton bud can be used to check the blink reflex), adjusting the setting on the vaporiser as necessary.

The large cornea should be protected by applying an ocular lubricant. Owing to the sensitivity of the rat lens and retina, thought should be given to taping the eyes shut to prevent damage caused by the brightness of the surgical lights.

Intubation

I rarely intubate rats, but if I am performing surgery on the head, intubation with a 18 g IV cannula (with the stylet removed) is possible using a variation of the direct visualisation method in rabbits, although passing the cannula to the side of the otoscope speculum rather than down it (Figures 4.11a & b). The diameter of a rats trachea is 1.6–1.7 mm and full extension of the neck can increase the length of the trachea by 50% without a change in the size of the lumen.

Another option is to utilise the fact rats are obligate nasal breathers, pass a nasogastric tube medially, and ventrally into the nostril approximately 0.5–1 cm, tape the tube to the head and administer the oxygen and gaseous anaesthetic mixture via the tube.

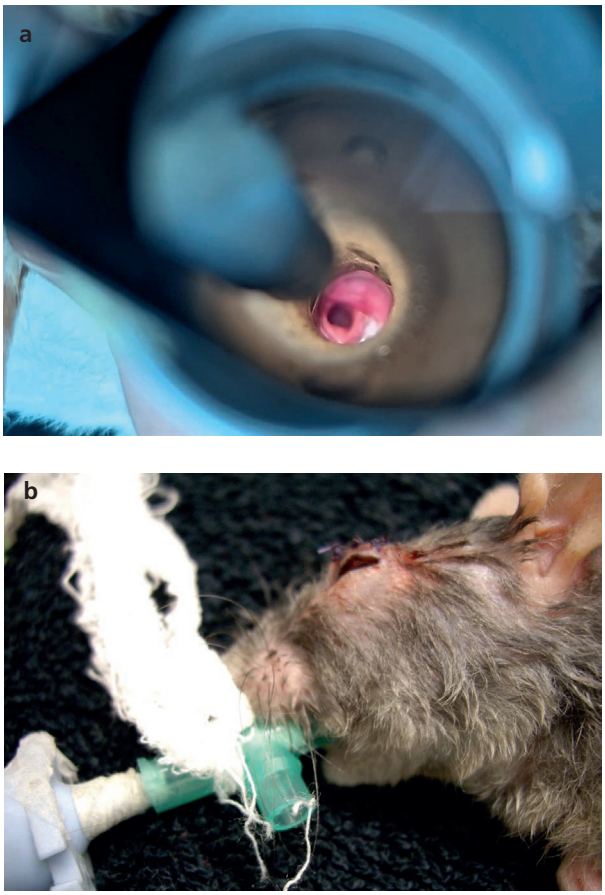


Figure 4.11
a Direct visualisation of a rat glottis using an otoscope.
b An intubated rat, using a 18 g catheter as an endotracheal tube. The rat had surgery to correct entropion.

Maintenance

I use a T-piece circuit, usually with an oxygen flow of 1.5–2 l/minute. Oxygen flow of below 0.5 l/minute results in inaccurate vaporiser output. *High fresh gas flow will result in increased chilling of the rat. To calculate a more exact flow rate required for each rat the below formula can be used:*

Oxygen flow rate = 2 × minute volume (MV)
MV = TV × RR

Tidal volume (TV) can be calculated by multiplying the weight of the rat (in kilograms) by 10 to 15. The respiratory rate (RR) for rats is 70–100 breaths per minute.

So, for example, for a 0.89 kg rat, the oxygen flow rate could be calculated:

TV = 0.89 × 10 = 8.9
MV = 8.9 × 114 = 1014 ml
Oxygen flow rate = 2 × 1014 ml = 2028 ml = 2 l/minute

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Monitoring

Monitoring the anaesthetic is the same as for more familiar species. Multiparameter monitoring is possible. MAI Animal Health makes the Vetcorder Portable Monitor, which is suitable for all species, including dogs and cats, for under £1,000. It can record: an electrocardiogram (ECG), heart rate, pulse oximetry (placed on tongue or paw), and rectal temperature. If recording an ECG rather than use Alligator clips (which can traumatise the skin), insert small gauge needles through the skin (25 g) and attach the clips to the needles.

If that is outside the practice's budget, a soft, flexible tipped rectal thermometer can be used to monitor rectal temperature. Hypothermia is a major risk factor in anaesthetic deaths. A human foetal Doppler can be purchased online (I used eBay) for around £50, ensure you request the 8 mHz probe rather than the standard 2 mHz one. An infant stethoscope can be used to monitor heart rate if the Doppler is not an option. Respiratory rates can be counted, and pulse oximeters can be used from other species but they may struggle to get an accurate reading due to the high heart rate (280–500 beats per minute in rats).

Blood pressure may be difficult to monitor as finding cuff sizes small enough for accurate readings in rats is a challenge.

If using a pulse oximeter please remember that medetomidine can cause peripheral vasoconstriction leading to inaccurate results in some patients, although in my experience if placed on the tongue I get good readings. Occasionally the spring on the oximetry probe is too strong and can occlude the vasculature, I place a cotton bud inside the probe (but not over the sensor) to alleviate the pressure and allow the reading to occur. Other options for probe placement include the feet.

Theatre

Rats have a high surface area to volume ratio that means they lose heat rapidly. The resulting hypothermia can cause fatalities. Keeping the ambient temperature in theatre high, wrapping the extremities in bubble wrap (including the tail), placing a towel on the operating table, and using a plastic drape (also allows visualisation of your patient!) are all easy ways to help mitigate heat loss. Minimise the number of times the door is opened as each time it happens the ambient temperature in the room will drop. Hypothermia leads to prolonged recovery and increased risk of death.

Positioning

Rats are diaphragmatic breathers. Their small thoracic cavity in comparison to their large abdominal cavity can make breathing more difficult due to the weight of the abdominal organs sitting against the diaphragm. Placing a folded drape or face cloth under the thorax elevates the thorax and allows the abdominal organs to move away from the diaphragm (Figure 4.12). *If the rat is not intubated, ensure the head and neck are straightened to allow for easier breathing.*



Figure 4.12 Elevating the thorax ensures the abdominal organs cannot hinder breathing. Note the injection being administered SC in the flank, rats have a lot of brown fat on the dorsum which reduces the absorption of drugs.

Surgical preparation

Rat skin is delicate and can be traumatised by inappropriate (and sometimes even appropriate) clipper technique. To conserve body temperature, minimise the surgical clip. Only one agent is used for surgical scrub, either chlorhexidine or an iodine-based skin disinfectant depending on the surgeon's preference. We do not use a surgical spirit-based spray as during evaporation this causes loss of heat. Using warm water for the surgical scrub will help reduce heat loss.

Transparent, plastic surgical drapes are ideal as they allow visualisation of the rat while conserving body heat. The required surgical window can be cut into the drape and a surgical spray adhesive is used in lieu of towel clips.

EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) can be applied to the wound postoperatively if no other local anaesthetic infiltration has been performed in the skin. This helps with the immediate post-operative skin pain as most rats show the most overt signs of pain 12 h post-operatively. *Since I have started using this technique, I have seen a faster return to eating on recovery.*

Recovery

If medetomidine has been given it can be reversed with 0.5–1.5 mg/kg IM or SC atipamezole, reversing the medetomidine will also reverse the analgesia provided by medetomidine but that is not reason enough for not reversing.

Maintain the rat on oxygen, and if intubated, extubate when the jaw tone increases. Shivering thermogenesis is a sign of recovery from anaesthesia. Continue to monitor while in an incubator or a warm room (ideally around 26–29°C (80–85°F)) until up and moving. If the rat is hypothermic, continue to take the rectal temperature every 15 minutes until it reaches within 1°C (33.8°F) of normal then reduce the temperature of the room or incubator to 22°C (72°F).

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Offer favourite foods once the rat is fully conscious. Many rats will readily take warmed human baby food from a spoon or a syringe. Alternatively, a high energy, easily digested diet such as Harrisons Recovery or the semi-elemental diet EmerAid Omnivore can be fed. In most cases with normal body temperature and good analgesia, eating will not be an issue (see Table 4.3).

Dental disease in rats

It is only the incisors that grow continuously in the rat. The incisors are yellow/orange due to iron deposition within the enamel. The molars are multi-rooted. The dental formula in rats is: 2 (I 1/1, C 0/0, P 0/0, M 3/3) = 16.

Rats can develop periodontal disease, dental caries, tooth decay and abscessation especially if fed on an inappropriate, high sugar diet. In fact, rats are used as human caries models.

Dental disease associated with the molars should ideally be managed in a similar manner to more familiar species, but the small size of the patient, the limited oral space, and the small size of the molars can make molar extraction challenging.

Incisors

In my experience, malocclusion in pet rats is uncommon. When it occurs, it is generally caused by trauma, tooth root infection, or neoplasia. Subtle signs of abnormal incisor wear may be seen if the rat is preferentially gnawing/chewing on one side due to e.g. TMJ pain, otitis externa, or a Zymbal gland tumour (Figure 4.13). I always recommend radiographing the skull in all cases of rat incisor malocclusion.

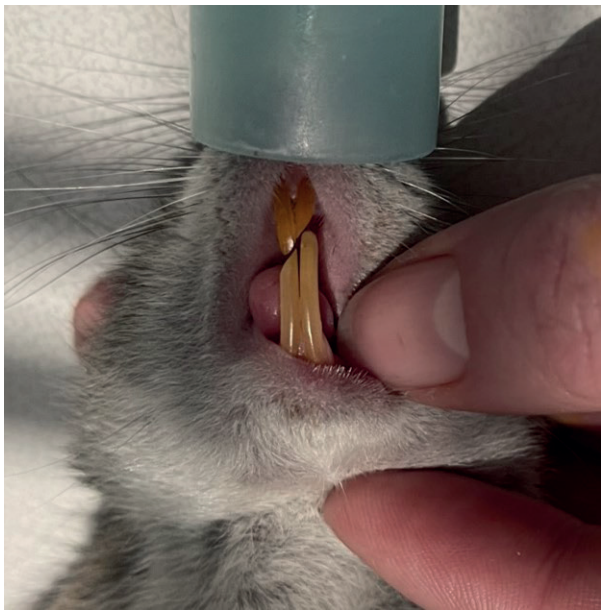


Figure 4.13 Subtle signs of incisor malocclusion can be easily missed. In cases of Zymbal gland tumour or otitis externa it is important to check the incisors as preferential gnawing may lead to malocclusion.

The literature records that: inappropriately soft diet in young growing rats; hypovitaminosis A, D, or E; calcium or magnesium deficiency; osteoarthritis of the temporomandibular joint; genetics will also predispose.

Unopposed, continuously growing incisors in the rat can erupt as fast as 1 mm daily! The malocclused incisors can cause significant soft tissue trauma to the lips, tongue, and hard palate, causing *pain, secondary infection and difficulty eating*.

The upper incisors will grow along the tight curve of the reserve crown and circle back into the oral cavity. Assess for the presence of an oronasal fistula in cases of malocclusion as the lower incisors will often cause soft tissue trauma caudal to the upper incisors and in severe cases the upper incisors can grow around into the hard palate.

Dental radiographs

General anaesthesia is required to obtain diagnostic radiographs. I mask induce with isoflurane (see Anaesthesia section).

The rostrocaudal view is best for visualising the temporomandibular joint surface, I use the lateral views to visualise the incisors and the molars. I prefer the ventrodorsal views for visualisation of the maxillary molars and any evidence of osteomyelitis associated with tooth root abscesses in the mandibular molars (Figure 4.14).

Treatment options

> Incisors should never be clipped with molar cutters or nail cutters. This practice is outdated and barbaric. Due to the conformation of the enamel, clipping causes the enamel to shatter leading to vertical fractures, pulpitis and potentially apical abscessation. There is no excuse for clipping incisors.

Uncomplicated incisor malocclusion (no secondary soft tissue trauma) can be managed with repeated trimming of the teeth (this can be done conscious in many patients) using a diamond cutting disc (use of guard advised) or a dental drill bit with the water turned off, as rat become distressed by the water being sprayed into their face. I find wrapping the rat burrito style and using the barrel of a 1 ml syringe behind the incisors to protect the soft tissues useful.

I will reduce the incisors to normal length and occlusion. The pulp cavity in normal incisors stops close to the gingival margin but in incisors which have an increased eruption rate the pulp cavity may also elongate. Taking the incisors abnormally short (e.g. in an attempt to reduce frequency of trimming) is not advised; it actually will result in faster regrowth (remember if unopposed can grow 1 mm/day) and runs the risk of penetrating the pulp cavity causing pain, pulpitis and potentially resulting in periapical abscessation.

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Figure 4.14 Radiograph of a rat who presented with left sided exophthalmos due to a tooth root abscess and osteomyelitis. *All cases of exophthalmos in rats should be radiographed to check for evidence of tooth root abscessation.*

If the rat becomes distressed or keeps trying to lick the drill bit, I advise a brief isoflurane general anaesthetic/deep sedation as that will carry less risk than caused by the surge of adrenaline and/or cortisol the fearful rat will experience. It also makes the procedure faster (Figures 4.15a & b).

Frequency

How frequently this will need to be performed will vary between individuals (the eruption rate is highly variable) which makes it impossible to have a hard and fast rule. I request owners check the teeth weekly and revisit as soon as they feel the teeth are too long and before any soft tissue trauma or impedance to eating occurs. In my experience this is anywhere between every 2 to 6 weeks.

Some rats will find frequent trimming stressful, even with anaesthesia, therefore if the incisors are non-functional, or are preventing normal chewing, extraction is recommended.

Incisor extraction

Owners may worry that their rat cannot eat without their incisors. Counsel the owners that cutting of foods into bite sized portions is all that is required to aid eating.

Incisors are also used during the grooming process and so owners may need to brush their rat especially if there is no companion rat. *If incisor extraction is performed, all the incisors should be extracted*. Incisors extraction in rats is more difficult than in rabbits.





Figure 4.15
a Incisor malocclusion.
b Using a dental drill to trim incisors.

Risks associated

Fracture of the incisor; if this happens continue to remove the rest of the incisors and reschedule removal of the remaining incisor once it has regrown 6–8 weeks later. Post-operative infection in the socket can occur. As the mandibular symphysis remains cartilaginous there is a risk of splitting the symphysis.

Equipment needed

One inch 23 gauge hypodermic needle, size 15 scalpel blade, sterile saline flush, dilute chlorhexidine swab, curved root forceps or curved artery forceps, 1.5 m (4/0) Polyglactin 910 with a swaged-on needle (Vicryl, Ethicon), needle holder.

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Technique

Extraction technique is similar to the rabbit but more challenging due to the length of the 'roots' and the cartilaginous mandibular symphysis. I will use the scalpel blade to incise the tissue of the gingival sulcus which opens the periodontal space.

Using the bevelled edge of the needle facing the tooth (use the hypodermic needle instead of the Crossley Luxator), introduce the needle into the periodontal space, held approximately at a 30 degree angle to the tooth and maintain constant downward pressure for 20–30 seconds, alternating which side of the tooth worked on. Keep repeating the procedure while placing the luxator deeper into the periodontal space. The bevelled edge of the needle cuts the ligament effectively and the needle can be bent to match the curve of the incisor.

Once the incisor is very loose it can be pushed in and out of the socket to destroy the germinal tissue at the apex of the tooth. When extracting the incisor, it is vital that the tooth is pulled along with the curve of the tooth to prevent fracture. Patience is key to preventing fracture of the root during extraction!

Flush the sockets with sterile saline and if no fracture has occurred, *I suture the gum across the sockets to prevent food or faeces entering the sockets (remember rats eat approximately 10% of their own and cohorts' faeces).*

Soft tissue trauma caused by incisor malocclusion

If there is soft tissue trauma, I recommend a general anaesthetic to allow full assessment and debridement of the wounds. Secondary bacterial infection is common in cases of soft tissue trauma.

In these cases, I will reshape the incisors using a diamond cutting disc and treat the secondary bacterial infections, allowing the wounds to heal up, the animal to gain condition and become more stable before electively extracting the incisors at a later date. This will also reduce the risk of post-operative extraction site infections.

Taking a swab for bacteriology and culture should be recommended to allow better targeting of antimicrobials. First line options while waiting on culture results include: clavulanate amoxicillin 50 mg/kg q12hrs or trimethoprim sulphonamide 30 mg/kg q12hrs providing the rat is not dehydrated.

Dentisept Oral Adhesive Paste (Forte Healthcare) is a vanilla flavoured, palatable oral paste containing chlorhexidine. When in contact with moisture it becomes incredibly sticky allowing it to coat mucosal, gingival, and periodontal surfaces, providing topical treatment for up to 24 h. *This can be useful in rats that enjoy the taste, potentially reducing the need for antimicrobials in some cases.*

We all know how debilitating oral pain can be. Splash blocking the wounds with local anaesthesia will result in faster return to normal eating. Ensuring adequate multimodal analgesia is dispensed for at least a week is imperative.

Facial abscesses associated with dental disease

Facial swellings and facial abscessation is often assumed to be due to molar root abscesses, however *the apices of the incisors are far reaching in the rat.* The mandibular incisor apices extend beyond the level of the third molar tooth, and the apices of the maxillary incisors extend for two-thirds of the diastema. *Tooth pulp abscessation of a damaged incisor (may not be visually damaged) can be a common cause of facial swellings in rats.*

Careful clinical examination (including under general anaesthesia) and radiographs are necessary to differentiate between: periodontal abscess, tooth pulp abscess, foreign body, or external wound as a causation of facial swelling. Dental abscesses are also a cause of exophthalmos in the rat (see Figure 4.14).

Treatment

Treatment can be challenging. *Failure to correctly diagnose the underlying problem will result in treatment failure.*

It is important that all affected teeth are extracted, the area should be curetted to remove all infected tissue (remembering that rodent pus is much thicker than dog or cat pus) and owners should be warned the wound will be left open to heal by secondary intention, which also allows for proper drainage. Extraction of the molars is challenging in rats due to the small size. Depending on the teeth involved, the masseter muscle may also be implicated.

Ophthalmology

The approach to the ocular exam is the same as with more familiar species. The notable differences in rats are: more laterally situated eyes (which gives a wide visual field), a low blink rate, poor vision (they evolved to have dim light vision being largely nocturnal or crepuscular which sacrificed visual acuity), rats with albino type eyes, that is, lacking pigment in their eyes have even poorer vision as they possess up to 30% fewer total photoreceptors than pigmented strains (Ilia and Jeffery, 2000), the bony orbit provides very little protection especially dorsally which may account for increased risk of ocular injury from bedding or GA mask, rats have a large Harderian gland lying at the base of the third eyelid and posterior globe occupying a large portion of the ventromedial and posterior orbit.

Sialodacryoadenitis virus in rats may result in swelling and inflammation of the Harderian gland (see Sialodacryoadenitis virus section for more information).

Conjunctivitis with or without blepharitis is reported as common in rats but in my experience is seen uncommonly in the clinic. Primary ocular infections can occur but may also be associated with respiratory disease, generalised

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skin disease, or secondary to inadequate husbandry (e.g. high ammonia levels or dusty bedding).

Chromodacryorrhea

Chromodacryorrhea is excess secretion of Harderian gland porphyrins which are red and is a *nonspecific sign of stress in rats*. As disease is a stressor it is not surprising that chromodacryorrhea will often accompany virtually any condition. Owners may report their rat is bleeding from its eyes or has a nose bleed if the excess secretion drains down the nasolacrimal duct.

Entropion

Entropion is uncommon but I have seen a couple of cases. Any rat with a Rex coat type who presents with a frequently recurrent conjunctivitis and overgrooming of the eyes should be examined closely. Entropion may develop secondary to trichiasis.

Trichiasis

Trichiasis is not uncommon especially in wire-haired breeds whose eyelashes may contact the ocular surface leading to secondary irritation and keratitis.

Treatment

As entropion or trichiasis are conformational issues, medical treatment will fail. Surgical correction should be considered using the same techniques as used in dogs (see Figure 4.11b).

Cataracts

Spontaneous cataracts are seen in rats and often do not seem to bother them (Figure 4.16). Exposure to high levels of light may induce cataract formation.

Glaucoma

Glaucoma is reportedly uncommon but may be underdiagnosed. Rebound tonometers have proven to be accurate and precise, even in conscious animals.

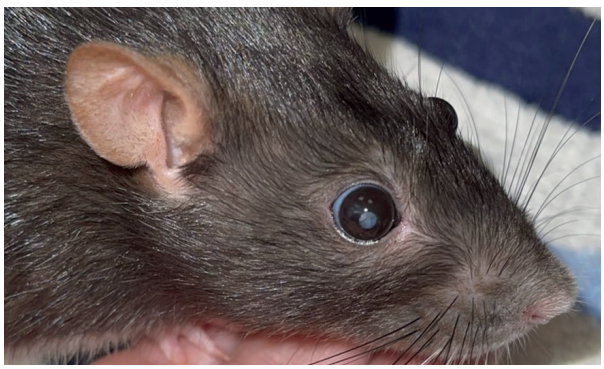


Figure 4.16 Spontaneous cataracts are seen in rats and often do not seem to bother them.

Intraocular pressure

Wang *et al.* (2005) established 18.34 mmHg as the normal intraocular pressure (IOP) in conscious male Wistar domestic rats using a rebound tonometer and noted that if the rat became even slightly agitated there was a significant increase in the IOP General anaesthesia resulted in a reduction in IOP. Rodrigues *et al.* (2021) demonstrated that even if the Icare Tonovet rebound tonometer is used within the manufacturer recommended 4–8 mm probe to cornea distance there is a significant increase in IOP measurements as the tonometer increases in distance from the cornea. *Any rat presenting with globe enlargement, pupil dilation, and/or signs of vision loss, glaucoma should be on the differential list.* Treatment of glaucoma is similar to more familiar species, both dorzolamide and/or timolol applied q8–12hrs have been shown to decrease the IOP. End stage glaucoma requires enucleation.

Exophthalmos

Exophthalmos is likely to result in an impaired blink, exposure keratitis, pain, and variable levels of ocular discharge (Figure 4.17). Exophthalmos may be associated with retrobulbar abscessation, neoplasia (rare, but if present they are likely to be malignant, Montiani-Ferreira *et al.*, 2022), or trauma. Radiography is advisable prior to enucleation to check for bone involvement (see Figure 4.14). If the bone is involved the prognosis is worse due to exenteration being required, and the treatment of osteomyelitis, rather than a relatively simple enucleation.



Figure 4.17 Exophthalmos is likely to result in an impaired blink, exposure keratitis, pain, and variable levels of ocular discharge. This rat had a retrobulbar abscess associated with a tooth root abscess, see Figure 4.14.

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Respiratory disease in Rats

Presentation for respiratory disease (RD) is extremely common in the pet rat. Owners should be advised that RD is *multifactorial and as poor husbandry is a predisposing factor*, full assessment of the enclosure and targeted questions on the stability of the mischief (name for a group of rats) is advisable.

> Overcrowding, poor ventilation, scented or dusty bedding, high ammonia levels, high humidity, sudden changes in temperature or chronic disease can all lead to stress-induced immunosuppression. Immunosuppression can result in even expected respiratory microbial flora to become opportunistic pathogens.

Ideally, when the appointment is made, *request several photographs*, taken from different angles to assess the enclosure for bottle necks or limited resources (see section on husbandry for current recommendations).

Dietary deficiencies such as low vitamin A or vitamin E have been shown to make the condition worse, so clients should be advised to store food appropriately and use within recommended manufacturers use by and after opening dates.

Inhalation of tobacco smoke has also been shown to affect RD in rats (Lindsey *et al.*, 1991), so *owners should be advised to never smoke in the same room as their rats* and I frequently advise clients to change clothes after smoking before handling their pets as the smoke leaches out of their clothing.

> It is important to advise owners that in cases of poor husbandry, failure to correct their home environment will lead to a failure of treatment. Owners should also be advised flare ups are common and that as virtually all cases involve Mycoplasma pulmonis infection, the rat will remain a carrier for life.

It is equally important to remember that although infectious disease is the most common cause of RD in rats, cardiac disease and neoplasia should be kept on the differential list as causes of dyspnoea in rats.

Species-specific anatomy of respiratory tract

Rats are obligate nasal breathers; their right lung is divided into four lobes, however their left lung is smaller and is not divided into lobes. Rats have a sensitive sense of smell and can find strong scents, e.g. scented candles, etc., overwhelming.

> The normal respiratory rate for rats is 70–100 breaths per minute. The pattern of breathing should also be assessed, e.g. are they abdominal breathing?

Rats normally have an extremely flexible thorax and loss of thoracic compliance indicates further investigation, i.e. radiography is required as there may be cardiac disease, neoplasia, abscessation or a pleural effusion.

The Harderian gland is part of their lacrimal system. This gland produces part of the tear film which is red in colour due to the porphyrin content and porphyrin will fluoresce pink under ultraviolet light. *The gland also produces an oil which is used during grooming – this is why we will sometimes see tinges of pink on the head, neck, and shoulders of white rats!*

Any form of stress can cause an increase in production from the Harderian gland, and these excess red tears are called *chromodacryorrhea. It is not uncommon for these red tears to be mistaken for blood, especially if they have travelled down the nasolacrimal duct and the owner mistakes them for a nose bleed!* Owners should be reassured that the presence of red tears is a *nonspecific sign of stress* in rats and although is commonly present in cases of disease, it can also be that they are stressed for a more innocent reason including they did not like the way they were caught!

Pathogens

Pathogens known to cause RD (not an exhaustive list):

- *Mycoplasma pulmonis*
- Sendai virus
- sialodacryoadenitis virus (SDAV)
- cilia associated respiratory (CAR) bacillus
- Streptococcus pneumoniae
- *Corynebacterium kutscheri*
- *Pseudomonas aeruginosa*
- *Pasteurella pneumotropica*
- *Bordetella bronchiseptica*
- *Pneumocystis* spp.

Clinical signs

> Clinical signs can be highly variable and significant pathology can be present without overt clinical signs.

Young rats will often have no clinical signs. Mild cases of sneezing with no other clinical signs may be allergic rhinitis. Questioning the owner about what bedding is used, e.g. if the rat is kept on shavings (change to paper-based bedding) or asking if the owner uses aerosols or burns incense or scented candles in the room the rat lives in (ask the owner to stop this), is advisable. Sometimes there is nothing in the history which is obviously causing a case of allergic rhinitis.

As the rat ages, nonspecific clinical signs such as weight loss, hunched appearance, poor-quality coat, inactivity, social isolation, rubbing of eyes, and chromodacryorrhea may be apparent. These signs may also be seen with

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comorbidities such as osteoarthritic pain and chronic pain is immunosuppressive.

Abdominal breathing, dyspnoea, wheezing, sneezing, nasal discharge, may all be present in advanced cases. Torticollis may be present if an ascending infection causes otitis media/interna. Mycoplasma can also cause endometritis and pyometra may be present. Suppurative rhinitis, laryngitis and tracheitis are often present but can be difficult to observe in rats.

I see cyanosis of the nail beds more frequently in cardiac disease than in RD, however we should remember both conditions can co-exist.

Chronic RD is common and the associated chronic inflammation can cause permanent damage to the respiratory tract (fibrosis and consolidation). Suppurative bronchopneumonia, atelectasis, bronchiectasis and or abscesses are frequent findings.

Investigation of RD

The differential diagnosis list for RD (includes diseases affecting the thoracic cavity as they may present as respiratory disease) comprises pneumonia, pleuropneumonia, abscessation, pericarditis, pleural effusion, cardiomyopathy, lymphoma, pulmonary neoplasia, or pulmonary metastatic disease (uncommon). Pleural effusions were seen in both infectious and neoplastic conditions. *Age of the rat should not be used to differentiate between infectious or neoplastic disease.*

Stress is known to have a huge impact on expression of disease, so any comorbidities (e.g. osteoarthritis causing chronic pain) should be diagnosed and treated as part of the overall case management.

A full husbandry review is required, including taking the age and mobility of the rat into account (see Pain section).

Mycoplasma pulmonis is considered ubiquitous in rats and virtually all infectious RD in rats is the result of *Mycoplasma*, usually in combination with one or more of the above pathogens (frequently CAR bacillus and/or Sendai virus).

> As *Mycoplasma* is ubiquitous (RD in rats is often referred to as murine respiratory mycoplasmosis), it is impractical or indeed unnecessary for vets to attempt to identify the causative agent. I advise, in rats, to assume it is *Mycoplasma* as part of a mixed infection and be aware other pathogens can be significant contributors.

Nasal washes and tracheal washes can be performed if the clinician wants to get bacteriology and cytology. Warmed sterile saline, 2 ml/kg, is used but this is not something I perform in this species. Owing to *Mycoplasma* being difficult to culture, serology is considered more reliable than culture.

In my opinion, trying to establish the chronicity and severity of the disease process is a better use of the owners' finances as it will enable us to provide a more realistic prognosis. Significant disease can be present with minimal clinical signs.

Auscultation of the lungs should be performed as per more familiar species although using an infant diaphragm will make it much easier. The list of adventitious noises that may be heard are exactly the same as in more familiar species – it is just lungs after all. Of equal importance is lack of sound, i.e. no air movement where air movement would be expected. This would indicate areas of consolidation, e.g. abscessation, tumour or pleural effusion.

A reduction in thoracic compliance immediately alerts us to the presence of a space occupying lesion which could be tumour, abscess, or an enlarged heart.

Radiography

Radiography of the skull may show turbinate atrophy. Thoracic radiography using standard positioning (DV, right lateral, left lateral) is recommended in virtually all cases.

> Always ensure the dorsoventral radiograph is taken first to avoid hypostatic congestion of the most dependant lung complicating interpretation.

Radiographic findings may include abnormalities of bronchoalveolar pattern, multifocal areas of pulmonary infiltrates, pulmonary densities (abscesses or neoplasia), widened mediastinum (often related to neoplasia – see thoracic masses), and increased size of cardiac silhouette (see section on cardiac disease). *Examine the full radiograph including the skeleton for any signs of degenerative joint disease or narrowing of intervertebral disc spaces.*

Treatment for RD

Husbandry improvements may be one of the most important tools we can utilise for controlling RD in rats. Reducing stress and thereby improving the immunocompetency of the rat will help reduce severity of the disease and minimise flare ups.

Cases with a suspected allergic component I will trial an antihistamine as well as making any obvious husbandry adjustments. Chlorpheniramine maleate (0.6 mg/kg q12–24hrs) is usually my first preferred option for an antihistamine. Diphenhydramine (1–2 mg/kg q12hrs) is my second choice, if the chlorpheniramine maleate does not make an improvement.

Antibiotic therapy will be necessary in many cases. In mild cases with minimal lung pathology, I will reach for either clavulanated amoxicillin or trimethoprim sulphonamide (30 mg/kg q12hrs) for 10 days.

The dose recommended for clavulanated amoxicillin varies widely in the literature from 100 mg/kg q12hrs (Keeble and Meredith, 2009; Hedley, 2020) to 20 mg/kg q12hrs (Carpenter and Marion, 2022; Sinclair, 2021).

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Higher doses may be associated with softer droppings. Personally, I tend to use 50 mg/kg q12hrs.

For non-responsive or severe cases I will use a combination of enrofloxacin (10–30 mg/kg q12hrs) and doxycycline (5 mg/kg q12hrs) as the combination is often more successful against *Mycoplasma* than when the drugs are used individually. Doxycycline can be ordered in a palatable form from compounding companies such as Summit or Bova. Prolonged courses of antibiotics or eight weeks or more may be required in severe cases. *Oral doxycycline should not be mixed with dairy products as the calcium can bind the drug and prevent absorption.*

Rats that owners are finding difficult to medicate, may benefit from once weekly long-acting doxycycline subcutaneous injections (50 mg/kg weekly) for three treatments. Some authors have found using azithromycin (30 mg/kg orally q24hrs) useful. See Table 4.6 For commonly used antibiotics in rats.

> Ultimately, antibiotic therapy is rarely going to cause a complete cure of RD in rats and owners should be counselled that we are looking for improvement of clinical signs and quality of life but that future stressful events will likely result in a flare up of disease.

Owing to the inflammation caused by the disease process and that older rats are likely to have concurrent degenerative joint disease, I tend to use meloxicam (0.6–2 mg/kg q12hrs, I start at 1 mg/kg) as soon as I see the rat. *Reducing pain is vitally important to welfare and general wellbeing.*

Bromhexine (0.5 mg/kg q12hrs) can be used to help break up mucous and ensure easier clearance from the respiratory tract. Accurate dosing can be difficult and as multiple papers demonstrate that even at extremely high doses (300 mg/kg) there were no adverse effects noted, I advise a pinch of the bromhexine (10 mg/g strength) onto a favoured food item twice daily for one week. *It is important we manage owners' expectations and remind them that once the mucous starts to break up, the rat will sneeze or cough more to clear the secretions.*

Sildenafil citrate (5 mg/kg q24hrs, one trade name is Viagra, Pfiser) can act as a pulmonary protectant. It helps prevent further fibrosis of the lungs and helps prevent thickening of the airways in the lung. In addition, it also has a protective effect on pulmonary vasculature and pulmonary parenchyma. It can be used in moderate to severe cases alongside the above medications.

Severe cases of RD may benefit from a short course of steroids, prednisolone (0.5 mg/kg q12–24hrs). Just as in more familiar species, steroids and non-steroidal anti-inflammatories should not be used concurrently and the dose should be tapered. *Personally, I rarely use steroids in rats, mainly due to potential immunosuppressive effects.*

Nebulisation therapy

Mesh nebulisers are much quieter, produce less vibration, and so are less stressful for the rat. Thankfully they can now be bought inexpensively online. Desprez *et al.* (2019) demonstrated that a droplet size produced of 3 μm reached all areas of the rat lung. See Table 4.7 for nebulisation drug doses.

Mucolytics and antibiotics can be delivered using nebulisation. F10 disinfectant (bactericidal, virucidal, fungicidal) can safely be nebulised using a 1:250 dilution of the concentrated product, for 15 minutes twice daily. Mild cases of RD may be successfully managed with the use of F10 alone.

Table 4.6 Antibiotics commonly used in rats.

Drug	Dose
Azithromycin	30 mg/kg PO q24hrs
Ciprofloxacin	10 mg/kg PO q12hrs
Chloramphenicol	30–50 mg/kg SC, IM, IV, PO q8–24hrs
Clavulanate amoxycillin	50mg/kg PO q12hrs
Doxycycline (often used in conjunction with enrofloxacin in mycoplasma respiratory disease)	5 mg/kg PO (no dairy) q12hrs, long-acting formulation 50 mg/kg q7days SC
Enrofloxacin	10–20 mg/kg PO SC q12hrs (IM painful and may result in sterile abscess/muscle necrosis)
Marbofloxacin	5 mg/kg PO q12–24hrs
Metronidazole	15–20mg/kg PO q12–24hrs
Oxytetracycline	50 mg/kg PO q12hrs
Silver sulfadiazine cream	Topical q12–24hrs
Trimethoprim sulfamethoxazole	30 mg/kg PO q12hrs

Table 4.7 Nebulisation doses.

Drug	Dose (most nebulisers will hold 10 ml)
Saline	10 ml good for hydration of respiratory tract
Acetylcysteine (mucolytic)	10–20 mg per 1 ml saline (higher doses may be associated with bronchospasm)
Bromhexine (mucolytic)	0.15 mg per 1 ml saline
F10	1:250 or ready to use (already prediluted)
Aminophylline	3 mg per 1 ml saline
Amikacin	5 mg per 1 ml saline
Doxycycline	13 mg per 1 ml saline
Enrofloxacin	10 mg per 1 ml saline
Gentamicin	5 mg per 1 ml saline

Videos can be found on YouTube to direct owners on how to set up their own nebulisation cage at home.

Thoracic masses

Although respiratory infections are one of the most common conditions seen, thoracic neoplasia is not uncommon. Both primary pulmonary neoplasms and secondary neoplasms are recorded in the rat.

These cases will frequently present with respiratory signs and may have a reduction in thoracic compliance.

An enlarged or widened mediastinum increases the index of suspicion for neoplasia. Rats with cranial mediastinal masses are most often affected by round cell neoplasia such as lymphoma.

Ultrasound is recommended in cases of suspected cranial mediastinal masses, to rule out abscessation and ultrasound guided fine needle aspirate (FNA) of the mass may provide a definitive diagnosis.

Lymphoma in rats

Although the incidence of lymphoblastic lymphoma increases with age in rats, infectious causes also increase with age therefore age cannot be used to differentiate between infectious versus neoplastic, and both conditions have been reported in rats under 9 months of age.

Owing to the fact the most common presenting sign of lymphoma in rats is respiratory compromise, it is highly likely that lymphoma is underdiagnosed in rats. Purely due to the fact many will be euthanised on quality-of-life concerns without a definitive diagnosis, being attributed to intractable RD. Weight loss, hepatomegaly, splenomegaly, intestinal involvement, and a massive lymphocytosis have all been reported in rats diagnosed with lymphoma although it should be noted that these signs have variable degrees of occurrence compared to respiratory compromise which was seen in every case.

Any rats presenting with RD, especially with increased thoracic compliance, or failure to respond quickly to antimicrobial therapy should be radiographed and investigated for underlying neoplasia or cardiac disease.

Unfortunately, diagnosis of thoracic/mediastinal lymphoma is likely to have a grave prognosis. Faye-Fierman *et al*. (2022) while performing a retrospective study, found that of the two rats treated for lymphoma, one lived for only 11 days post-diagnosis and treatment, and the other one lived for 24 days. Both rats were euthanised due to poor-quality of life. Mediastinal lymphoma carries a poorer prognosis in our canine patients.

Although we cannot definitively draw the same conclusion in our rat patients, especially with such a small sample size, we should be cognizant of the risk of inducing severe bronchopneumonia when using chemotherapeutic drugs due to the immunosuppressive effects. The ubiquitous nature of infectious RD in rats means that in most cases antibiotics should be started prior to any immunosuppressive drugs being given. As always, *our aim should be to improve quality of life not quantity of life*.

Cardiac disease

Cardiac disease is not uncommon and most likely underdiagnosed, possibly because we do not always look for it. As RD is so common in rats, if we do not include cardiac disease on our list of differential diagnosis when presented with a rat exhibiting signs RD, it is easy to see how we could misdiagnose.

The common clinical signs of cardiac disease are nonspecific, including dyspnoea, lethargy, anorexia, and weight loss, which adds to the difficulty of diagnosis, although in my experience, *cyanosis of the nail beds is more common in cardiac disease* than RD (Figure 4.18).

Dilated cardiomyopathy (DCM) with or without pleural effusion is the most commonly diagnosed cardiac disease in rats. There is likely a genetic component for development of DCM in rats as certain lines will have a higher incidence.

There is *often a reduction of thoracic compliance* due to the cardiac enlargement and standard thoracic dorsoventral and lateral radiographs should be taken under sedation/general anaesthesia. Radiographic findings may include raised trachea, cardiomegaly, pleural effusions, and pulmonary oedema.

Assessing cardiac size radiographically

The normal position of the heart is lying between the third to fifth ribs and should be 2–2.5 intercostal spaces.

Vertebral heart score

Dias *et al*. (2021) established a vertebral heart score (VHS) for rats, which can be used as a tool to aid in the diagnosis



Figure 4.18 Cyanosis of nail bed in a rat with cardiac disease.

and monitoring of cardiomegaly. The method is very similar to what we perform in canine VHS using a right lateral radiograph. A line is drawn from the carina to the cardiac apex to form the long axis (LA), the short axis (SA) line is drawn perpendicular to the LA along the widest point of the heart. Both the LA and the SA are measured independently using the vertebrae, starting at the fourth thoracic vertebrae (T4), to nearest 0.1 of a vertebra. Both measurements are added together to form the VHS.

The normal VHS for rats was established to be: 7.7 (7.0–8.5), with the male rats having larger hearts than female rats. It should be remembered that a normal VHS does not necessarily rule out heart disease as, for example, concentric hypertrophic cardiomyopathy or specific cardiac chamber enlargement will not always result in an increased VHS. Ultrasonography is recommended if there is a high index of suspicion of cardiac disease.

It is advisable to ultrasound the heart to check for the presence of a pericardial effusion.

Treatment of cardiac disease

If there is a pericardial effusion present, then drainage via ultrasound guided needle under a general anaesthesia is recommended.

Furosemide 2–5mg/kg PO or SC q12hrs can be given for pulmonary oedema. A human liquid preparation of furosemide called Frusol (Rosemount Pharmaceuticals Ltd) is available at variable strengths which some patients find palatable.

Pimobendan 0.6 mg/kg q12hrs. I dilute the contents of 5mg capsule in a known quantity of water, e.g. 2 ml to make a 2.5 mg/ml solution. I advise we do not know how long the medication is stable for and advise owners how to make the solution and to make it fresh weekly. Enalapril can also be used 0.5–1 mg/kg q24hrs, alternatively benazepril 0.05–1 mg/kg q24hrs, or digoxin 0.005–0.01 mg/kg q12–24hrs.

Given the high prevalence of RD, consideration of antibiosis should be given especially in high stressed individuals.

My personal preference is to start on furosemide and pimobendan, then reassess in a week. Ideally taper furosemide to the lowest controlling dose and add in enalapril if needed. Prognosis is variable depending on severity of heart disease at first presentation and concurrent comorbidities, but I have seen rats get up to almost a year on medication.

Ectoparasites

- *Notoedres muris*: also known as the ear mange mite due to the crusty, proliferative dermatitis the burrowing mite causes bilaterally on the pinna, nose and tail. *Owners should be advised that this mite can be zoonotic*. Diagnosis is via skin scraping and skin biopsy.
- *Demodex*: not commonly seen in my experience but will present as ventral alopecia.
- *Radfordia ensifera*: also known as the rat fur mite. Rather than any damage the mite causes, it is an allergy to the mite that causes the pruritus. Self-trauma leads to excoriation and crusting lesions, often over the head and shoulder regions. Diagnosis is via skin scraping.
- *Ornithonyssus bacoti*: also known as the tropical rat mite. Ironically this mite is rarely seen in pet rats, and I have only seen it in large breeding colonies of rats. This mite feeds on, but does not live on the rat and will be seen as black/dark brown dots in the substrate. Owners should be counselled that it is zoonotic (although usually will not preferentially bite humans) and that due to most of the life cycle occurring off the host, complete eradication can be difficult. The mite may carry diseases (for example, murine typhus, Q fever, both of which are rare in the UK) transferable through the bite both to rats and humans (Figure 4.19).
- *Polyplax spinulosa*: also known as the spiny rat louse, is a sucking louse. Lice infestations are uncommon in pet rats. They may be seen in debilitated animals. Diagnosis is via tape test.

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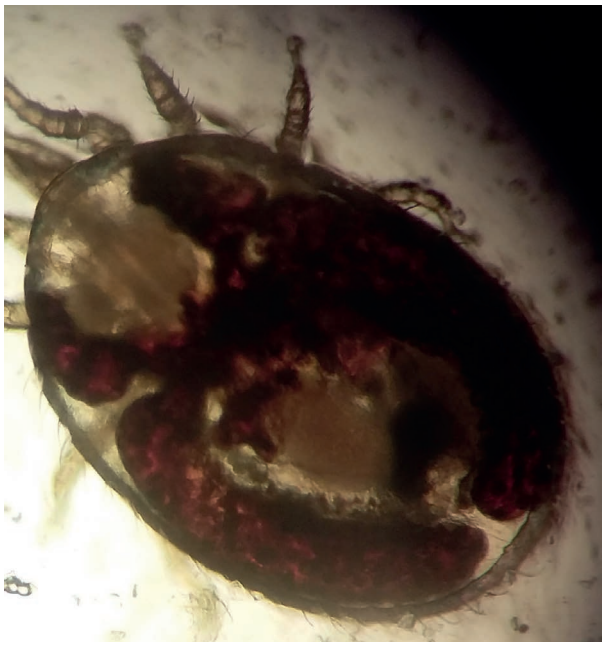


Figure 4.19 Ornithonyssus bacoti a.k.a. tropical rat mite.

Treatment

Mites and lice are easily treated in pet rats with selamectin (15–24 mg/kg), single application is likely to be curative in most cases, although another application can be applied 30 days later.

Endoparasites

Syphacia obvelata, also known as the common mouse pinworm, can occasionally cause perianal irritation and tail based mutilation. Diagnosis is via tape test strips applied perianally and examined for the banana shaped eggs.

Zymbal gland neoplasia

The Zymbal glands are small, modified sebaceous glands located around the proximal aspect of the external ear canal in rodents. The gland has three sections (anterior, posterior, and submucosal) which drain via a duct within the ear canal, near the tympanic membrane.

Neoplasia associated with the Zymbal gland include adenocarcinomas, adenomas, squamous cell carcinomas, and squamous papillomas.

Clinical signs

Early cases may present with an ulcer ventral to the opening of the ear canal. More commonly will present with a swelling ventral, cranioventral or caudoventral to the opening of the ear canal (Figure 4.20). These masses will commonly grow rapidly, frequently ulcerate, can be locally invasive and may metastasise. Because of their initial appearance, they can be mistaken for bite wounds/abscesses, or may be mistaken for otitis externa. *Owing to the proximity to the temporomandibular joint, pain associated with the swelling may cause secondary incisor malocclusion.*



Figure 4.20 Swelling due to a Zymbal gland tumour.

Treatment

Surgical excision is only successful in cases of early diagnosis. Early diagnosis via biopsy, especially in cases of otitis externa with swellings or ulceration at the base of the ear, may allow diagnosis to be achieved in time to provide a surgical cure.

Mammary tumours

Mammary tumours are the most common spontaneous tumour seen in the pet rat. Female rats have six pairs of mammary glands, and mammary tissue extends from the neck to the inguinal region. This means mammary tumours can occur anywhere, over a relatively large area. Male rats also have a smaller amount of mammary tissue and mammary tumours are seen in male rats albeit less commonly.

It is uncommon for mammary tumours to occur in rats less than one year of age. *Ninety per cent of mammary tumours in rats are benign (fibroadenomas)*, the remainder are carcinomas.

Mammary tumours can grow to approximately 10 cm in diameter, and it is the sheer size they can obtain that causes the issue. Some books report that they are slow growing, but in my experience, they can be fast growing also.

Owners should be counselled that the skin may ulcerate (and be prone to secondary bacterial infection) due to stretching, or trauma from dragging on the ground. *Although the tumour itself does not appear to be painful, the sheer weight tugging on the soft tissues and of course the altered centre of gravity along with the altered gait will likely cause secondary pain*. Depending on location, the tumour may also interfere with the prehension of food.

Treatment

Surgical excision of the tumour is the treatment of choice, ideally before the tumour has gotten too large. Lumpectomy rather than attempting a mammary strip is recommended due to the extensive mammary tissue present in the rat. Rats

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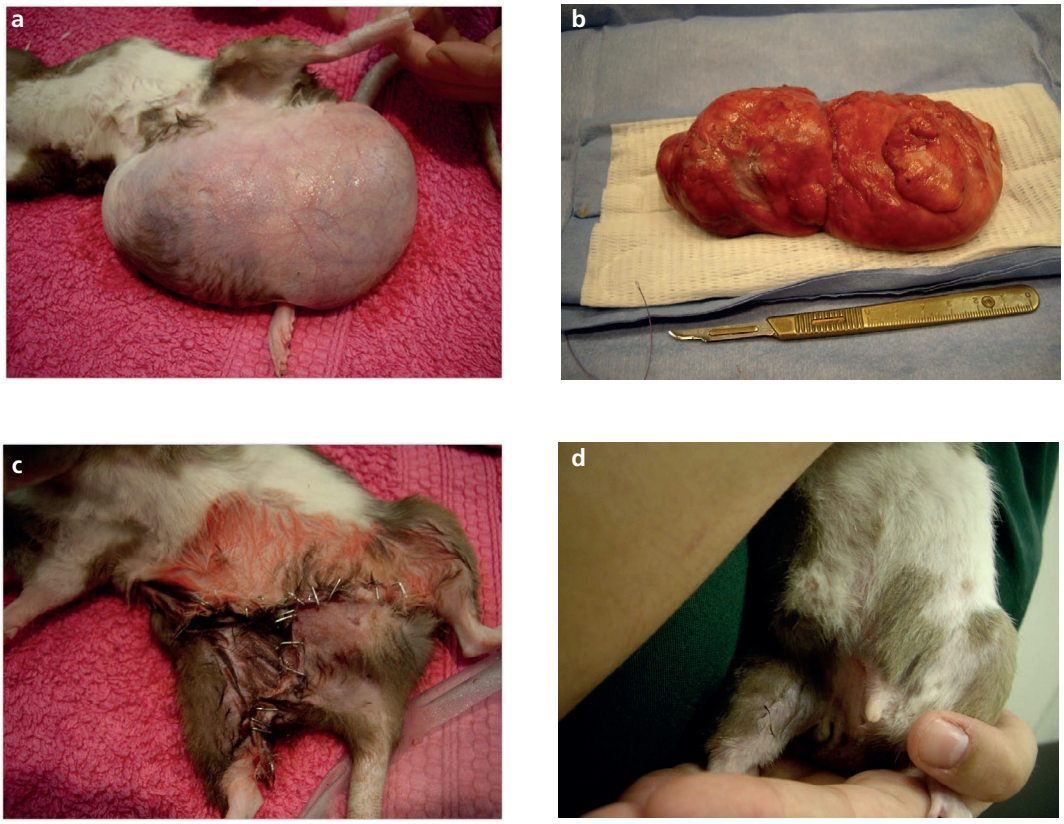


Figure 4.21
a A large mammary mass, the owner had been told the mass was not resectable and to euthanise when it affected the rats quality of life. The owner sought our advice the day he decided to euthanise and asked if we would remove the mass.
b The resected mass.
c Owing to the large size of the wound, closure was performed using surgical staples.
d The same rat fully healed.

generally heal very well post-surgery, even in cases where I have removed the rat from the lump! (see Figure 4.21a–d).

Owners should be counselled that it is common for a second mammary tumour (unrelated to the first one) to develop within a few months. I advise owners to set a limit on how many times we operate for mammary tumours on the same rat, which will be different for each vet. Personally, I struggle on the ethics of operating more than twice on the same rat for mammary tumours. Approximately 75% of rats with mammary tumours have been shown to concurrently have pituitary adenomas.

The latest research suggests that deslorelin implants after mammary tumour removal will not prevent further mammary tumours developing. Anecdotally, ovariohysterectomy after mammary tumour removal may prevent further mammary tumours developing but more research is needed.

Surgical tips

Check the skin for the line of tension as per more familiar species, to allow appropriate orientation of the skin incision. Once I have sharply incised the skin elliptically (I often include the nipple in the initial incision), and started to bluntly dissect the tumour from the skin with blunt/blunt curved scissors, I will frequently change to using my fingers to blunt dissect around the tumour, essentially 'shelling' out the tumour and allowing identification of vasculature needing ligation. Sterile cotton buds (regular ones can be put through the autoclave which helps reduce costs), are useful for tissue manipulation and swabbing small areas.

Depending on the size of the mass, there may be a lot of excess skin which can be resected to reduce dead space. *Drains are not advisable as rats will chew them out*.

Small skin deficits (3 cm or smaller), I close with 1.5m (4/0) Polyglactin 10 (Vicryl) on a needle using an intradermal suture pattern. EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) application to the skin after closure will provide further analgesia.

Large skin deficits I close using surgical staples (Figure 4.21c). They are fast to place and, in most rats, easy to remove conscious, ten days later. I have only ever anaesthetised one rat to remove staples. EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) application to the

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skin after closure will provide further analgesia. The best size of staples I have found is 35W.

Owners should be advised that sometimes rats like to play with the staples and can sit and spin them through the skin. This can occasionally make removal more challenging as the staples may need to be manipulated back to the correct position to facilitate removal.

Prevention

Restriction of calories to 70% of ad lib feeding has been shown to greatly reduce the incidence of mammary tumours, which is a great incentive for owners to prevent obesity. Several studies have also shown that *early ovariectomy or ovariohysterectomy (between 5–7 months of age), dramatically reduces the incidence of mammary tumours, from 73.8% to 5.3%* in one study (Planas-Silva *et al.*, 2008).

Sialodacryoadenitis virus

Also called the rat coronavirus or rat mumps, it is infrequently seen in pet rats, in my experience.

In laboratory colonies the virus has a high morbidity but low mortality. This may be in part due to limited exposure to secondary pathogens such as *Mycoplasma pulmonis*.

In pet rats, subclinical infection with *Mycoplasma*, among others, is considered ubiquitous, which can become secondary pathogens when the rat has been immunocompromised by sialodacryoadenitis virus (SDAV). SDAV primarily attacks the Harderian gland, lacrimal glands, salivary glands, and respiratory epithelium. Some strains of SDAV will cause interstitial pneumonia leading to permanent respiratory damage.

Clinical signs may initially be of photophobia, conjunctivitis and chromodacryorrhea (red tears). Bulging of the eyes can occur due to swelling of the lacrimal glands, leading to secondary exposure keratitis. Swelling of the salivary glands may lead to mass development around the head and neck (hence the name rat mumps). Without secondary pathogen involvement, many rats will remain bright and appetent.

Diagnosis is often presumptive on clinical signs but a polymerase chain reaction (PCR) test is available.

Treatment is often symptomatic with meloxicam and antibiotics may be indicated in animals with lower respiratory tract disease. Some rats will develop permanent lung damage due to interstitial pneumonia.

Renal disease

Chronic progressive nephrosis is very common in rats over 1 year of age and males are overrepresented.

> It is important to remember male rats have renal tubular production of alpha globulins therefore, proteinuria (<0.3 g/l) is a normal finding in male rats.

Clinical signs

Clinical signs include weight loss, significant proteinuria (which will progress with time), polyuria (normal urine output is 5.5 ml/kg body weight), polydipsia (normal water consumption 8–12 ml/100 g body weight), elevated plasma creatinine, protein casts may be found in the urine and isosthenuria. Differential diagnosis should include chronic bacterial pyelonephritis, hydronephrosis, ischaemic injury and toxic nephrosis.

Diagnosis

Rats will often spontaneously void urine during handling especially if they feel nervous. While not ideal, some urine can be gathered in this way as a 'free catch' sample to allow basic urinalysis to be performed (dipstick, specific gravity). See Table 4.8 for urinalysis normals.

A full biochemistry and haematology should be advised. The rat lateral saphenous or lateral tail vein are beautiful veins for sample collection (see Venipuncture section). The biochemistry and haematology can run in-house using the standard dog and cat profiles (rats are mammals after all) or sent to an external laboratory.

Radiography and ultrasonography will aid in the differential diagnosis. Cystic calculi are uncommon in rats but can result in hydronephrosis. Calcification of the renal pelvis can occur also which will further exacerbate renal disease. Urine may be collected by cystocentesis or sterile urinary catheterisation under general anaesthesia to be sent for urinary culture and sensitivity.

Treatment of renal disease

Chronic progressive nephrosis

Restricting calorific intake to approximately 70% of adlib feeding and restricting protein intake to 4–7% will both reduce the severity and the progression of the condition. Educating owners on dietary restriction of rats can help prevent the occurrence of renal disease in some rats (see Diet section).

The ACE inhibitor enalapril (0.5–1 mg/kg q24hrs) may help.

Providing plenty of absorbent bedding will help absorb the excess urine associated with polyuria. Constant access to fresh drinking water and regular cleaning of the cage will improve quality of life. A multivitamin may be considered

Table 4.8 Urinalysis normal.

Colour	Variable depending on diet
Protein	+ (<0.3 g/l)
pH	7–7.4
Specific gravity	1.022–1.050
Sediment examination	Calcium carbonate and triple-phosphate crystals

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as water soluble vitamins may become deficient due to the diuresis.

The rat should be monitored for comorbidities as concurrent osteoarthritis or spinal disease will reduce the grooming ability of the rat and may contribute to urine scald. *Renal disease will not prevent me using a NSAID such as meloxicam*. Multimodal analgesia reduces the dose given of each agent thereby reducing the potential side effects. Also, *I have a very honest and frank discussion with owners that quality of life not quantity of life should be our ultimate aim*.

Pseudomonas, *E. coli*, *Proteus mirabilis*, and *Klebsiella* are some of the bacteria commonly isolated in cases of bacterial pyelonephritis. Ideally antibiotic choice should be guided by culture and an *extended course of 6 weeks will be required along with supportive care*.

Urinary calculi

In my experience calculi are uncommon in pet rats. Water restriction has been suggested as a possible predisposing factor, although in the cases I have dealt with that was not the case. Urinary calculi may be found in the kidney, ureter, bladder, or urethra. Calculi in the bladder may be palpable. Radiographs will help with diagnosis (Figure 4.22).

The position of the calculi dictates the severity and acuteness of clinical signs much as with more familiar species. For example, blockage of a ureter will result in hydronephrosis, bladder calculi will cause signs of cystitis and blockage of the ureter will result in acute presentation of a blocked bladder. *The calculi cannot be dissolved using diet and surgery is indicated as per more familiar species*. Fluid therapy, analgesia, warmth, and antibiosis (guided by culture) are all required, just as in more familiar species (Figures 4.23a–d).

Euthanasia

End of life conversations can be a difficult area to broach with some clients. Occasionally owners will wait for us to broach the topic as they are 'worried' if they do so we will think they are not dedicated enough. Reassuring clients that sometimes we need to love our pets enough to let them go when the time is right can be invaluable.

Many owners will want to stay with their rat during this procedure and the process should be fully explained including possible events such as squeaking with injection of premedication, sudden jumping, the need to change veins or gasping. I administer a pre-med (e.g. medetomidine/butorphanol/ACP injected SC over the lateral flank), once the rat has fully relaxed, I will use the lateral saphenous vein by choice, to inject the pentobarbitone, to allow the owners to stroke their pets head. If the owners do not want to stay I will mask induce with isoflurane and inject pentobarbitone intracardially.



Figure 4.22 Radiograph showing the bladder full of calculi. This particular rat was radiographed conscious by placing her in a plastic tub.

I advise allowing the bonded cage mate to see its companion after euthanasia. I advise clients that I believe it allows the cage mate to accept that its companion will not return. Over the years, many owners have reported to me they felt the cage mate was looking for its companion constantly after losing them and certainly anecdotally this is reduced when they get the opportunity to 'say goodbye'. Whether this is purely anthropomorphic or not is up for debate, however it demonstrates to your owners that you care and you listen to their concerns.

Many owners will opt for private cremation with return of their pet's ashes and all options that your practice offers for dogs and cats should also be made available to your rat clients. I offer a lock of fur, footprints (coloured ink pad from craft shop and a card). I send sympathy cards if I know the patient and owner well.

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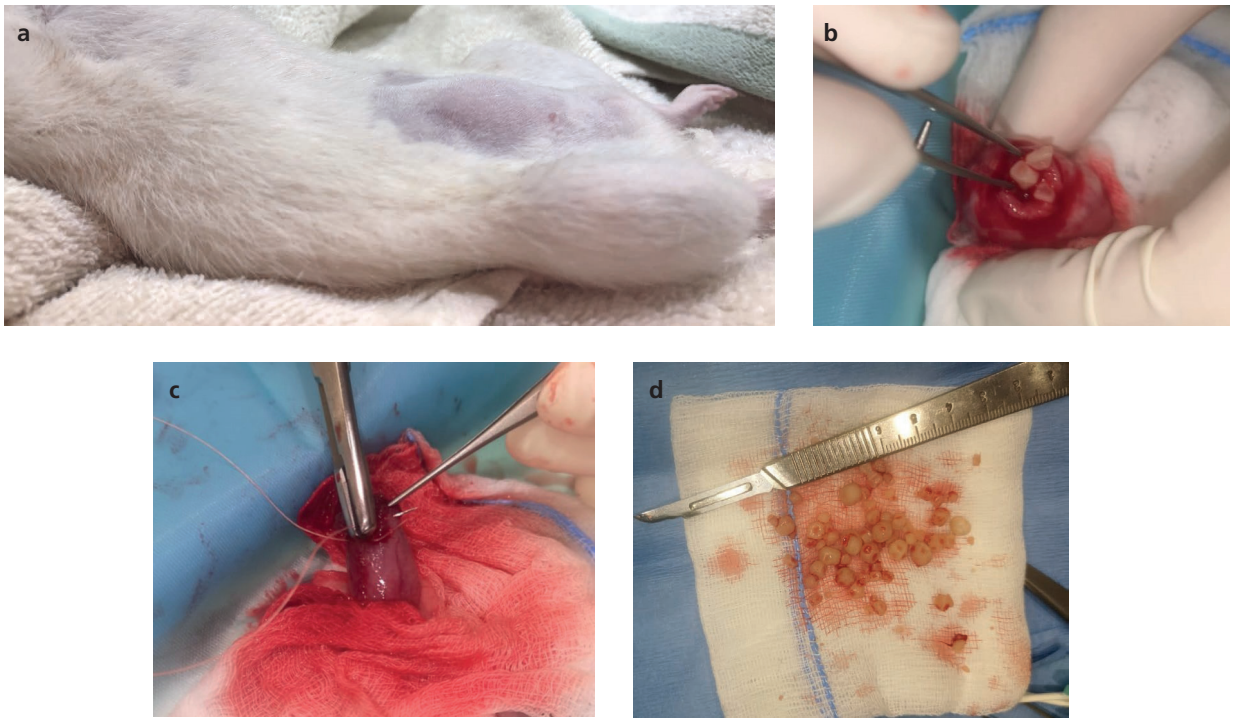


Figure 4.23

a The rat radiographed in Figure 4.22 prepared for cystotomy surgery. The bladder is so full of calculi it is visible through the abdominal wall.

b The cystotomy is performed the same as more familiar species.

c The bladder is repaired the same as more familiar species.

d The stones removed from the bladder.

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5 Hamsters

Hamsters are commonly thought of as children's pets but, in my clinic, they are frequently presented by their adult owners. The most commonly presented species of hamster is the Syrian hamster (*Mesocricetus auratus*) occasionally called the golden hamster (the long-haired variety is known as teddy bear hamster) and is the largest of the hamsters kept as pets. *Traditionally considered nocturnal, new research suggests most hamster species are crepuscular in the wild*.

The Syrian hamster originated from northern Syria and southern Turkey. They traditionally occupied rocky areas of unforested grassland and could also be found in sandy clay soil of agricultural land (likely due to being near an easily accessible food source). Their burrows vary in depth from 36 cm to 106 cm and the mean length from 200 cm to 900 cm. They excavate multiple chambers and each system is occupied by a single animal. *They are highly territorial and should be kept singly*. They are generally easy to handle and once fully awake they generally will not bite if they have been regularly well handled.

O'Neill *et al*. (2022) found that after the Syrian hamster, the Djungarian hamster was the second most commonly presented hamster in the UK with the Roborovski being the third most commonly presented.

The dwarf hamsters kept as pets are all members of the *Phodopus* genus and go by multiple names, which can be very confusing (I found it confusing for a long time).

The Djungarian hamster (*P. sungorus*) is also known as the Russian dwarf, striped dwarf, Siberian hamster, Siberian dwarf, and the winter white dwarf. In the wild this species changes to a completely white coat, however this is rarely seen in captivity due to static day lengths being provided. They originate from Kazakhstan, Mongolia, and Siberia. There are various coat colours available now, but they all have a thick dark grey dorsal stripe, furry feet, and the fur on their ventrum is white.

The Roborovski hamster (*P. roborovskii*) is also known as the desert hamster, Robo dwarf hamster, and dwarf hamster. They originate from the desert regions of Central Asia, Kazakhstan, Mongolia, and China. They are found in areas of loose sand and vegetation. There are various coat colours available now, but they have *no dorsal stripe* and they have eyebrow-like white spots above their eyes.

The Campbell's dwarf hamster (*P. campbelli*) looks very similar to the Djungarian hamster and is also occasionally called by the name Siberian hamster to confuse matters further. They have smaller ears than the Djungarian hamster, no dark fur on their crown, a thinner dorsal stripe, and the fur on the ventral abdomen is grey. They originate from Kazakhstan, Mongolia, China, and Russia. They inhabit burrows in open grassland and semi-arid desert regions.

The dwarf hamsters are more social and can be kept in single or mixed sex groups. Adult animals should not be introduced to an established group as fighting to the death may occur. If the enclosure is not big enough, fighting may occur even in established groups.

They are myomorph rodents, *their incisors continuously grow*, and the labial surface of the incisors have iron deposition within the enamel resulting in an orange brown colouration. Conversely, their *cheek teeth do not grow throughout life* and are close rooted. The gestation period varies slightly between species (see Table 5.1 for biological data) and they birth altricial young.

The average lifespan is commonly quoted as 2–3 years, although in my experience 1.5–2 is much more commonly seen. O'Neill *et al*. (2022) found that the median age at death across all hamster species was 21 months (1.75 years). As a prey species, they have evolved to *hide disease and pain* to prevent predation. Despite being kept in captivity for

Table 5.1 Biological data.

Parameter	Reference range
Average longevity	1.5–2 years
Respiratory rate	35–200 bpm
Heart rate	276–500 bpm
Body temperature	38–39.5°C (100.4–103.1°F)
Maintenance fluid therapy	75–100 ml/kg/day
Sexual maturity	6–8 weeks
Gestation	15–18 days depending on species

various reasons (lab, pets), hamsters still maintain many of their wild ancestors' behaviours.

Educating owners on husbandry and how to improve the welfare of their pet (e.g. environmental enrichment) should be part of a standard veterinary visit. New pet owners may be more likely to seek advice but every day is a school day and we need to be aware that owners may confuse love and dedication to their pet with appropriate welfare, this is where husbandry registration forms are invaluable to allow us to have a non-confrontational overview of how the pet is being kept.

The ethology of hamsters in the wild can provide important insight on husbandry practices that meet the hamsters' evolutionary requirements.

Enclosure and enrichment

The temperature inside a wild hamster burrow is approximately 16.5 °C (62 °F), as a result hamsters may find very warm rooms stressful. During the summer months in particular, care should be taken to ensure the hamster does not overheat. They evolved to live in areas with relative humidity of 30–40% and may find higher levels of humidity difficult to deal with. *Syrian hamsters (but not dwarf hamsters), may enter a state of hibernation if exposed to temperatures of 5 °C (41 °F) or lower*. Any seemingly dead Syrian hamster that has been exposed to low temperatures should be gently warmed (unless it is in rigor mortis) to confirm death, as the heartbeat may be extremely slow and difficult to detect.

Positioning of the enclosure within the house is important. Hamsters hear into the ultrasonic range of frequencies and can find ultrasonic sounds emitted by televisions, stereo systems, or refrigerators distressing. Likewise, vibrations caused by washing machines or tumble driers may also result in chronic stress.

Hamsters have large home territories and are extremely active animals, who may travel several miles a night in the wild. *Most commercially available hamster cages are far too small*. Fischer *et al*. (2007) demonstrated that hamsters kept in cages with less than 1 m^2 floor surface area displayed abnormal behaviours. *Climbing along the top of the bars of the cage is an indicator the enclosure is too small*.

Therefore, we should be recommending a floor surface area of a minimum 1 m^2 when choosing a cage. A cage 80 cm × 50 cm only has 0.4 m^2 of floor surface area, whereas a cage 120 cm × 90 cm has 1.08 m^2 of floor surface area.

They are burrowers by nature and so burrowing is an instinctive requirement. A deep substrate of at least 40 cm should be provided to allow this natural behaviour. Insufficient substrate depth is associated with an increase in stereotypical behaviour, aggression, and reduced food intake.

Various substrates are available. The ideal is absorbent, dust free and free from perfumes. Shavings may be suitable for some hamsters, whereas others may develop allergic rhinitis. Paper-based bedding with dust extracted is available. Allowing the hamster to shred cardboard and or toilet paper to mix through the substrate provided environmental enrichment while potentially making the substrate more suitable for building tunnels.

Commercially there is a huge range of toys aimed at rodents. It is important to provide safe wooden toys to provide ample gnawing opportunity which is important for maintaining the length of their incisors while also satisfying their innate requirement to gnaw. Opaque, plastic tubes can be buried in the substrate to provide a fake tunnel which has been shown to reduce stress, but should be monitored for signs of damage caused by gnawing, which may result in sharp edges.

Solid wheels can be provided for running. When rodent wheels have been placed outdoors, various wild rodent species have chosen to utilise it, spending time running. This demonstrates that it is indeed a pleasurable or at least a valued resource. Pet hamsters have been shown to travel up to 4 miles in an evening running on their wheels! To prevent painful spinal conditions, it is important to provide a large enough wheel that they do not have to bend their back while running. They should be able to run with a straight back and without having to lift their head. For a dwarf hamster, that means providing a wheel of at least 20 cm in diameter and for a Syrian hamster a 30 cm diameter solid sided wheel.

Prey species find wide open spaces with no ability to hide extremely stressful. *Hamster balls essentially mean the hamster is in a wide open space with no ability to hide and so should not be used*.

A nest box with soft nesting material should be provided. *Cotton wool type nesting material should be avoided as it can become tangled around toes leading to avascular necrosis*. Using kitchen or toilet roll paper as nesting material gives the added fun of allowing the hamster to shred the paper to their preferred length and width for nesting.

Weekly cleaning is advised but it is *important to leave some soiled bedding behind to ensure the familiar smell is maintained* for the pet which reduces their stress.

Diet

Hamsters are omnivores and exhibit coprophagy. They gather food into their large cheek pouches and often retreat to a hide to consume. Food will be cached, which can make it *difficult for owners to establish how much the hamster is actually consuming*. Hoards should be regularly cleared to prevent spoilage of food.

Muesli type diets lead to selected feeding and nutritional imbalances; therefore, a complete pelleted diet should be given. Oxbow Animal Health Essentials, Burgess and Supreme Petfood Science Selective are three options for pelleted complete hamster diets. This can be supplemented, for enrichment, with small amounts of fresh vegetables

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(e.g. broccoli, herbs, carrot) and fresh non-citrus fruits (e.g. apples, pears, and berries) several times weekly. Live foods such as mealworms or crickets may be offered once or twice weekly, but it is important that owners feed the insects a balanced diet or they will be of poor nutritional value to the hamster. For owners that cannot stand the thought of feeding live food, small pieces of cooked lean meat, or small pieces of boiled egg can be offered. Hay can be offered to increase fibre intake. *Introduce any new food type slowly as diarrhoea can result from sudden dietary changes* in hamsters.

Sugary treats should be avoided as diets high in sugar lead to periodontal disease, caries, and colo-colic intussusception is reported in hamsters fed high levels of sucrose. *Sticky foods such as sweets and cooked rice can lead to impacted or prolapsed cheek pouches*.

Water

Clean water should be provided by sipper bottles. The average water consumption is: 5–10 ml/100 g body weight/day, and *female Syrian hamsters drink significantly more than males*.

Sexing

Sexing hamsters is performed by looking at the genital–anal distance.

Male

Male hamsters have a much longer distance between the genital opening and the anus compared to the female. Mature male hamsters also have very large, long testicles which are situated caudally to the prepuce and often obscure the anus, unless of course he has been castrated. This gives the hind end of the hamster a much more rounded appearance than the female (Figure 5.1).

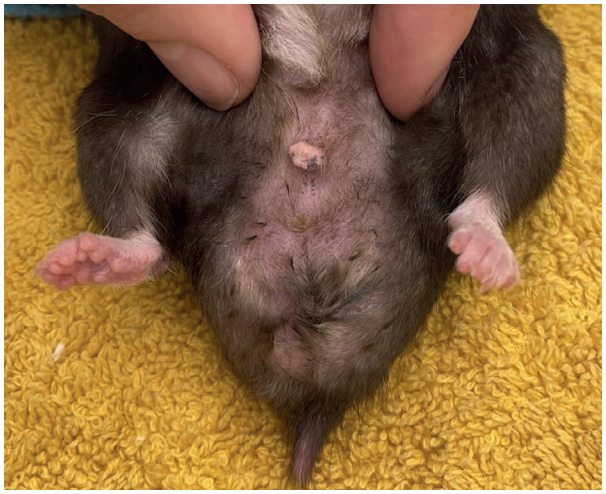


Figure 5.1 Male hamster, note the prominent testis and the long genital anal distance (the anus is obscured by the scrotum).

Male hamsters do not have nipples. Owners will occasionally present male hamsters to the clinic, worried about tumours, when they see the testicles fully descended into the scrotum.

Female

Female hamsters have six to seven pairs of nipples. The female hamster has three urogenital openings close together. The urethral papillae, the vulvar opening (covered with a membrane unless in oestrus) and then the anus (Figure 5.2).

Preventative health care

There are no vaccinations available for hamsters and routine worming or ectoparasite control is unnecessary.

Hospitalisation

They are a prey species, and so are extremely sensitive to stress caused by the sight, sound, and smell of predators. The cage set up in the hospital is the same as advised for rats (see Chapter 4).

Venipuncture sites

Hamsters find restraint incredibly stressful and are much faster to resort to biting than the other rodents in my experience. *I always anaesthetise* hamsters prior to venipuncture, even though anaesthetics may result in alterations of haematology.

Owing to the small size of hamsters, especially dwarf hamsters, a smear should be made at time of sampling and be examined in-house or sent to an external laboratory for haematological examination. That way, the volume collected can be used for biochemistry. The maximum recommended volume of blood that should be taken is 1% of the body weight but in ill rodents we should aim to take less than this.

Many exotics texts describe performing a cranial vena cava stick to draw blood. This carries the risk of death due to uncontrollable haemorrhage. Laboratory vets and technicians have perfected this technique with very few bad outcomes, but they are performing this very regularly whereas clinical



Figure 5.2 Female hamster, note the short urogenital anal distance.

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veterinary staff do not. Personally, I would not do this to one of my own pets therefore I will not do it to a client's.

Gingival vein

This is a good vein to use and results in no haematoma, minimal bruising, and no impact on food prehension. Once anaesthetised, the hamster is placed in dorsal recumbency. A 26 g needle and a 1 ml syringe is required. The vein is located midline, immediately below the gingival – incisor edge, between the mandibular incisors. Pull the lower lip caudally (Figure 5.3) The vein is entered in a caudal direction using a 30–60 degree angle and penetrating the mucosa 2–4 mm deep. Once there is blood in the hub of the needle the lower lip can be released.

In Syrian hamsters, the lateral saphenous vein can also be used.

Biochemistry profiles can be run 'in-house' using our usual mammalian profiles or sent to an external laboratory. Both options have pros and cons. The main pro for running in-house is that you obtain your results quickly whereas the main con is that you have to interpret whereas many labs will provide interpretation upon request.

There is a diurnal variation in the leukocyte concentration so time of sampling will impact the results. *The main leucocyte is the lymphocyte which can account for 75% of the leucocyte count*. See Tables 5.2 and 5.3 for reference ranges for biochemistry and haematology. The erythrocyte lifespan is 50 – 78 days.

Dehydration and fluid therapy

The same methods of assessment and treatment used for rats can be used in hamsters (see Dehydration section in Chapter 4).



Figure 5.3 The gingival vein.

Table 5.2 Syrian hamster biochemistry.

BIOCHEMISTRY (Heatley & Russell, 2020)	REFERENCE RANGE (UK units)	REFERENCE RANGE (US units)
ALKP	107–127 IU/l	107–127 U/l
ALT	81–112 IU/l	81–112 U/l
AST	40–51 IU/l	40–51 U/l
AMYL	1012–1512 IU/l	1012–1512 U/l
GGT	4.2–7.0 IU/l	4.2–7.0 U/l
LDH	136–167 IU/l	136–167 U/l
CK	467–1281 IU/l	467–1281 U/l
LIPASE	256–285 IU/l	256–285 U/l
GLU	9.1–11.3 mmol/l	164–204 mg/dl
CHOL	2.9–3.4 mmol/l	111–131 mg/dl
TRIGYLCERIDES	2.2–3.1 mmol/l	197–275 mg/dl
TBIL	3.08–4.10 µmol/l	0.18–0.24 mg/dl
UREA	7.85–9.28 mmol/l	22–26 mg/dl
CREA	28.3–33.6 µmol/l	0.32–0.38 mg/dl
TP	65–68 g/l	6.5–6.8 g/dl
ALB	39–42 g/l	3.9–4.2 g/dl
GLOB	14–34 g/l	1.4–3.4 g/dl
CA	3.1–3.5 mmol/l	12.5–13.9 mg/dl
PHOS	2.4–2.7 mmol/l	7.4–8.4 mg/dl
K	3.9–5.5 mmol/l	3.9–5.5 mEq/l
Na	147–149 mmol/l	147–149 mEq/l

Table 5.3 Syrian hamster haematology.

HAEMATOLOGY (Heatley & Russell, 2020)	UNITS	REFERENCE RANGE
Red blood cells	10^{12}/l	6.6–10.0
Packed cell volume	%	44–47
Haemoglobin	mmol/l (g/l)	8.87–10.74 (143–173)
Mean corpuscular volume	fl	59.8–62.0
Mean corpuscular haemoglobin	pg	19.5–22.1
Platelets	10^{9}/l	282–360
White blood cells	10^{9}/l	4.4–8.3
Differential blood count (relative values)		
Monocytes	%	4–8
Lymphocytes	%	65–73
Neutrophils	%	20–28
Eosinophils	%	2–6
Basophils	%	0–1

I do not agree with subcutaneous fluid administration. It is *incredibly painful* and dehydrated patients will not absorb fluids via this route as efficiently. Hamsters hide pain so relying on the fact your patient 'allows' you to perform subcutaneous administration is not rationale for it not being painful. The fact that hamsters have a large amount of movable skin on their dorsum is not rationale for it being non-painful in my opinion.

Intravenous (IV) catheter placement is difficult in Syrian hamsters and virtually impossible in the dwarf hamsters. Hamsters that are over 5% dehydrated will require either intraperitoneal fluids or a brief anaesthetic and intraosseous catheter placement in the proximal tibia as per rats. I will use a 26 g needle in dwarf hamsters. Ongoing analgesia is important, intraosseous (IO) catheter flushing and administration of fluids is painful in humans therefore should be considered painful in other species.

Consider using midazolam (0.5–2 mg/kg SC, IM) to reduce stress during hospitalisation and treatment.

Drugs contraindicated in hamsters

If in doubt, always check the formulary as it will often tell you whether it is safe to use a drug in any given species. My favourite formulary is James Carpenter's *Exotic Animal Formulary* now on its sixth edition (Carpenter & Marion, 2022).

Hamsters are *particularly prone to developing fatal clostridial enterotoxaemia following oral administration* of the following antibiotics, and they should never be administered penicillins (including ampicillin, amoxicillin), cephalosporins, clindamycin, erythromycin, lincomycin, vancomycin, dihydrostreptomycin, streptomycin, bacitracin, and oral gentamicin.

Tylosin should be used with extreme caution in hamsters as although there are published doses for hamsters (I have on occasion used Tylosin without ill effect in hamsters), there are also reports of toxicity and fatal clostridial enterotoxaemia.

Prednisolone should be avoided or used with extreme care due to the immunosuppressive side effect in this species.

Species-specific anatomy/behaviour

Hamsters produce 'red tears' (porphyrin from the Harderian gland) which can be excessive during times of stress or disease.

Obligate nasal breathers.

Hamsters are myomorphs with elodont–hypsodont incisors (elodont: continuously growing throughout life; and hypsodont: long anatomical crown compared to the root). They have two incisors in the maxilla and two in the mandible. The lower incisors are much longer than the upper incisors. The incisors usually have a yellow orange colouration due to the iron deposition within the enamel.

The cheek teeth are anelodont (anelodont: limited growth period therefore truly rooted).

Dental formula: $2 \times (\text{I } 1/1; \text{C } 0/0; \text{P } 0/0; \text{M } 3/3) = 16$.

The mandibular symphysis remains cartilaginous throughout life.

Hamsters have large cheek pouches which they use to carry food, bedding, and even young. The cheek pouches extend caudally to the scapula. *It is important when performing a subcutaneous injection to administer them over the middle or caudal part of the body. This is to prevent accidental injection into the cheek pouches*. Hamsters use their front legs to empty their cheek pouches. In cases of forelimb amputation, the hamster may struggle with emptying the ipsilateral pouch. Owners should be advised to monitor for impaction of the pouch.

The tight cardiac stomach sphincter prohibits vomiting or eructation.

Hamsters have a stomach quite unlike other rodents. There is a distinctly compartmentalised stomach: a forestomach which is similar to a rumen (they can absorb the volatile fatty acids (VFA) that are produced by fermentation in the forestomach) and a glandular stomach. They also utilise hindgut fermentation and perform coprophagy.

Male hamsters have a prominent urethral papilla, large testicles, open inguinal canals, and a longer genital anal distance.

Female hamsters have three openings: urethral papillae, vaginal opening, and the anus. They have a shorter genital anal distance. They have short gestation periods (which vary slightly depending on species) and birth altricial young.

Female Syrian hamsters may show a copious, creamy coloured, vaginal discharge just after ovulation (day 2 of oestrus cycle). This post-ovulatory discharge is extremely 'stringy' and touching it will result in it stretching between the probe and the vagina (Figure 5.4).



Figure 5.4 Although this hamster also has diarrhoea, the vaginal discharge is the normal post-ovulatory discharge that Syrians can display. It is easy to see why visually it can be mistaken for a pyometra.

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Visually it can be confused with a pyometra, therefore it is important that further diagnostics are undertaken before diagnosing a pyometra in a Syrian hamster, such as making a smear, staining, and examining the discharge microscopically.

Female hamsters will have a fertile post-partum oestrus.

Syrian hamsters will enter a state of hibernation if exposed to temperatures of 5°C or lower, the other species of hamsters do not.

Syrian hamsters have large bilateral scent glands (which are often pigmented) on their flanks. The dwarf hamsters have ventral scent glands. The Djungarian hamster also has a scent gland at the commissures of the mouth.

Hamsters hide illness and pain. Again, from an evolutionary point of view this makes complete sense because if you show illness or weakness, you get eaten. So, by the time a hamster looks overtly ill or painful, they literally no longer care if they live or die. Many owners underestimate this and unfortunately many of us, the veterinary profession also underestimate it.

Pain in hamsters

One of our jobs as veterinary professionals is to *actively look for pain*. In Chapter 1 we discussed that all vertebrates have the same anatomical and physiological pain pathways.

> If we accept that animals and people share similar mechanisms of pain and analgesic action then we should realise that the variation of analgesic efficacy in different individual people means that it is highly likely that a similar variation will be seen in animals – further reinforcing the need for regular pain assessments.

Previously published behaviours related to pain in hamsters include adopting a hunched position with head down, reluctance to move, increased depression, increased aggression, extended sleep periods, and weight loss. *Observation of pain related behaviours will likely be complicated by the nocturnal/crepuscular nature of these animals*.

Edmunson *et al*. (2021) looked at indicators of post-operative pain in Syrian hamsters. They developed a Syrian grimace scale by adapting from the rat and mouse grimace scales. The outcome of this study was that they *could not find a consistent, reliable behaviour to evaluate pain in hamsters*. This makes assessment of pain even more challenging in our hamster patients.

In my experience, healthy hamsters will have wide opened round eyes. *Hamsters in pain are more likely to appear to be squinting*. I am aware that not all hamsters in pain will exhibit this and also that it may well vary with severity of pain. The stress of the journey will likely also cause pain to be masked. *I tend to err on the side of caution, and administer analgesia to ill hamsters. If the same condition or procedure is considered painful in another species, it will be painful in hamsters*. Hamsters in pain are much more likely to bite.

There is not much information available in the literature for analgesic doses in hamsters. Generally, I will use the same doses as for rats albeit I will start at the low end of the dose rate (see Chapter 4, Pain management in rats section).

Clinical exam

Hamsters that are regularly handled by the owners are often easy to examine, but I will always ask the owner if they are friendly with strangers, and bear in mind that if in pain or frightened then they are likely to bite. I will ensure I have a face cloth or dish towel available for handling the hamster if necessary.

I rarely scruff a hamster anymore. The risk of exophthalmos (especially in Russian hamsters), distress to the hamster, the amazing ability of hamsters to spin in their scruff and still bite, and the distress to the owners all outweighs any potential benefits in my opinion.

With Syrian hamsters, if they are asleep in a nest area that is liftable, I will gently tip the hamster out and allow it to fully wake up before assessing the temperament of the animal. If the animal seems friendly, relaxed, and interested in interacting, I will use both hands to scoop the hamster up. If the hamster is rearing, hissing, or screaming (all signs of fear or aggression) I will use a small towel to scoop the hamster up and hold the hamster in a modified bird ringer's grip through the towel (see Figure 5.5). *Consider the administration of midazolam (0.5–5 mg/kg IM), or anaesthetising with isoflurane briefly, in fearful animals to facilitate a better, safer clinical exam. Full written consent of the owner should be sought before administration*.

Dwarf hamsters can be very fast and challenging to catch. I will either request the owner catch the hamster (if the owner is confident and regularly handles the pet), or I will use the cardboard roll from inside toilet paper as a disposable tunnel to capture the animal and tip the hamster into a face cloth or small towel for restraint. *Dwarf hamsters are much more likely to jump* in my experience, therefore I will always examine them over a table.

I perform the same clinical exam as I do for any species (with the exception that I will palpate and if needed empty the cheek pouches) (see Chapter 4 Clinical exam section for more detail). When presented with friendly Syrian hamsters, I will examine them either on my knee or arm. Fractious animals are examined while restrained in the towel, on the table.

General anaesthesia

Hamsters cannot vomit and therefore they do not need to be starved before an anaesthetic. Owners are used to starving themselves or their dogs and cats prior to surgery, so we

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Figure 5.5 The hamster is being held in a modified bird ringer's grip, with the head between the middle and pointer finger and body being cupped by the palm and other fingers.

need to be clear when we book hamsters in for surgery that they should not be starved. Advise owners to bring a 'packed lunch' of favourite and familiar foods.

> Bonded cage mates (dwarf hamsters only) should always be admitted, not only will this reduce stress during the hospitalisation period, help protect the bond between conspecifics, it also has been shown to reduce healing times and increase coping ability for aversive or painful experiences (Denommé and Mason, 2022).

Allow at least 1 hour to acclimatise to the ward after travelling to the clinic to reduce stress levels prior to induction.

I will remove food an hour before surgery to reduce the risk of food being in the cheek pouches.

Pre-anaesthetic blood tests

Owing to the small volume of blood that can be safely taken from hamsters, I rarely perform pre-anaesthetic blood tests. *All surgical procedures will result in some blood loss, taking a blood sampling prior to surgery may overwhelm the animal's ability to withstand the blood loss during surgery* (see Tables 5.2 and 5.3 for reference ranges).

Pre-emptive analgesia

Once admitted (and if not already on medications) I administer meloxicam 0.6–1 mg/kg and/or another analgesic if appropriate depending on the surgical procedure. These can either be given orally or by subcutaneous (SC) injection.

Pre-medication

Whether I administer a pre-medication is dependent on the stress levels of patient and the surgical procedure being performed. *It is important to consider the stress of being handled and being injected. Rodents find injections very stressful and as they have small muscle masses IM injections are very painful and may need to be given in multiple sites depending on volume.* I think very carefully before I administer pre-medicants in rodents as I have to consider the type of surgery (and which analgesics will be required) and decide whether the stress and pain of the pre-med is worse than the mask induction. In my experience mask induction while wrapped in a towel burrito, or using a dog mask as an induction chamber results in a fast, seemingly minimal stress induction, whereas injections always result in vocalisations and obvious stress. The drugs used for this are the same drugs we use in more familiar mammals. The doses can be found in Table 5.4.

Local anaesthesia for muscle splash blocks or infiltration

Combining the drugs allows for greater effect with a lower recommended dose. Lidocaine has faster onset but a shorter duration of action (60–120 minutes) whereas bupivacaine has a slower onset of action but a longer duration (180–480 minutes). Mixing 4 mg/kg of lidocaine with 2 mg/kg of bupivacaine in a syringe then diluting the mixture with saline to increase the volume of the solution and allow easier splash blocking (exactly what it sounds like). Drip the solution onto the wound in the muscles) or infiltrate an area. Pulling up the required dose and pre-diluting, prevents accidental overdose.

Induction

There are several anaesthetic protocols available for use in hamsters. There is an argument to be made that the safest anaesthetic is the one you are used to.

Table 5.4 Premedicant dose.

Drug	Dose and route	Reversal
Midazolam	1–2 mg/kg IM SC	Flumazenil 0.1 mg/kg SC
Midazolam + butorphanol	1–2 mg/kg IM SC + 0.2–0.5 mg/kg SC	Flumazenil 0.1 mg/kg SC
Medetomidine + butorphanol	0.1 mg/kg IM + 2 mg/kg IM	Atipamezole 0.5–1.5 mg/kg IM SC



Figure 5.6 My preferred method of induction is to use a face mask as an induction chamber.

My preferred inhalational gaseous anaesthetic is isoflurane. Reportedly, most rodents react more favourably to the smell of sevoflurane but in my experience, many dislike the smell just as much and in the UK, isoflurane is the only licensed gaseous inhalational anaesthetic for rodents.

My preferred method of induction is to use a face mask as an induction chamber (Figure 5.6). Massage any food out from the cheek pouches prior to placing in the face mask. Have cotton buds (Q tips) to hand to swab the mouth clear of food or saliva post-induction.

Allow four breaths of 100% oxygen (this is all that is required to fully saturate the blood with oxygen), then turn the vaporiser straight to 5% isoflurane. Initially the hamster may wriggle but it will quickly become induced (most are less than 30 seconds) with minimal stress. In most cases the hamster will not have even passed urine or faeces (which frequently occurs due to stress and commonly passed when induction chambers are utilised).

Turn the vaporiser to 2.5% and assess anaesthetic depth (using the same reflexes we are used to in more familiar mammals a moistened cotton bud (Q tip) can be used to check the blink reflex), adjusting the setting on the vaporiser as necessary.

The large cornea should be protected by applying an ocular lubricant. *Owing to the sensitivity of the hamster lens and retina, thought should be given to taping the eyes shut to prevent damage caused by the brightness of the surgical lights.*



Figure 5.7 The nose of the hamster may fit perfectly into the end of the anaesthetic circuit.

Maintenance

Tightly fitted face masks can be made from syringe cases or a latex glove taped over the end of anaesthetic circuit with a small hole made in the centre of the glove for the nose to fit through. Alternatively, the nose of the hamster may fit perfectly into the end of the anaesthetic circuit (Figure 5.7).

Another option is to utilise the fact hamsters are obligate nasal breathers, pass a nasogastric tube medially, and ventrally into the nostril approximately 0.5 cm, tape the tube to the head and administer the oxygen and gaseous anaesthetic mixture via the tube.

Both of the above methods help reduce environmental contamination with gaseous anaesthesia.

I use a T-piece circuit, usually with an oxygen flow of 1.5–2 l/minute. Oxygen flow of below 0.5 l/minute results in inaccurate vaporiser output. High fresh gas flow will result in increased chilling of the hamster due to respiratory heat loss. To calculate a more exact flow rate required for each hamster the below formula can be used:

Oxygen flow rate = 2 × minute volume (MV)
MV = TV × RR

Tidal volume (TV) can be calculated by multiplying the weight of the hamster (in kilograms) by 10 to 15. The respiratory rate (RR) for hamsters is 35–200 breaths per minute.

Monitoring

Monitoring the anaesthetic is the same as for more familiar species. Multiparameter monitoring is possible. MAI Animal Health makes the Vetcorder Portable Monitor which is suitable for all species, including dogs and cats, for under £1,000. It can record: an electrocardiogram (ECG), heart rate, pulse oximetry (placed on tongue or paw), and rectal temperature. If recording an ECG rather than use

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Alligator clips (which can traumatise the skin), insert small gauge needles through the skin (25 g) and attach the clips to the needles.

If that is outside the practice's budget, a soft, flexible tipped rectal thermometer can be used to monitor rectal temperature. Hypothermia is a major risk factor in anaesthetic deaths. A human foetal Doppler can be purchased online (I used eBay) for around £50, ensure you request the 8 mHz probe rather than the standard 2 mHz one. An infant stethoscope can be used to monitor heart rate if the Doppler is not an option. Respiratory rates can be counted, and pulse oximeters can be used from other species but they may struggle to get an accurate reading due to the high heart rate (276–500 beats per minute in hamsters).

Occasionally the spring on the oximetry probe is too strong and can occlude the vasculature, I place a cotton bud (Q tip) inside the probe (but not over the sensor) to alleviate the pressure and allow the reading to occur. Good readings are often possible from the foot (not in the Russian hamsters due to the fur on the feet).

Theatre

Hamsters have a high surface area to volume ratio that means they lose heat rapidly. The resulting hypothermia can cause fatalities. Keeping the ambient temperature in the theatre high, placing a towel on the operating table, and using a plastic drape (also allows visualisation of your patient!) are all easy ways to help mitigate heat loss. Minimise the number of times the door is opened as each time it happens the ambient temperature in the room will drop. Hypothermia leads to prolonged recovery and increased risk of death.

Positioning

Rodents are diaphragmatic breathers. Their small thoracic cavity in comparison to their large abdominal cavity can make breathing more difficult due to the weight of the abdominal organs sitting against the diaphragm. Placing a folded drape or face cloth under the thorax elevates the thorax and allows the abdominal organs to move away from the diaphragm. Ensure the head and neck are straightened to allow for easier breathing.

Surgical preparation

Hamster skin is delicate and can be traumatised by inappropriate (and sometimes even appropriate) clipper technique. To conserve body temperature, minimise the surgical clip. Only one agent is used for surgical scrub, either chlorhexidine or an iodine-based skin disinfectant depending on the surgeon's preference. We do not use a surgical spirit-based spray as during evaporation this causes loss of heat. Using warm water for the surgical scrub will help reduce heat loss.

Transparent, plastic surgical drapes are ideal as they allow visualisation of the rat while conserving body heat. The required surgical window can be cut into the drape and a surgical spray adhesive is used in lieu of towel clips.

EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) can be applied to the wound postoperatively if no other local anaesthetic infiltration has been performed in the skin. This helps with the immediate post-operative skin pain. Since I have started using this technique, I have seen a faster return to eating on recovery.

Recovery

If medetomidine has been used it can be reversed with 0.5–1.5 mg/kg atipamezole SC, reversing the medetomidine will also reverse the analgesia provided by medetomidine but that is not reason enough for not reversing.

Maintain the hamster on oxygen. Shivering thermogenesis is a sign of recovery from anaesthesia. Continue to monitor while in an incubator or a warm room (ideally around 26–29°C (80–85°F)) until up and moving. If the hamster is hypothermic, continue to take the rectal temperature every 15 minutes until it reaches within 1°C of normal (38°C (100.4°F)) then reduce the temperature of the room or incubator to 22°C (72°F).

Offer favourite foods once the animal is fully conscious. Many hamsters will readily take warmed human baby food from a spoon or a syringe. Alternatively, a high energy, easily digested diet such as Harrisons Recovery or the semi-elemental diet EmerAid Omnivore can be fed. In most cases with normal body temperature and good analgesia, eating will not be an issue (see Table 5.3).

Specific conditions in hamsters

Cheek pouch conditions

Impaction

Owners like to feed their pets treats and unfortunately, just as in more familiar species, the inappropriate food types can cause issues. *Sticky foods such as boiled sweets and cooked rice can become adhered to the lining of the cheek pouches*. This can lead to either impaction or prolapse of the cheek pouch (due to the hamster trying to empty the food item). Over stretching the pouch with dried foods and bedding materials may also lead to impaction.

Impaction may present with facial swelling, salivation, and anorexia. The hamster may be pawing at the pouch and there may or may not be a smell associated with the impaction. Abscessation of the cheek pouch may occur due to the impaction or due to damage caused by sharp food stuff being stored in the pouch. Owners should be advised that dental pathology or infection are common predisposing factors for cheek pouch impaction.

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Treatment of impaction

A general anaesthetic is required for full assessment and treatment (see Anaesthesia section). The impaction can then be massaged out of the pouch, cotton buds or forceps can be used to assist. As dental pathology is a recognised predisposing factor, a careful examination of the oral cavity and dental arcades should be performed. Warmed saline can be used to clean the mucosa. The mucosa should be assessed for infection, consider biopsy if there are areas of swelling. Any abscesses should be opened (ideally take a swab for culture and sensitivity) and flushed out with warmed saline. Leave the abscess open to allow for drainage and healing by secondary intention. Analgesia should be given and trimethoprim sulphonamide 30 mg/kg q12hrs can be started in cases of infection while waiting on the bacteriology report.

Prolapse

Cheek pouch prolapse may occur secondary to inappropriate foods, impaction, inflammation, infection, or neoplasia. The prolapsed pouch can be quite dramatic in appearance. Hamsters may chew or traumatise the everted cheek pouch. Necrotic, severely traumatised, cheek pouches or neoplastic lesions will necessitate the amputation of the cheek pouch. General anaesthesia is required for treatment.

Replacement of prolapse

If the tissue is severely oedematous, reduction of the oedema can be attempted by applying cotton wool soaked in a supersaturated saline or sugar solution, prior to replacement.

If the cheek pouch is healthy, it can be replaced using a 1 ml syringe to ensure the pouch is fully relocated. A single cruciate stay suture should be placed to prevent the cheek pouch from immediately prolapsing on recovery from the anaesthesia. Using a swaged-on, 4/0 (1.5 m), non-absorbable suture material, place the suture percutaneously full thickness into the cheek pouch, using the 1 ml syringe as a guide to 'bounce' the needle off. Remove the suture 7–10 days later.

Cheek pouch amputation

If the cheek pouch is necrotic, severely traumatised or contains a neoplastic lesion, then surgical amputation of the cheek pouch can be performed.

Clean the tissue with warm, dilute chlorhexidine. Place a haemostat across the base of the cheek pouch and amputate the tissue. Remove the haemostat and repair the tissue with cruciate sutures using a synthetic absorbable swaged-on suture material. Apply a local anaesthesia splash block to improve analgesia in the immediate postoperative period.

Advise owners to clean out the enclosure and ensure they remove all hoarded food. Feed the hamster on a suitable soft diet such as Harrisons Recovery, or EmerAid Omnivore for 3–5 days, either via syringe or offered in a small bowl. No dry food should be given for at least 3–5 days. This is to prevent the hamster trying to fill the pouch with food which will lead to stretching of the surgical site and potentially result in wound dehiscence. Continue analgesia for at least ten days.

Dental disease in hamsters

It is only the incisors that grow continuously in the hamster. The incisors are yellow/orange due to iron deposition within the enamel. The molars are multi-rooted. The dental formula is: 2 (I 1/1, C 0/0, P 0/0, M 3/3) = 16.

Hamsters can develop periodontal disease, dental caries, tooth decay and abscessation especially if fed on an inappropriate, high sugar diet. Maxillary molar abscessation may result in a retrobulbar abscess.

Incisor malocclusion may be congenital, secondary to trauma, secondary to infection, or secondary to neoplasia. Incisor malocclusion may be managed in the same way recommended for the rat (Figure 5.8 and see Chapter 4).

Exophthalmos

Inappropriate restraint via scruffing may result in bilateral exophthalmos which in most cases is temporary, the eyes returning to normal position once released. Unilateral causes include trauma, tumour, infection, tooth root abscess, and foreign body.



Figure 5.8 Extraction of a hamster's incisors using a 21 g needle. Photo: Dr Krissy Green.

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Protrusion of the eye in most cases will present as a unilateral condition, which is progressive in severity (unless caused by trauma which will present acutely), the eye will often remain sighted and the normal retropulsion will be lost. Exposure keratitis may be present and there will be a variable ocular discharge.

Owing to the *high occurrence of tooth root abscesses, radiographs are advised* to check for dental disease. If dental disease is present, enucleation alone will not be sufficient, the underlying dental disease will also require treatment. The recommended technique for enucleation is via the transconjunctival approach as described for the rabbit in Chapter 2.

Diarrhoea

Just as in more familiar species there are a range of causes of diarrhoea in hamsters, from dietary indiscretion (owner changing food too quickly, diet too high in fruit/vegetables, or giving too many treats), to infectious causes (bacterial and parasitic), to iatrogenic drug induced causes.

Minimising stress is important as in hamsters, *stress and the associated immunocompromise are common predisposing features of developing diarrhoea*.

> Many owners will describe their hamster as having 'wet tail', meaning diarrhoea; whereas the term 'wet tail' refers to a specific condition in hamsters called proliferative ileitis, see below.

Salmonella typhimurium and *S. enteritidis* are both reported as causing diarrhoea in hamsters, but treatment is not recommended in these cases due to the risk of zoonosis and that the hamster may become a lifelong carrier of *Salmonella* if it survives.

In general, cases of diarrhoea should be treated aggressively to ensure adequate hydration. If *Clostridial* spp. infections are suspected, then the use of doxycycline or metronidazole is advised as per below. If *Clostridial* spp infections are not suspected then trimethoprim sulfamethoxazole 30 mg/kg q12hrs for 1 week may be used, however care should be taken in dehydrated animals when using this antibiotic due to the risk of renal damage.

Dietary indiscretion

Treat with a correct diet, supportive care (fluids, warmth, food), bismuth subsalicylate 17.5 mg/kg q12hrs, and probiotics (Pro C Professional, Vet Ark, or Bene Bac Plus Small Animal, Pet Ag). Most cases should resolve with symptomatic treatments, but should antibiosis be required, metronidazole 15–20 mg/kg q12hrs can be given for 5–7 days or trimethoprim sulfamethoxazole 30 mg/kg q12hrs for 1 week.

Iatrogenic through inappropriate antibiotic use

> The gut flora in the hamster is mainly Gram positive, therefore antibiotics with strong activity against Gram positive bacteria will cause a potentially fatal dysbacteriosis, allowing overgrowth of opportunistic pathogens such as *Clostridium difficile* or *Escherichia coli*. *Clostridium difficile* produces toxins which are fatal to hamsters, inducing a haemorrhagic ileocolitis.

The clinical history will reveal recent antibiotic usage. *The following antibiotics should never be administered to hamsters*: penicillins (including ampicillin, amoxicillin), cephalosporins, clindamycin, erythromycin, lincomycin, vancomycin, dihydrostreptomycin, streptomycin, bacitracin, and oral gentamicin. *Tylosin should be used with extreme caution in hamsters as although there are published doses for hamsters* (I have on occasion used Tylosin without ill effect in hamsters), there are also reports of toxicity and fatal clostridial enterotoxaemia.

Clinical signs usually develop 3–5 days after oral administration of antibiotics.

Clinical signs include diarrhoea (may or may not be haemorrhagic), perineal staining, dehydration, emaciation, poor fur condition, and hunched posture, which may indicate abdominal pain.

Treatment must be aggressive, stop antibiotics and start supportive care. Supportive care includes fluid therapy (dehydration and electrolyte imbalance), provide thermal support in cases of hypothermia, assisted feeding, transfaunation (syringe feeding a slurry made from normal faeces), analgesia, and bismuth subsalicylate 17.5 mg/kg q12hrs.

Prognosis is guarded to poor.

Tyzzer's disease

Tyzzer's disease has a high mortality and is caused by an overgrowth of *Clostridium piliforme*. Clinical disease is not seen in immunocompetent hamsters and clinical outbreaks are always precipitated by severe stress (e.g. overcrowding, high environmental temperature and humidity, high parasite burdens, or severely inadequate diet).

Clinical signs can include depression, unkempt coat, acute pale yellow diarrhoea, dehydration, hunched abdomen, hepatitis, or sudden death with no prior clinical signs.

Diagnosis is suspected based on history and clinical signs but can be confirmed via Polymerase chain reaction (PCR) of faeces.

Prognosis is poor and treatment involves aggressive supportive care (fluids, warmth, assisted feeding), liver support (silymarin 50–200 mg/kg/day) and suggested antibiotics include doxycycline or metronidazole 15–20 mg/kg q12hrs.

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Proliferative ileitis

Proliferative ileitis, *also known as wet tail*, is a condition in 3–10 week old hamsters caused by an intracellular Gram negative bacterium called *Lawsonia intracellularis*. Stress is the main predisposing factor for developing clinical signs and the *mortality rate is in the region of 90%*.

Predisposing stressors

Stress-induced immunocompromise plays a role in development of disease. Stressors associated with development of clinical signs include weaning, dietary change, poor hygiene, multiple animals in close proximity, and transportation.

Clinical signs include foul smelling, watery diarrhoea (may be haemorrhagic in acute stage), wet perineum and back end, dehydration, rapid weight loss (emaciation), lethargy, pain (hunched back posture), irritability, abdominal distention, and bloody anus (with or without intussusception, with or without rectal prolapse). Death usually occurs 24–48 h after developing clinical signs. Immediately before death, commonly experience a sudden drop in core body temperature and convulsions.

Diagnosis is mainly based on clinical signs as the organism cannot be cultured using standard microbiological techniques.

If proliferative ileitis is suspected then aggressive treatment is immediately required but even with prompt, aggressive treatment the prognosis is poor, as *cases that initially survive may then develop intestinal obstruction, intussusception, or rectal prolapse*.

Owners should be advised that even if treatment works it may result in chronic subclinical infection, which raises the ethical quandary of whether we should be treating these cases at all.

Treatment

Treatment is aggressive and intensive. Fluid therapy (IV, IO, or intraperitoneal (IP)) to correct the dehydration and life-threatening electrolyte imbalances is required. Assisted feeding (for example, Harrison's Juvenile Hand Rearing Formula, or EmerAid Omnivore), warmth, analgesia, and bismuth subsalicylate 17.5 mg/kg q12hrs. The recommended antibiotic combination is: enrofloxacin 10–20 mg/kg q12hrs and metronidazole 15–20 mg/kg q12hrs. (See Table 5.5 for the commonly used antibiotics in hamsters.)

Rectal prolapse

Rectal prolapse develops secondary to severe diarrhoea or prolonged tenesmus. As with more familiar species, prolonged tenesmus may be secondary to diarrhoea, endoparasites, inflammation, intussusception (which may present as constipation), or neoplasia.

Prognosis is dependent on multiple factors including the cause, length of tissue prolapse, time prior to presentation, and the viability of the prolapsed tissue. *The hamster will be in a lot of pain, and full assessment should be made under general anaesthesia and analgesia must be given.*

In cases of a simple, non-traumatised, small rectal prolapse that is quickly presented the prognosis is favourable. Under general anaesthesia, palpate the abdomen to rule out any masses indicating intussusception or neoplasia. Using a lubricated cotton bud (Q tip), gently reposition the prolapse and ensure patency. Place a purse string suture around anus, making sure that the hamster can still pass faeces (advise owner to monitor for faecal output). Apply EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) to the anus post-operatively. Leave the suture in place for at least 3 days

Table 5.5 Antibiotics commonly used.

Drug	Dose
Azithromycin	30 mg/kg PO q24hrs
Ciprofloxacin	10–20 mg/kg PO q12hrs
Chloramphenicol	30–50 mg/kg SC, IM, IV, PO q8–24hrs
Doxycycline	5 mg/kg PO (no dairy) q12hrs, long-acting formulation 50 mg/kg q7days SC
Enrofloxacin	10–20 mg/kg PO SC q12hrs (IM painful and may result in sterile abscess/muscle necrosis)
Marbofloxacin	5 mg/kg PO SC q12–24hrs
Metronidazole	15–20 mg/kg PO q12–24hrs
Oxytetracycline	50 mg/kg PO q12hrs
Silver sulfadiazine cream	Topical q12–24hrs
Trimethoprim sulfamethoxazole	30 mg/kg PO q12hrs
Tylosin (use with caution: reports of toxicity and fatal clostridial enterotoxaemia in hamsters)	2–10 mg/kg PO SC q12hrs 0.5 mg/ml (500 mg/l) drinking water

(max 7 days). Analgesia should be given for 1 week and the underlying cause should be treated if identified.

If the hamster has chewed the prolapsed tissue, or the tissue is necrotic then euthanasia is advisable as it would be very difficult to perform a bowel resection in this region of prolapse.

If a large portion of intestine has prolapsed via the rectum, then there is a high risk that an intestinal intussusception is present. *Intussusception carries a poor prognosis*. Provided the tissue is viable and the owner wants to try surgery, then exploratory surgery would be required, both to reduce the prolapsed intestine, and surgically resect the intussusception (or neoplasia) and perform an end-to-end anastomosis. The surgical technique is the same as in more familiar species, just much smaller.

Endoparasites

Hamsters can be infected with a variety of endoparasites. *The clinical significance of most of these parasites is unknown and therefore it should be remembered that presence does not correlate with cause of disease*.

Several species of protozoa (*Hexamita* spp., *Giardia* spp., *Tritrichomonas muris*, and *Entamoeba muris*) are reported in hamsters, although confusingly, it is reported that they are found in equal numbers in healthy and sick hamsters. Metronidazole can be given if there is suspicion of clinical disease associated with the protozoa.

Hamsters have their own species of pinworm, *Syphacia mesocricetus*, but they can also become infected with the mouse pinworm *S. obvelata*, and the rat pinworm, *S. muris*. Heavy burdens may be associated with poor coat, cachexia, and rectal prolapse. Diagnosis is via tape test of anus or faecal flotation. Treatment requires topical application of ivermectin 2 mg/kg and thorough cleaning of enclosure.

The only tapeworm seen in hamsters is *Rodentolepis nana*. Infection is usually asymptomatic and adult hamsters develop immunity, however, heavy burdens may result in poor weight gain, diarrhoea, abdominal distension, and even obstruction or impaction of the bowel. It is the number of parasites present more than the actual presence of the parasite that is the important factor. If treatment is required, praziquantel 10 mg/kg repeated in 10 days can be given. *This tapeworm is a potential zoonosis*, although human to human transmission is considered the main method of contraction.

Dermatology in hamsters

Just as in more familiar species, a full clinical history and assessment of pattern of clinical signs may give clues as to the underlying cause. As dermatological conditions are often multifactorial, any underlying environmental stressors must be identified and rectified.

Owners will not always notice pruritus, as they may interpret the behaviour as an increase in grooming. Signs of excoriation, or inducement of a scratch itch reflex during gently stroking against the lay of the coat may confirm pruritus. Failure to elicit the reflex does not mean the animal is not pruritic, as their ability to hide disease means that during periods of stress (e.g. the clinical exam), they may mask the itch.

Secondary bacterial infections are common and may complicate the picture especially if the animal is presented late in the course of the disease. Asking if the owner has pictures of when they first noticed a problem may be beneficial.

See Neoplasia section for skin masses.

Scent gland conditions

The glands may become wet and matted during periods of sexual excitement, which may mimic impaction. *Examination of an impression smear will differentiate infected glands from normal secretion*.

Treatment of infected or impacted glands include clipping of the overlying fur, a bacterial swab can be taken for culture and sensitivity, cleaning of the gland with dilute chlorhexidine, and application of F10 wound ointment.

The paired flank scent glands in Syrian hamsters can be the site of neoplasia with male hamsters being overrepresented. Dwarf hamsters are more prone to neoplasia of the ventral scent gland. Surgery may be curative even in cases of malignancy (see Neoplasia section).

Alopecia

Mild, patchy, thinning of the coat is a normal change seen in aged hamsters. Pattern of alopecia, progression, presence, or absence of pruritus, and any other clinical signs may guide further questioning or tests as in more familiar species.

Fur loss over the bridge of the nose is commonly caused by bar biting. *Bar biting is a stereotypical behaviour caused by boredom due to lack of space and lack of enrichment*. Without the owner being willing to increase the size of the enclosure and add in toys (e.g. cardboard tubes, suitable wooden toys, a wheel of suitable size and type) this behaviour will never stop.

Alopecia is commonly associated with hyperadrenocorticism (HAC) (see section on Hyperadrenocorticism) which may be complicated by a flare up of *Demodex* or secondary bacterial infection. Epitheliotropic lymphoma may initially present as alopecia early in the course of the disease.

Alopecia associated with pruritus should increase the index of suspicion for ectoparasites and skin scrapes and tape strip samples should be advised, or selamectin may be applied as a therapeutic trial.

Alopecia associated with crusting without pruritus should increase the index of suspicion of dermatophytosis, but dermatophytosis is uncommon in hamsters.

Cystic ovaries are reported in hamsters and may present with symmetrical, bilateral, non-pruritic alopecia.

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Diagnosis may be achieved via ultrasound of the ovaries. Ovariohysterectomy is the treatment of choice.

Ectoparasites

The most common ectoparasite in hamsters is *Demodex spp.* and is spread from dam to suckling young.

Demodex is found in skin scrapes from normal animals and clinical disease is usually seen in animals that are immunocompromised due to concurrent disease. Two species of *Demodex* are found in hamsters: *D. criceti* is short, fat bodied, lives in the keratin layer, and is usually non-pathogenic; *D. aurati* is cigar shaped, lives in the hair follicles (requires a deep scrape to diagnose, best performed under a quick general anaesthetic) and causes a dry scaly alopecia initially on the dorsum but may spread. Unless secondarily infected, the skin is normally non-pruritic. Selamectin 15–20 mg/kg applied topically twice 10 days apart will treat the mites but *the underlying cause of the immunosuppression requires investigation and treatment*.

Notoedres notoedres (ear mite) and *N. cati* (cat mange mite) are both reported in the hamster. Thick yellow crusts, alopecia, erythema, and pruritus are seen on the ears, nose, feet, and perianally. Female hamsters may only show disease on the pinnae.

Acarus farris (forage mite) is reported in Syrian hamsters and identified on skin scrapes or hair plucks. Although non-parasitic it is thought to cause contact dermatitis due to an allergy to either the mite itself or the droppings. Forage mites have been documented causing skin disease in multiple species.

Diagnosis is via a trichogram in liquid paraffin.

Treatment is both environmental and application of fipronil spray topically. Spray onto a paper towel and wipe onto the hamster rather than spray directly. Environmental control involves freezing all new bedding and food at –18°C for at least 48 h to kill any new mites, and transferring the hamster to a newly cleaned enclosure after treatment.

Ornithonyssus bacoti (tropical rat mite) has been reported in hamsters. They present with poorly demarcated alopecia, erythema, and pruritus over the dorsum. The mites are large enough to be seen with the naked eye as dark brown or black mites and will be seen both on the animal and *in the substrate* (other species of mites seen on hamsters are not visible to the naked eye and would not be in the substrate even if they were!). *O. bacoti is zoonotic* and can be associated with pruritic papules and vesicles on the forehead, limbs, and other areas in humans. This mite does not live on the animal, it lives in the environment, only moving onto the animal to feed. *Eradication may be challenging and requires treatment of the animal and the environment*. One case that full eradication was achieved used two topical treatments on the hamsters of a spot on containing 1% moxidectin and 10% imidacloprid (dose 0.5 mg/kg) 15 days apart and treated the environment with a miticide spray containing 1% piperonyl butoxide, 0.3% permethrin and 0.2% tetramethrin (Neo Foractil, Formevet; Milan, Italy).

Neoplasia in hamsters

Both Syrian and dwarf hamsters can be affected by a variety of tumours. There is an increased frequency of neoplasia in pet dwarf hamsters compared to pet Syrian hamsters. Whereas dwarf hamsters are more likely to present with integumental tumours, Syrians are more likely to present with lymphoma.

Integumental tumours

The median age for integumental tumours is reported to be 12 months. Papilloma and squamous cell carcinoma occur almost solely in the epidermis of the head and the extremities. Russian hamsters have a high prevalence of papilloma of head and especially the pinnae (Figure 5.9).

Trichoepithelioma is considered pathognomonic for an infection with Hamster polyomavirus (HaPyV) in Syrians.

The paired flank scent glands in Syrian hamsters can be the site of neoplasia (often melanomas) with male hamsters being over represented (Figure 5.10 for a normal flank scent gland). Dwarf hamsters are more prone to neoplasia of the ventral scent gland (Figures 5.11a & b). Melanoma of the scent glands is commonly reported (melanoma is also a cutaneous neoplasm) with a 10:1 male:female. Surgery may be curative, although owners should be advised that



Figure 5.9 Russian hamsters have a high prevalence of papilloma of head and especially the pinnae.

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Figure 5.10 A normal flank scent gland in a Syrian hamster. Note the poor-quality coat as this aged animal has not been grooming properly due to OA.

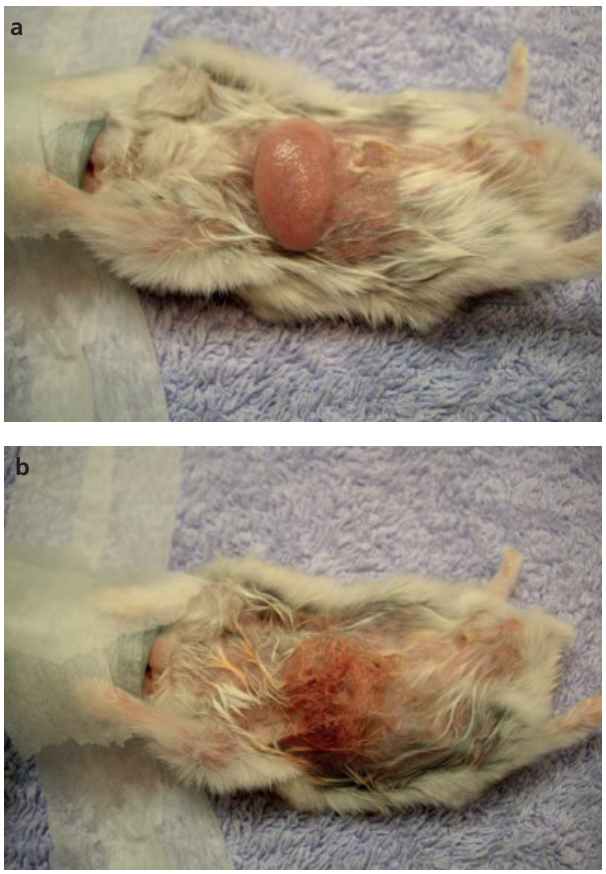


Figure 5.11
a A ventral scent gland tumour in a dwarf hamster being prepared for surgical removal.
b Post-operative image of the hamster in 5.11a.

metastasis of melanoma has been documented, therefore thoracic radiographs should be considered.

Cutaneous mast cell tumours are documented in dwarf hamsters. Cheek pouch neoplasia is seen in both Syrians and dwarfs with mainly fibrosarcoma or fibromyxosarcoma being documented.

Mammary neoplasia

Mammary neoplasia is rare in hamsters. Of the cases documented, dwarf hamsters are overrepresented, as are female hamsters in general. Almost one-half of mammary neoplasia is malignant in one study, and over one-half was malignant in another study. Radiography to check for thoracic spread (challenging to fully rule out due to small size of the thorax therefore good quality images and good positioning vital), and fast surgical intervention may be curative. See Figure 5.12.

Lymphoma

Lymphoma is rare in dwarf hamsters but has been documented. It is *much more commonly diagnosed in Syrian hamsters*. There are three main presentations: cutaneous lymphoma, multicentric lymphoma, and epizootic lymphoma.

Cutaneous lymphoma

Also known as epitheliotropic lymphoma or mycosis fungoides (resembles this condition in humans). Seen in aged (median 19 months old) Syrian hamsters. They present with alopecia (patchy or generalised), pruritus, generalised scaling and crusting and there may be ulcerated nodules or plaques. *Early stages may be mistaken for hyperadrenocorticism*. The skin is often thickened and painful when touched (Figure 5.13). Owing to immunosuppression, secondary bacterial or fungal infections are common and demodicosis may be present. Weight loss may be noted due to reluctance to eat (pain related). Biopsy or fine needle aspirate (FNA) of the skin is diagnostic. The condition often progresses rapidly, and prognosis is grave. Palliative care with prednisolone 0.5–1 mg/kg q12hrs, selamectin 15–20 mg/kg q14 days (for *Demodex*), and antibiosis for secondary skin infection may be attempted but euthanasia should be discussed.

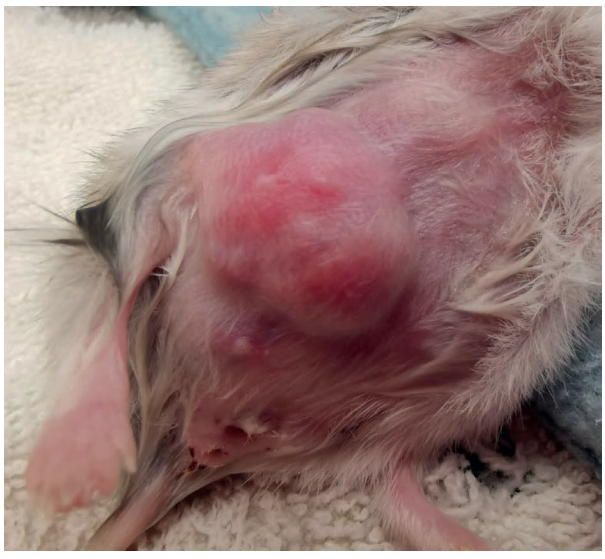


Figure 5.12 Mammary tumour in a dwarf hamster.

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Figure 5.13 Cutaneous lymphoma in a hamster, which may resemble hyperadrenocorticism in the early stages.

Multicentric lymphoma

Sometimes referred to as haematopoietic lymphoma. Seen in aged Syrian hamsters. The clinical signs will vary depending on the organ affected but any combination of anorexia, lethargy, ataxia, dyspnoea, peripheral lymphadenopathy, and palpable abdominal masses can be seen. Singular or multiple masses may be found in the thymus, lymph nodes, liver, spleen, testis, or any other organ. Anecdotal treatment has been attempted modifying chemotherapy protocols from other species. Prednisolone may be given for palliative care along with supportive care if necessary. Prognosis is grave and quality of life should be closely monitored.

Epizootic lymphoma

Epizootic lymphoma is caused by the young, naive hamster contracting hamster polyomavirus.

Hamster polyomavirus in syrians

Hamster polyomavirus (HaPyV) is a naturally occurring, highly transmissible virus that is transmitted horizontally through urine. The virus is associated with development of lymphoid and hair follicle tumours. *There is high morbidity and high mortality associated with infection, with up to 90% of naive hamsters dying 4–30 weeks post-infection.*

There are three main outcomes seen once exposed:

- subclinical infection (often older hamsters and will result in persistent viruria due to the virus persisting in the renal tubules)
- tumours of the hair follicles (trichoepitheliomas are pathognomonic for HaPyV exposure)
- tumours of the haemopoietic system (usually lymphoma).

Trichoepitheliomas are benign; however, they can coalesce. The sheer size and number that may form can become debilitating.

There can be a devastating epizootic lymphoma which will result in clinical signs referable to the affected organ, but many will have nonspecific signs, and the hamster may be thin with palpable abdominal masses. The organs that may be affected in order of descending frequency: mesenteric lymph nodes; liver; small intestine; thymus; kidneys; spleen; and peripheral lymph nodes.

The abdominal masses are soft, pale tan in colour and bulge when sectioned. There is no treatment possible. The virus is markedly resistant to environmental decontamination and a full colony cull would be required to try and eradicate the virus.

Owners will understandably find this devastating when their hamster requires euthanasia due to incurable cancer at such a young age. In my experience owners will often blame themselves and seek reassurance that they have not done something that has caused this. I try to reassure owners that their beloved pet already had the virus prior to purchase but there was just no way to know. Cases seem to present in a cyclical fashion, likely due to resurgence in breeding colonies. The virus is only infective to Syrian hamsters. Contrary to some texts, *I have seen this frequently in pet hamsters*.

Hepatic cysts

Seen in older hamsters (>12 months old), they often present with abdominal distension. There may be dyspnoea due to the pressure the cyst will place upon the diaphragm. The main issue being the sheer size the cyst gains, becomes a space occupying lesion (Figure 5.14). This may result in

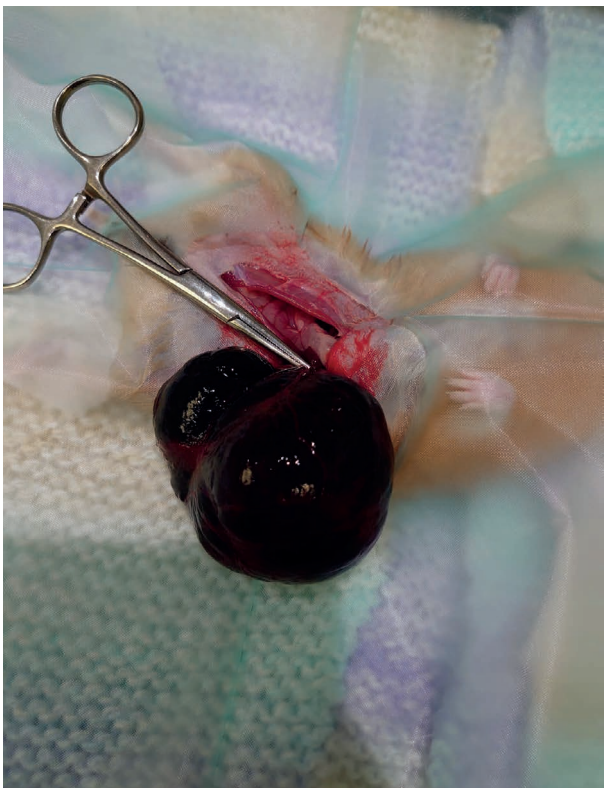


Figure 5.14 Surgical removal of a large hepatic cyst, note the large size. The hamster had respiratory distress due to the pressure on the diaphragm.

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weight loss as the animal will be unable to eat properly (no room for stomach distension), and a decrease in activity. There may be pain associated with the stretching of the abdominal muscles and the owner may note the animal is more aggressive or touch aversive than normal.

Diagnosis is easily achieved via ultrasound of the abdomen (the cysts are fluid filled). In cases of a single (even large) cyst, surgical resection may be possible by removing the affected liver lobe. In cases of multiple cysts or if surgery is declined but the owner would like palliative care, ultrasound guided, percutaneous drainage of cyst under a short general anaesthesia. The fluid will reaccumulate over time. Euthanasia is advised in severe cases.

Adrenal neoplasia

Syrian hamsters are prone to adrenal gland neoplasia. Adrenocortical adenocarcinoma, adrenocortical adenoma, and adrenocortical hyperplasia have all been recorded. Pituitary adenoma stimulating the adrenal glands have also been documented. Hormonally active masses result in hyperadrenocorticism.

Hyperadrenocorticism

Primary hyperadrenocorticism (HAC, also called Cushing's disease) occurs due to neoplasia (adrenocortical adenocarcinoma or adenoma) and is reportedly more common in males and older animals. Secondary HAC occurs due to pituitary neoplasia excreting excess ACTH. Iatrogenic HAC may occur due to excessive glucocorticoid treatment (which would be evident from the clinical history).

Clinical signs

The clinical signs in Syrians are the same seen in canine patients with HAC: polydipsia, polyuria, polyphagia, non-pruritic progressive alopecia (may start as symmetrical flank alopecia), skin thinning, loss of skin elasticity, comedones, and urinary tract infections (Figures 5.15a & b).

Owing to the immunosuppressive effects of the endogenous steroids, if the hamster presents with a pruritic progressive alopecia, demodicosis should be considered. Diagnosis of demodicosis is achieved via a deep skin scrape performed under general anaesthesia. Treatment for demodicosis is selamectin 15–20 mg/kg q14–21 days, with treatment continued past resolution of the clinical signs, in this case the itch.

Diagnosis

Definitive diagnosis may be challenging due to several factors. The volume of blood that can be safely taken prevents ACTH stimulation tests being run. The literature reports that a raise in serum alkaline phosphatase (reference range: 107–127 iu/l) and serum cortisol (reference range: 13.8–27.6 nmol/l) may be seen and aid diagnosis. However, *hamsters produce both serum cortisol and serum corticosterone*





Figure 5.15
a A suspected case of hyperadrenocorticism in a Syrian hamster with bilateral flank alopecia.
b The same Syrian hamster in Figure 5.15a showing abdominal alopecia, and thinning of the abdominal skin making the abdominal musculature more visible.

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therefore diagnosis on serum cortisol alone may not be accurate. Urine cortisol creatinine ratio has also been described as a diagnostic tool, but there is not yet an established reference range in the healthy hamster. The costs involved may be prohibitive for some clients.

As there are very few other clinical presentations (early stage epitheliotropic lymphoma may present as non-pruritic alopecia), it is reasonable, after full frank discussion with the owner, to use a therapeutic trial as a means of definitive diagnosis.

Treatment

There are two drug options for treatment in the literature. *The owners should be advised to wear gloves for administering the medication*. A compounding company should be used to source the medication. Owners should be advised that in cases of secondary HAC, treatment will not prevent growth of the pituitary neoplasia. Once neurological signs occur, euthanasia is necessary.

- Metyrapone 8 mg orally, q24hrs for a 30-day course was successful in one hamster, achieving complete regrowth of hair after 12 weeks.
- Trilostane 2.5 mg orally, q24hrs, resulted in complete resolution of most clinical signs within 6 weeks.

Reproductive tract neoplasia

Leiomyoma, leiomyosarcoma, endometrial polyps, uterine adenocarcinoma, and ovarian tumours (granulosa cell and theca cell tumours) have all been documented in Syrians and dwarf hamsters.

Clinical signs associated with neoplasia of the reproductive tract include anorexia, lethargy, abdominal distension, and vaginal discharge. Ultrasound of the abdomen may help with diagnosis. Surgery may be curative although the owner should be advised that we just do not yet know the rate of metastasis for these tumour types in hamsters yet.

Thyroid neoplasia

Thyroid neoplasia is rare in hamsters and all of the documented cases have been carcinomas.

Renal neoplasia

Renal neoplasia is uncommon and has only been documented in aged animals over 22 months of age. Both renal adenocarcinoma and nephroblastoma have been reported.

Pyometra

Female Syrian hamsters may show a copious, creamy coloured, vaginal discharge just after ovulation (day 2 of oestrus cycle) (see Figure 5.4). This post-ovulatory discharge is extremely 'stringy' and touching it will result in it stretching between the probe and the vagina. Visually it can be confused with a pyometra, therefore it is important that further diagnostics are undertaken before diagnosing a pyometra in a Syrian hamster, such as *making a smear, staining, and examining the discharge microscopically to confirm a purulent discharge*. Other clinical signs that may be present with pyometra include swollen vulva, malodorous discharge (abdominal palpation may result in further discharge), a reduction in food intake, polydipsia, and an increase in body weight (due to fluid accumulation in the uterus).

Diagnosis is achieved by clinical signs, examination of a smear of the discharge and ultrasound of the reproductive tract. Ultrasound will show the familiar pattern of dilated fluid filled uterine horns and body, the endometrium may be thickened (2 mm is upper end of reference range) and may be cystic. Once confirmed, owners should be counselled that without prompt treatment, the condition will become fatal.

The definitive treatment for a pyometra is an ovariohysterectomy. In cases where the owner declines surgery or immediate surgery is deemed too risky, the use of aglepristone (Alizin, Virbac) has been described in the literature as a successful treatment (Pisu *et al.*, 2012). As there is no recognised protocol yet for use in hamsters, an empiric dose of 20 mg/kg SC on days 1, 2, 8, and 15. Marbofloxacin was also administered as a broad-spectrum antibiotic, 2 mg/kg q24hrs for 2 weeks to resolve the infection and prevent an ascending uterine infection via the open cervix. The pyometra fully resolved and the hamster displayed normal oestrus behaviour with no recurrence of the pyometra.

Respiratory disease

Compared to other rodents, respiratory disease (RD) is uncommon in hamsters.

Allergic rhinitis with associated upper respiratory signs of sneezing may respond to husbandry changes (e.g. change of substrate, stop aerosol use, stop smoking, or use air purifier) and an antihistamine such as chlorpheniramine maleate 0.6 mg/kg q12–24hrs or diphenhydramine 1–2 mg/kg q12hrs.

The most common bacterial infection associated with RD in hamsters is reportedly Streptococcus pneumoniae, other *Streptococcus* spp,, and *Pasteurella pneumotropica*.

Diagnosis is via clinical signs, although radiography is strongly advised as cardiac disease is an important differential diagnosis in hamsters that present with RD.

Treatment

Antimicrobial therapy is limited in hamsters due to the sensitivity of their gut microbiome. Enrofloxacin 10–20 mg/kg q12hrs or trimethoprim sulfamethoxazole 30 mg/kg q12hrs have good activity against the commonly isolated bacteria.

RD is uncomfortable at best, painful at worst therefore analgesia should be given. Meloxicam 0.6–1 mg/kg q12hrs is often readily taken due to the sweet nature of the carrier.

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If mucous is suspected to be a component of the condition, then administration of a mucolytic such as bromhexine 0.3 mg per hamster q12hrs onto favourite food or syringed via water can be given. It is important to advise owners that when the mucous breaks down, initially any coughing or sneezing may get worse as the animal works to clear the mucous. Without managing the owner's expectations, we increase their stress levels which in turn will result in increases of our stress levels.

Treatment may also be given via nebulisation (see Chapter 4 for more information and Table 5.6 for doses).

Cardiac disease

Cardiac disease is likely underdiagnosed in hamsters as it will often present with respiratory signs (if we think about it, so do Cavalier King Charles Spaniels!). Dilated cardiomyopathy, hypertrophic cardiomyopathy, arteriosclerosis, and atrial thrombosis are all recognised in Syrian hamsters and clinical signs develop when congestive heart failure develops.

Unfortunately, *because of their mainly nocturnal/crepuscular activity levels, owners may not recognise clinical signs until they are advanced*. Prognosis is guarded as hamsters tend to be presented late in the course of heart failure. *Any hamster presenting with respiratory signs should be radiographed to assess the lung field and cardiac silhouette*.

Dilated cardiomyopathy

Dilated cardiomyopathy is the most common cardiomyopathy in hamsters and should be suspected in aged hamsters (>1.5 years old) that present the following clinical signs: lethargy, anorexia, dyspnoea, tachypnoea, generalised oedema, and cyanosis. Diagnosis is achieved via clinical signs, radiography, and ultrasound.

Table 5.6 Nebulisation doses.

Drug	Dose (most nebulisers will hold 10 ml)
Saline	10 ml good for hydration of respiratory tract
Acetylcysteine (mucolytic)	10–20 mg per 1 ml saline (higher doses may be associated with bronchospasm)
Bromhexine (mucolytic)	0.15 mg per 1 ml saline
F10	1:250 or ready to use (already prediluted)
Aminophylline	3 mg per 1 ml saline
Amikacin	5 mg per 1 ml saline
Doxycycline	13 mg per 1 ml saline
Enrofloxacin	10 mg per 1 ml saline
Gentamicin	5 mg per 1 ml saline

Treatment

The mainstay of treatment in hamsters is very similar to more familiar species: drugs to improve cardiac function, drugs to reduce oedema, and drugs to prevent thrombus formation.

Pimobendan 0.6 mg/kg q12hrs, furosemide 1–4 mg/kg q8–24hrs, enalapril 0.5–1 mg/kg q24hrs, and prophylactic anticoagulants. Oxygen therapy may be required until oedema is under control.

Atrial thrombus

Atrial thrombosis is reportedly common in hamsters over a year of age both as a primary condition and secondary to heart failure. It is seen at a younger age in female (13.5 months) than male (21.5 months) hamsters. Sichuk *et al*. (1965) described that castrating male hamsters was linked to an increase in occurrence of atrial thrombosis.

Clinical signs include acute dyspnoea, cyanosis, cold extremities, and sudden death. Prognosis is grave and post mortem will reveal pale adherent plaques of thrombi in the left atrium or auricle.

Renal disease

Renal disease is common in aged hamsters with a prevalence of 47.8% (Reavill & Imai, 2020). Chronic nephropathy (arteriolar nephrosclerosis) is the most common condition, but chronic pyelonephritis and renal amyloidosis are also common causes in hamsters over 1.5 years old.

Arteriolar nephrosclerosis in Syrian hamsters is associated with ageing and is more frequently seen in female hamsters, often at a younger age than in male hamsters. Risk factors for developing arteriolar nephrosclerosis are thought to be excess dietary protein, underlying chronic viral infection, and renovascular hypertension. May also be seen concurrently with renal amyloidosis.

Amyloidosis is considered a common geriatric disease in Syrian hamsters, and may be associated with nephrotic syndrome, but the exact pathogenesis is poorly understood. Amyloid is deposited in the kidney, liver, spleen, and adrenals. There is an increased frequency in female hamsters.

Clinical signs

Clinical signs will vary with the severity of the disease (much like in more familiar species) but will include polyuria, polydipsia, anorexia, weight loss, lethargy, dehydration, poor coat, abdominal pain, abdominal distension (ascites), and dyspnoea (due to ascites).

Diagnosis

Diagnosis is via clinical signs, urinalysis, biochemistry, and radiographs to check for uroliths. Urine can be collected by placing the hamster in a clean plastic tub (they often pass urine spontaneously likely due to stress) or by very gentle manipulation of the bladder.

Table 5.7 Hamster urinalysis normal

Colour	Variable depending on diet
Protein	Moderate amounts present
Glucose	Trace amounts can be present due to stress
pH	6.9–9
Specific gravity	1.034–1.060
Sediment examination	Calcium carbonate and triple-phosphate crystals

Urinalysis may show severe proteinuria, sediment examination may show casts, crystals, inflammatory or neoplastic cells and bacteria. Isosthenuria may be present (reference range for urinary specific gravity in a hamster is 1.034–1.060). Biochemical changes may include hypoalbuminaemia, hyperlipidaemia, increased urea, increased creatinine, increased phosphate, lowered or raised calcium, and lowered or raised potassium. (See Table 5.7 Normal urinalysis.)

Treatment

Treatment is mainly palliative to improve quality of life and delay progression of disease. Fluids should be given to promote diuresis. If hyperphosphatemia is present phosphate binders can be given (aluminium hydroxide 30–90 mg/kg/day divided and administered with food), a potassium supplement can be given if low potassium is present.

Owing to the high risk of increased gastric acidity, famotidine 0.5 mg/kg q24hrs or ranitidine 1–2 mg/kg q12hrs should be administered. As most vitamins are water soluble and a polyuria is often present, administration of a multivitamin may be considered.

If dyspnoea is present due to ascites, therapeutic abdominocentesis can be performed to provide temporary relief.

In cases of suppurative pyelonephritis/nephritis and cystitis, prolonged antibiotic treatment for at least 4–6 weeks is required. Ideally antibiotic therapy should be based on culture and sensitivity but for empirical treatment or if culture is negative despite high clinical suspicion of bacterial involvement, then *use of antibiotics that are effective against Gram negative bacteria and are renally excreted (will reach high tissue concentrations in target areas)* should be used such as: trimethoprim sulfamethoxazole 30 mg/kg q12hrs or enrofloxacin 10–20 mg/kg q12hrs.

The angiotensin-converting enzyme (ACE) inhibitor enalapril (0.5–1 mg/kg q24hrs) as given in rats may help.

Dietary management includes reducing sources of protein and administering omega 3 fatty acids. Owners should be counselled to monitor quality of life parameters.

Euthanasia

End of life conversations can be a difficult area to broach with some clients. Occasionally owners will wait for us to broach the topic as they are 'worried' if they do so we will think they are not dedicated enough. *Reassuring clients that sometimes we need to love our pets enough to let them go when the time is right can be invaluable.*

Many owners will want to stay with their hamster during this procedure and the process should be fully explained including possible events such as sudden jumping, or gasping. I administer *a deep sedation* (e.g. medetomidine/butorphanol/ACP injected SC), once the pet has fully relaxed, I will use the kidney by choice, to inject the pentobarbitone, to allow the owners to stroke their pet's head. *Intra-abdominal or intra-cardiac injections should never be administered in a conscious hamster as it is considered extremely painful.*

Many owners will opt for private cremation with return of their pet's ashes and all options that your practice offers for dogs and cats should also be made available to your hamster clients. I offer a lock of fur, footprints (coloured ink pad from craft shop and a card). I send sympathy cards if I know the patient and owner well.

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6 Mongolian gerbils

Mongolian gerbils (*Meriones unguiculatus*) are social, burrowing, diurnal rodents, evolved to live in the semi-arid, unforested, open, sandy, desert grasslands (steppe regions) of northern China, Mongolia, and Russia. They are occasionally called jirds, clawed jirds, sand rats or antelope rats. During the hot summer months, they may change their activity levels to be primarily nocturnal to escape the high temperatures. The temperature in their natural habitats vary from –20°C (–4°F) to +20°C (68°F), so they are tolerant to a wide variation in temperature by utilising their burrows to escape the extremes of the temperatures. The relative humidity is low.

Gerbils occupy areas with short vegetation and dry, loose, sandy soil. They are a social species, forming family groups of between 2 to 17 individuals in the wild. The whole colony shares a 'mutual scent' which allows colony members to recognise each other. Family groups generally will consist of one or two related males and a harem of females.

> They have large home territories of 325 m^2 to 1,550 m^2 with the larger groups occupying the larger territories. Male gerbils were generally found to be more active than the females (Agren *et al.*, 1989).

Wild diet

They preferentially feed on the seeds of broad leafed, flowering plants (dicots) and some foliage (leaves and stems) but will feed on agricultural grains. Gerbils communally gather and hoard food in the burrows before winter and will behave cooperatively to defend territory, dig burrows, and raise young. It is fair to say that gerbils have a complex social organisation. Thiessen and Maxwell (1979) found that complex tunnel systems were dug, with multiple entrances and exits, the average tunnel length was 40 cm and the maximum tunnel length was 130 cm, were functionally compartmentalised and the floors of the tunnels were heavily lined with gravel.

The average lifespan quoted varies between sources ranging 2–5 years.

Companionship

Gerbils are highly social and have an innate requirement for conspecific company. Even the most dedicated owner cannot fulfil the behavioural repertoire of another gerbil.

Single sex groups or mixed sex, neutered (usually males are neutered) can live peaceably provided they have been introduced before 8 weeks of age.

Enclosure and enrichment

Inadequate enclosure design, inadequate substrate depth and inadequate environmental enrichment will result in stereotypical behaviours such as compulsive digging and bar chewing. *Development of stereotypical behaviour can be seen as a poor welfare indicator*.

The recommended temperature range for captive gerbils is 18–22°C (50–68°F). They evolved to live in areas with relative humidity of less than 50% and higher levels of humidity may lead to the fur coat becoming matted and nasal dermatitis developing.

Positioning of the enclosure within the house is important. Like most rodents, gerbils hear into the ultrasonic range of frequencies and can find ultrasonic sounds emitted by televisions, stereo systems, or refrigerators distressing. Likewise, vibrations caused by washing machines or tumble driers may also result in chronic stress.

Gerbils have large home territories (see above) and are extremely active animals, who may travel several miles foraging in the wild. *Most commercially available gerbil cages are far too small*.

Therefore, we should be recommending a floor surface area of a minimum 1 m^2 when choosing a cage. A cage 80 cm × 50 cm only has 0.4 m^2 of floor surface area, whereas a cage 120 cm × 90 cm has 1.08 m^2 of floor surface area. Gerbilarium's can be made from fish tanks with a wire cage attached to the top. *It is important to position the gerbilarium out of direct sunlight*. Multiple levels can be added to the wire cage, increasing enrichment opportunities.

They are burrowers by nature and so burrowing is an instinctive, innate requirement. A deep substrate of at least 40 cm (ideally 70 cm) should be provided to allow this natural behaviour. *Insufficient substrate depth is associated with*

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an increase in stereotypical behaviour (e.g. frantic digging in the corner), aggression, and reduced food intake.

Various substrates are available. The ideal one is absorbent, dust free and free from perfumes. Shavings may be suitable for some gerbils, whereas others may develop allergic rhinitis. Paper-based bedding with dust extracted is available. A mixture of soil, sand and chopped straw helps maintain tunnel integrity but may stain the fur of light-coloured gerbils. Allowing the gerbil to shred cardboard and or toilet paper to mix through the substrate provided environmental enrichment while potentially making the substrate more suitable for building tunnels. Giving access to hay may be used by the gerbils to line the tunnels or chambers as nesting material.

Commercially there is a huge range of toys aimed at rodents. It is important to provide safe wooden toys to provide ample gnawing opportunity which is important for maintaining the length of their incisors while also satisfying their innate requirement to gnaw. Untreated, natural branches from fruit trees (not cherry) can be given to maintain incisors.

Opaque, plastic tubes can be buried in the substrate to provide a fake tunnel which has been shown to reduce stress, but should be monitored for signs of damage caused by gnawing, which may result in sharp edges.

Solid wheels can be provided for running. When rodent wheels have been placed outdoors, various wild rodent species have chosen to utilise it, spending time running! This demonstrates that it is indeed a pleasurable or at least a valued resource. *To prevent painful spinal conditions, it is important to provide a large enough wheel that they do not have to bend their back while running. They should be able to run with a straight back and without having to lift their head*. Most gerbils can achieve this with a 25–30 cm diameter wheel.

A nest box with soft nesting material should be provided. *Cotton wool type nesting material should be avoided as it can become tangled around toes/limbs leading to avascular necrosis*. Using kitchen or toilet roll paper as nesting material gives the added enrichment of allowing the gerbils to shred the paper to their preferred length and width for nesting.

Access to a sand bath is required a few times weekly to maintain coat quality. Chinchilla sand in a wide dish can be offered for 20 minutes at a time.

Gerbils produce dry faeces and small volumes of very concentrated urine (desert species adapted to conserve water) so cleaning is advised every couple of weeks, but it is important to leave some soiled bedding behind to ensure the familiar smell is maintained for the pet which reduces their stress. As they often toilet in the same area, placing a jar on its side containing faeces and bedding with urine can encourage toilet training which will allow for spot cleaning rather than regular full substrate changes.

Diet

Gerbils are omnivores but they do not tend to exhibit coprophagy unless the diet is insufficient. Muesli type diets lead to selected feeding and nutritional imbalances; therefore, a complete pelleted diet should be given. Obesity seems to be more common in gerbils fed on muesli type diets.

Gerbils have high levels of plasma lipase and cholesterol, and require a high protein (18–20%), low fat (<4%) pelleted diet to prevent problems such as hepatic lipidosis and gallstones. Oxbow Animal Health Essentials, Burgess and Supreme Petfood Science Selective are some options for pelleted complete gerbil diets. Average food consumption is 8–10 g/100 g/day, which should be scattered on the substrate or mixed into a small amount of hay to provide foraging enrichment.

This can be supplemented, for enrichment, with small amounts of fresh vegetables (e.g. broccoli, herbs, carrot, sweet pepper) and fresh non-citrus fruits (e.g. apples, pears, and berries) several times weekly. Live foods such as mealworms or crickets may be offered once weekly but it is important that owners feed the insects a balanced diet or they will be of poor nutritional value. For owners that cannot stand the thought of feeding live food, small pieces of cooked lean meat, or small pieces of boiled egg can be offered. Hay can be offered to increase fibre intake. *Introduce any new food type slowly as diarrhoea can result from sudden dietary changes*.

Pumpkin seeds can be used as treats rather than more fat laden seeds such as sunflower hearts. *Gerbils offered a diet high in sunflower seeds (high in fat low in calcium) will preferentially consume the sunflower seeds, making them prone to obesity, hypercholesterolemia, and nutritional osteodystrophy which in advanced cases may lead to pathological fractures*.

Sugary treats should be avoided as diets high in sugar lead to periodontal disease, caries, secondary dental abscesses, diabetes mellitus, and diarrhoea.

Food may be cached, which can make it *difficult for owners to establish how much the gerbils are actually consuming*. Hoards should be regularly cleared to prevent spoilage of food, which may be challenging as an underground chamber may be utilised.

Water

Clean water should be provided by sipper bottles. The average water consumption is low, only 2–4 ml/100 g body weight/day as they are uniquely adapted to conserve water. Water is absorbed from the fresh components of the diet. *Owners should be advised if they see their gerbils drinking more frequently, a veterinary visit is recommended* as gerbils are prone to renal disease.

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Sexing gerbils

Sexing gerbils is performed by looking at the genital anal distance.

Male

Male gerbils have a much longer distance between the genital opening and the anus compared to the female. Mature male gerbils also have large, testicles which are situated caudally to the prepuce and the scrotum is pigmented (Figure 6.1).

Female

Female gerbils have four pairs of nipples (two thoracic, two inguinal). The female gerbil has three urogenital openings close together. The urethral papillae, the vulvar opening (covered with a membrane unless in oestrus) and then the anus (Figure 6.2) *The urethral papillae is well developed in female gerbils and may be mistaken for a prepuce.*

Preventative health care

There are no vaccinations available for gerbils and routine worming or ectoparasite control is unnecessary.

Hospitalisation

They are a prey species, and so are extremely sensitive to stress caused by the sight, sound, and smell of predators. As the bond is scent based, cage mates should be admitted both to maintain the bond and to reduce stress. The cage set up in the hospital is the same as advised for rats, but a deeper substrate is recommended.

Venipuncture sites

I anaesthetise gerbils with isoflurane for venipuncture as they find restraint for venipuncture incredibly stressful.



Figure 6.1 A male gerbil. Note the prominent testis, longer genital–anal distance, and the pigmented scrotum.



Figure 6.2 A female gerbil with a short genital–anal distance and a well-developed urethral papillae which may be mistaken for a prepuce.

Using EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) makes venipuncture much easier. Ensure the room temperature is approximately 25°C (77°F) as cold temperatures result in venoconstriction.

My vein of choice is the lateral saphenous vein or gingival vein (see Chapter 5 venipuncture) for blood sampling. I often use only a 25g needle and allow the blood to drip directly into the blood collection tube if using the lateral saphenous vein. *Having your nurse massage the vein or 'pump' the vein by very slowly raising and releasing while you flex and unflex the foot allows good flow of the blood.* Alternative sites for venipuncture are the jugular vein, femoral vein, ventral tail artery, or dorsal tail vein. The jugular runs in a shallow groove running from the caudal border of the mandible to the thoracic inlet and is entered via a blind stick.

> Gerbils have very thin skin on their tail which is prone to degloving so I tend to only use the tail vein as a last option.

I apply approximately 1 mm thick layer of EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) enough so there is a noticeably white patch, then I get the rest of my equipment organised. in my experience, EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) takes around five minutes to cause venodilation, some local anaesthesia and does not require a dressing to work. *Pick your needle size judged on the size of the vein, not the size of your patient!*

The maximum volume of blood that can be safely taken is 1% of their bodyweight. Therefore, in a 100 g gerbil we could safely take 1 ml. Most biochemistry machines can run a profile on 0.3 ml of blood.

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Biochemistry and haematology profiles *can be run 'in-house' using our usual mammalian profiles* or sent to an external laboratory. Both options have pros and cons. The main pro for running in-house is that you obtain your results quickly whereas the main con is that you have to interpret whereas many labs will provide interpretation upon request.

Haematology

Gerbils red blood cells have a short circulating life of only approximately 10 days. This is why there is often basophilic stippling when a smear is examined.

Gerbil haematology exhibits sexual dimorphism with male gerbils having a high red blood cell volume, higher haemoglobin levels, high total white blood cell counts and high lymphocyte counts.

The main leucocyte in gerbils is the lymphocyte which accounts for approximately 75% of the leukocytes in the peripheral blood, there is a lymphocyte:neutrophil of 3:1. See Tables 6.1 and 6.2 for reference ranges for biochemistry and haematology. Acute stress does not affect the neutrophil/lymphocyte ratio but chronic stress results in an increased ratio due to a relative neutrophilia and a lymphopenia, each still being within recognised reference ranges.

Dehydration

Dehydration can be assessed in the same way it is for rats, but due to gerbils being from an arid environment and able to conserve water, *their maintenance fluid therapy volume is lower than rats, and 40–70 ml/kg/day is recommended*. Fluids should be warmed to body temperature (38°C (100.4°F)) prior to administration. *I do not agree with subcutaneous fluid administration*. It is *incredibly painful* and dehydrated patients will not absorb fluids via this route as efficiently.

An intravenous catheter may be placed in the lateral saphenous vein but it is challenging. Intraosseous (IO) fluid administration is much easier. IO administration of fluids should be given to any gerbil considered to be >5% dehydrated.

IO catheter placement

My preferred IO site is the proximal tibia, as the tibial crest is easy to find. General anaesthesia (if not moribund) is required as placement is considered painful. After surgical preparation of the site, instil lidocaine (2 mg/kg) at the level of the periosteum. Using a 25g needle (surgical steel suture material (sterile) as a stylet, or replace the needle) is placed in a normograde fashion, a radiograph should be taken to ensure correct placement as inadvertent puncture of both cortices is a common complication in the tiny species. *IO placement is contraindicated in any patients that have sepsis, osteomyelitis, or metabolic bone disease*.

IO catheters can be connected to syringe drivers or boluses can be administered every 30–60 minutes. The bag of fluids, or syringes of fluids, can be maintained at the correct temperature using a baby bottle warmer. *Ongoing analgesia is important, IO catheter flushing and administration of fluids is painful in humans therefore should be considered painful in other species.*

Hydration status should be assessed throughout the day which enables you to alter the rate and volume of fluids therapy as required. My intravenous fluid of choice in most cases is saline.

Assisted feeding

The term assisted feeding is preferable to force feeding when discussing with owners.

There are high-quality, purpose made foods available such as: Harrison's recovery or EmerAid Omnivore.

Table 6.1 Gerbil biochemistry table.

BIOCHEMISTRY (Heatley & Russell, 2020)	REFERENCE RANGE (UK units)	REFERENCE RANGE (US units)
ALKP	12–37 IU/l	12–37 U/L
GLU	2.8–7.5 mmol/l	50.45–135.12 mg/dl
CHOL	2.34–3.9 mmol/l	90.35–150.58 mg/dl
TBIL	3–10 µmol/l	175.4–584.67 mg/dl
UREA	6.1–9.6 mmol/l	36.64–57.66 mg/dl
CREA	53–124 µmol/l	0.6–1.4 mg/dl
TP	43–125 g/l	4.3–12.5 g/dl
ALB	18–55 g/l	1.8–5.5 g/dl
GLOB	12–60 g/l	1.2–6.0 g/dl
CA	0.93–1.55 mmol/l	3.7–6.2 mg/dl
PHOS	1.2–2.26 mmol/l	3.72–7 mg/dl
K	3.3–6.3 mmol/l	3.3–6.3 mEq/l
Na	141–172 mmol/l	141–172 mEq/l

Table 6.2 Gerbil haematology table.

HAEMATOLOGY (Heatley & Russell, 2020)	UNITS	REFERENCE RANGE
Red blood cells	10^{12}/l	7.0–10.0
Packed cell volume	%	35–45
Haemoglobin	mmol/l (g/l)	7.5–10.49 (121–169)
Mean corpuscular volume		46.6–60
Mean corpuscular haemoglobin concentration		30.6–33.3
Mean corpuscular haemoglobin		16.1–19.4
Platelets	10^{9}/l	400–600
White blood cells	10^{9}/l	4.3–21.6
Differential blood count (relative values)		
Monocytes	%	0.0–9.0
Lymphocytes	%	32.0–97.0
Neutrophils	%	3.0–41.0
Eosinophils	%	0.0–4.0
Basophils	%	0.0–2.0

Herbivore recovery food may also be used. The recommended volume is 3 ml/feed q4hrs. Some gerbils will require wrapping in a towel (burrito technique) for assisted feeding. Some individuals will tolerate smaller, more frequent feeds better.

Drugs contraindicated in gerbils

If in doubt, always check the formulary as it will often tell you whether it is safe to use a drug in any given species. My favourite formulary is James Carpenter's *Exotic Animal Formulary* now on its sixth edition (Carpenter & Marion, 2022).

Gerbils are also *particularly prone to developing fatal clostridial enterotoxaemia following oral administration of the following antibiotics, and they should never be administered*: penicillins (including ampicillin, amoxicillin), cephalosporins, clindamycin, erythromycin, lincomycin, vancomycin, dihydrostreptomycin, streptomycin, bacitracin, and oral gentamicin.

Prednisolone should be avoided or used with extreme care due to the immunosuppressive side effect in this species.

Acepromazine has been reported to induce seizures in gerbils, and so is not recommended to use in this species (Harkness & Wagner, 1983).

Species-specific anatomy/behaviour

Gerbils are obligate nasal breathers.

They are myomorphs with elodont–hypsodont incisors (elodont: continuously growing throughout life and hypsodont: long anatomical crown compared to the root). They have two incisors in the maxilla and two in the mandible. The lower incisors are much longer than the upper incisors. The incisors usually have a yellow orange colouration due to the iron deposition within the enamel.

The cheek teeth are anelodont (anelodont: limited growth period therefore truly rooted).

Dental formula: $2 \times (\text{I } 1/1; \text{C } 0/0; \text{P } 0/0; \text{M } 3/3) = 16$.

Gerbils produce 'red tears' (porphyrin from the Harderian gland) which can be excessive during times of stress or disease. *Reduction in grooming behaviour (common with pain or disease) may lead to accumulation of the secretion at the nares rather than the eyes leading some owners to misinterpret this as their gerbil having a nose bleed*. Methods of differentiating between porphyrin and blood include UV light (porphyrin fluoresces) and cytological examination.

The Harderian gland secretion contains lipids, protein, and porphyrin. *The secretion is important for ocular lubrication, a source of pheromones (helps with individual identification) and as a method of thermoregulation*. During cold weather, the production of the secretions is increased. The secretions are then mixed with saliva and spread over the coat during grooming. This may provide improved protection from wetting (lipid) and increased heat absorption by darkening of the fur. If the gerbil is too hot, the production of secretion is reduced and the gerbil will use saliva to groom over the coat, thereby removing the lipid and providing evaporative cooling.

Muscular hindlimbs which allow jumping proportionately long distances to their size. Gerbils may rest standing on their hind legs and will thud their hind feet as an alarm if fearful.

They have large tympanic bullae and can hear into the ultrasonic range.

The right lung has four lobes, the left lung has three lobes.

Gerbils have a unique water metabolism, resulting in them requiring relatively little water to function and they can absorb sufficient water from their diet, provided fresh plant material is given. The kidney has a high percentage (96%) of long loop nephrons allowing effective concentration of their urine. Their digestive system is very efficient at absorbing water. Fresh water should always be offered.

The thymus is large compared to other rodents and persists into adulthood, intrathoracic, cranial to the heart.

The adrenal glands are large, compared to body weight they are approximately three times the size of rat adrenal glands. It is believed that the adrenal gland also has a role in water conservation in the gerbil.

The tight cardiac stomach sphincter prohibits vomiting or eructation. The gestation period is 24–26 days and the young are altricial.

Both sexes have a ventral scent gland. It is small in female gerbils but is obvious, large, oval in shape, and orange/brown in males. *Owing to the midline, ventral position of the gland, entry to the abdomen should be via a paramedian ventral incision to avoid damaging the scent gland*.

Using a towel or a blanket on the table to reduce the slippery nature of the surface makes gerbils feel safer and they are less likely to struggle, although I tend to perform most of my clinical exams with them either in my arms or on my knee.

Males have open inguinal canals. Failure to appreciate this and take steps to close the canal during castration can lead to inguinal herniation of abdominal organs.

The skin on the tail is loosely attached compared to other rodents. While this potentially allows escape from predators in the wild, in captivity it can lead to degloving of the tail if grasped, necessitating tail amputation.

Gerbils hide illness and pain.

Pain in gerbils

Due to their tendency to spend a lot of time in their burrows, some owners may not notice signs of illness until it is advanced. *Any change in their normal behaviour is likely to be significant, no matter how subtle, indicating: stress, pain, or illness*.

One of our jobs as veterinary professionals is to *actively look for pain*. In Chapter one we discussed that all vertebrates have the same anatomical and physiological pain pathways.

If we accept that animals and people share similar mechanisms of pain and analgesic action then we should realise that the variation of analgesic efficacy in different individual people means that it is highly likely that a similar variation will be seen in animals – further reinforcing the need for regular pain assessments.

In my experience, healthy gerbils will have wide opened round eyes, sleek coats and are inquisitive. *Gerbils in pain are more likely to appear to be squinting, have unkempt coats and are more likely to hide*. I am aware that not all gerbils in pain will exhibit this and also that it may well vary with severity of pain. The stress of the journey will likely also cause pain to be masked. *I tend to err on the side of caution, and administer analgesia in ill gerbils. If the same condition or procedure is considered painful in another species, it will be painful in gerbils*. Gerbils in general rarely bite but if they are in pain, they are much more likely to bite.

Unfortunately, there is a lack of publications on efficacy and analgesic doses in gerbils necessitating extrapolation of doses used in other small mammals. I tend to use the lower end of the rat doses but will regularly (first recheck is one week) reassess for adequate pain control. I will increase the doses if necessary (See Chapter 4 Pain in Rats and Pain Management in Rats).

Clinical exam

See Table 6.3 for biological data.

To catch the gerbil, I will either use a hide to guide the gerbil into or I will cup my hands around the gerbil to lift. I will ensure I have a face cloth or dish towel available for handling if necessary. Never catch a gerbil by its tail as degloving of the tail is highly likely.

This is performed in a similar fashion to rats, with three notable differences. The ventral scent gland should always be examined (especially important in male gerbils as they are over represented for developing infections, impactions, and neoplasia of the gland), the tail is not manipulated in the same way as it is not partially prehensile in gerbils (and is prone to degloving) and the mandibular symphysis is calcified therefore the lower incisors are rigid.

General anaesthesia

Gerbils cannot vomit and therefore they do not need to be starved before an anaesthetic. Owners are used to starving themselves or their dogs and cats prior to surgery, so we need to be clear when we book gerbils in for surgery that they should not be starved. Advise owners to bring a 'packed lunch' of favourite and familiar foods.

Table 6.3 Biological data.

Parameter	Reference Range
Average Longevity	3–4 years
Respiratory Rate	70–130 bpm
Heart Rate	260–450 bpm
Body Temperature	38°C (100.4°F)
Maintenance Fluid Therapy	40–70ml/kg/day
Sexual Maturity	8–10 weeks
Gestation	24–26d

Bonded cage mates should always be admitted, not only will this *reduce stress during the hospitalisation period, help protect the bond between conspecifics, it also has been shown to reduce healing times and increase coping ability for aversive or painful experiences* (Denommé & Mason, 2022).

Allow gerbils at least an hour to acclimatise to the ward after travelling to the clinic to reduce stress levels prior to induction.

With only a few exceptions (e.g. bloat), ill gerbils should be fully stabilised (rehydrated, passing faeces and starting to eat) before an anaesthetic should be administered.

Pre-anaesthetic blood tests

Due to the small volume of blood that can be safely taken from gerbils, I rarely perform pre-anaesthetic blood tests. *All surgical procedures will result in some blood loss, taking a blood sampling prior to surgery may overwhelm the animal's ability to withstand the blood loss during surgery* (see Tables 6.1 and 6.2 for reference ranges).

Local anaesthesia for muscle splash blocks or infiltration

Combining the drugs allows for greater effect with a lower recommended dose. Lidocaine has faster onset but a shorter duration of action (60–120 minutes) whereas bupivacaine has a slower onset of action but a longer duration (180–480 minutes). Mixing 4 mg/kg of lidocaine with 2 mg/kg of bupivacaine in a syringe then diluting the mixture with saline to increase the volume of the solution and allow easier splash blocking (exactly what it sounds like). Drip the solution onto the wound in the muscles, or infiltrate an area. Pulling up the required dose and pre-diluting prevents accidental overdose.

Induction, maintenance, monitoring, theatre temperature, positioning, surgical preparation, and recovery is performed similarly to hamsters (see Chapter 5).

Specific conditions in gerbils

Compared to most of the species kept as pets, *gerbils are considered medically robust*.

Respiratory disease

Respiratory disease (RD) is uncommon in gerbils and can progress to inner ear infections with head tilt and circling developing. Upper RD may present as conjunctivitis and sinusitis and is mainly caused by *Streptococcus* spp. Lower respiratory infections can be caused by *Klebsiella pneumoniae*, *Pasteurella pneumotropica*, *Streptococcus pneumoniae*, and *Mycoplasma pulmonis*.

Clinical signs are as expected and as seen in more familiar species.

Diagnosis is via clinical signs, radiographs, and a nasal flush can be used for culture and sensitivity.

Treatment includes: antibiotics (ideally based on culture and sensitivity) although doxycycline 5 mg/kg q12hrs, trimethoprim sulfadiazine 30 mg/kg q12hrs or 50–100 mg/kg q24hrs or enrofloxacin 10–20 mg/kg q12hrs, may

Table 6.4 Antibiotics commonly used.

Commonly Used Antibiotics in Gerbils	
Drug	**Dose**
Azithromycin	30 mg/kg PO q24h
Ciprofloxacin	10 mg/kg PO q12h
Chloramphenicol	30–50 mg/kg SC, IM, IV, PO q8–24h
Doxycycline	5 mg/kg PO (no dairy) q12h, 50–100mg/kg q24hrs
Enrofloxacin	10–20 mg/kg PO, SC q12h (IM painful and may result in sterile abscess/muscle necrosis)
Marbofloxacin	5 mg/kg PO q12–24h
Metronidazole	15–20 mg/kg PO q12–24h
Oxytetracycline	50 mg/ kg PO q12h
Silver sulfadiazine cream	Topical q12–24h
Tetracycline	10–20 mg/kg PO q12h
Trimethoprim sulfamethoxazole	30 mg/kg PO q12h 50–100 mg/kg q24hrs
Tylosin (use with caution as has been associated with toxicity)	2–10 mg/kg PO, SC q12h 0.5 mg/mL (500 mg/L) drinking water

be used empirically, nebulisation with F10 RTU twice daily for 30 minutes, NSAID, mucolytics, and a full husbandry review. See Table 6.4 for commonly used antibiotics in gerbils.

Tail slip

The skin of the tail is more loosely adhered compared to other species. If grasped by the tail it is common for the tail to deglove, exposing muscle and bone. *This leads to painful desiccation of the tissues and necrosis*. Amputation of the tail is required.

Once surgically prepared, I will use a sterile gauze swab to push the remaining skin further down the tail vertebrae to enable cutting the bone (Mayo scissors are usually sufficient) below the level of the skin edge. In my experience there is no need to tie off the blood vessels associated with the tail. The skin edges should be freshened by trimming the skin, leaving the ventral aspect longer than the dorsal so that when closed (either by 1m (5/0) Vicryl on a needle or wound glue) the skin wound is on the dorsal aspect therefore not in contact with the ground.

Dental disease in gerbils

It is only the incisors that grow continuously. The incisors are yellow/orange due to iron deposition within the enamel. The molars are multi-rooted. The dental formula is: 2 (I 1/1, C 0/0, P 0/0, M 3/3) = 16.

Periodontal disease

Gerbils develop spontaneous periodontal disease and are used as models for periodontal disease studies in humans. Advanced disease can be common over two years of age but may be seen as early as 6 months of age.

Soft plaque forms and hyperplastic gingival margins develop. The earliest and most severe gingival changes are usually in the interdental areas (especially 1st and 2nd molars). Loss of the supporting tissue causes gaps to form between the molars leading to impaction of debris such as food, fur, and shavings. Acute inflammation and oedema of the gingiva will occur. The molars may loosen and become lost. Loss of the 3rd molars is common in older animals.

Dental caries, tooth decay and abscessation can occur especially if fed on an inappropriate, high sugar diet. Maxillary molar abscessation may result in facial swelling, exophthalmos (retrobulbar abscess) and secondary exposure keratitis of the cornea.

Due to the spontaneous, insidious, and progressive nature of periodontal disease, radiographs are required to fully assess the extent of the disease. Periodontal destruction is often more severe in the maxillary arcades than the mandible.

Diarrhoea

Just as in more familiar species there are a range of causes of diarrhoea in gerbils, from dietary indiscretion (owner changing food too quickly, diet too high in fruit/vegetables, or giving too many treats), to infectious causes (bacterial: Tyzzer's disease, *Salmonella* spp., *E. coli*, *Citrobacter rodentium*), intestinal amyloidosis, to iatrogenic drug induced causes. Minimising stress is important as *stress and the associated immunocompromise are common predisposing features of developing diarrhoea*.

Gerbils with diarrhoea dehydrate rapidly so it is important that supportive care is started quickly.

Tyzzer's disease

Tyzzer's disease is almost invariably fatal in gerbils and is caused by an overgrowth of *Clostridium piliforme*. *Clinical disease is seen in all ages of gerbils (more common in young animals) and clinical outbreaks can be precipitated by severe stress* (e.g. poor environmental conditions, overcrowding, shipping, management changes, concurrent disease, or inadequate diet). Gerbils with Tyzzer's disease can develop hepatic, myocardial and/or ileocecal necrosis.

Clinical signs can include depression, unkempt damp coat, acute pale yellow diarrhoea, faecal stained perineum, dehydration, hunched back, or sudden death with no prior clinical signs.

Diagnosis is suspected based on history and clinical signs but can be confirmed via polymerase chain reaction (PCR) of faeces.

Prognosis is poor and treatment involves aggressive supportive care (fluids, warmth, assisted feeding), liver support (silymarin 50–200 mg/kg/day) and suggested antibiotics include doxycycline or metronidazole 10–20 mg/kg q12hrs.

Hepatomegaly with multiple, small, pale foci will be commonly seen on post mortem examination.

Citrobacter rodentium

Clinical signs may resemble Tyzzer's disease, but the diarrhoea is usually bloody and there can be sloughing of the superficial epithelium into the gut lumen.

Diagnosis is via clinical signs and bacteriology, although reports are conflicting on ease of culture.

Prognosis is poor and treatment involves supportive care and antibiotics (ideally based on culture). De La Puente-Redondo *et al*. (1999) found that quinolone antibiotics were the most effective against their isolates, with tetracyclines being the next most effective.

Bloat

Like all rodents, gerbils cannot vomit or eructate. Distention of the stomach can occur due to: unsuitable or contaminated food, secondary to ileus, secondary to inappetence, or secondary to malocclusion.

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Clinical signs include: bruxism, distention of the abdomen, respiratory distress (pain and pressure on the diaphragm), cardiovascular impairment and signs of the predisposing condition.

Gastric bloat is often fatal. Supportive care (warmth, fluids, and nutrition) and analgesia should be started immediately. General anaesthesia is required for decompression of the stomach via a urinary catheter. Diagnosis and treatment of the primary condition is required.

Endoparasites

Disease due to endoparasites is uncommon in gerbils.

The gerbil pinworm *Dentostomella translucida* is found in healthy gerbils but may affect growth if there is a heavy burden. A heavy infestation may occur due to immunocompromise therefore may be a secondary condition. Diagnosis is via faecal flotation demonstrating the typical ovoid egg. Treatment is fenbendazole 20 mg/kg q24hrs for 5 days.

Two species of dwarf tapeworm have been documented in gerbils (uncommon in temperate climes): *Hymenolepis diminuta* and *Hymenolepis nana*. Heavy burdens may cause mucoid diarrhoea and dehydration. Treatment is praziquantel 30 mg/kg q14 days for 3 treatments.

Dermatology in gerbils

Just as in more familiar species, a full clinical history and assessment of pattern of clinical signs may give clues as to the underlying cause. As dermatological conditions are often multifactorial, any underlying environmental stressors must be identified and rectified.

Ectoparasites

Ectoparasites are rare in gerbils. Two main mites have been reported to cause clinical signs in gerbils.

Demodex meroni has been reported to cause alopecia, scaling, ulceration, and secondary bacterial infection primarily affecting the face, thorax, abdomen, and limbs.

Diagnosis is via examination of a hair pluck under microscope (trichogram) or a deep skin scrape (under a brief general anaesthetic to decrease stress). Immunosuppression may play a part in clinical disease so a full history and clinical exam looking for co-morbidities should be performed.

Treatment includes correction of any stressors, treatment of co-morbidities and selamectin 15–25mg/kg topically q2 weeks for three treatments. Cases involving secondary infection require antibiotics.

Acarus farris is an oval, non-parasitic forage mite which has been reported to cause alopecia, scaling, and thickening of the skin primarily over the tail, head, and hind end. Although non-parasitic it is thought to cause contact dermatitis due to an allergy to either the mite itself or the droppings. Forage mites have been documented causing skin disease in multiple species.

Diagnosis is via a trichogram in liquid paraffin.

Treatment is both environmental and application of fipronil spray topically. Spray onto a paper towel and wipe onto the gerbil rather than spray directly. Environmental control involves freezing all new bedding and food at –18°C (–0.4°F) for at least 48 hours to kill any new mites, lower humidity if it is too high, and transferring the gerbils to a newly cleaned enclosure after treatment.

Unkempt hair coat

Husbandry issues or underlying disease may result in poor coat condition.

Gerbils require regular access to sand baths for coat condition. Clinical history will reveal whether regular sand baths are offered. Ideally, a shallow dish with chinchilla sand should be placed in the enclosure for 30 minutes several times weekly.

Lack of ventilation may result in high relative humidity levels, especially if there are inadequate bedding changes. *Once humidity levels reach over 50% the hair coat becomes unkempt, greasy, and matted*. Ventilation needs to be improved to bring the humidity level below 50%. Bedding should be partially changed as full changes can be extremely stressful (scent is important to gerbils' sense of safety).

Nasal dermatitis

Excessive production of porphyrin from the Harderian gland leads to nasal irritation, progressive alopecia (from over grooming nose), dermatitis (secretion is irritant), and secondary self-trauma leading to secondary bacterial infection (frequently *Staphylococcal* spp.). Over secretion of porphyrin is often stress-induced, risk factors include humidity >50%, overcrowding, illness, fighting, poor sanitation, lack of dust bath, and inappropriate substrates (pine shavings may cause due to aromatic oils).

> The owners may mistake the porphyrin for blood, presenting the gerbil for a nose bleed.

Clinical signs can include erythema, alopecia, moist dermatitis, scabs/crusts around nares – which may progress to affect paws, face and ventral abdomen, secondary bacterial infections, and may even progress to sinusitis in severe cases. *Crusts will fluoresce under UV light due to the porphyrin*.

If the only clinical sign is excess porphyrin at the nares, then consideration should be given to whether it is genuinely excess secretion, or a lack of grooming due to illness or pain preventing the normal removal of the normal levels of secretion. Differentiating porphyrin secretion from blood is easily achieved by either cytology or using a Wood's Lamp to check for the fluorescence of the porphyrin.

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As environmental factors are involved, full husbandry assessment and corrections are required as part of the treatment. Medical treatment involves: analgesia, facial cleansing (e.g. dilute chlorhexidine), topical therapy (e.g. F10 wound ointment or Flamazine cream (Silver sulfadiazine) applied twice daily after cleaning, severe cases will need systemic antibiotics (ideally guided by culture and sensitivity, but trimethoprim sulfadiazine may be started empirically). *Owners should be counselled that severe cases may be fatal.*

Sand baths should not be given until the lesions have healed.

Bilateral flank alopecia

This may be seen with either ovarian cysts (common – see section on reproductive conditions), or hyperadrenocorticism (rare).

Hyperadrenocorticism can be either adrenal or pituitary and the gerbil will exhibit polydipsia/polyuria.

Ringworm

Dermatophytosis is rare in gerbils. Both *Trichophyton mentagrophytes* and *Microsporum gypseum* have been reported in gerbils although clinical infection is only seen in aged and/or *immunocompromised* individuals, therefore the underlying cause must be investigated and rectified also.

Clinical signs include mildly pruritic, focal areas (may be circular), of hair loss or broken hair shafts, and hyperkeratosis affecting the ears, head, back and legs. Owners should be advised of the zoonotic risk.

Diagnosis is via fungal media growth or identifying fungal hyphae on skin scrapes.

Treatment includes environmental decontamination (wooden items should be destroyed), any underlying comorbidities should be identified and treated, and oral itraconazole 2.5–10 mg/kg q24hrs should be given for 2 weeks post-resolution of clinical signs. Treat all in contact with gerbils as they will be asymptomatic carriers.

Neoplasia

Neoplasia is common in gerbils, mainly reported in gerbils over two years of age. The most commonly affected sites are the skin (and associated scent glands), ovary and the adrenal gland. 80% of spontaneous neoplasms in gerbils of 2–3 years of age are either squamous cell carcinoma of the ventral scent gland in male gerbils or granulosa cell tumours of the ovary in female gerbils (Fox *et al.*, 2002).

Scent gland conditions

Both male and female gerbils have ventral scent glands, albeit the females' gland is much smaller and much less obvious. The male gerbil has a comparatively large gland (approximately 2.5 cm long by 1 cm wide, size is directly related to testosterone levels), oval, and orangish in colour. The gland is used for territory mark and individual identification.

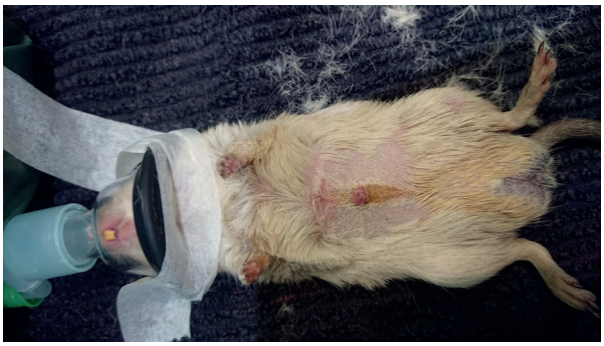


Figure 6.3 An early stage growth in the ventral scent gland in a male gerbil.

Male gerbils are overrepresented (almost exclusively) for neoplasia of the gland. Early cases of neoplasia may initially look like impaction or infection (which is uncommon), or a small raised area of ulceration (Figure 6.3).

Clinical signs

The first clinical sign owners may notice is a smear of blood on the bedding or focused grooming of the ventral abdomen. Many owners do not think to look at the ventral abdomen and will be shocked to hear there is a mass. Neoplasia of the gland causes pain and/or irritation resulting in self-trauma, haemorrhage, secondary bacterial infection thereby impairing quality of life (Figure 6.4).

A variety of neoplasms have been documented at this site including (in no order): glandular hyperplasia, adenoma, papilloma, epithelioma, adenocarcinoma, squamous cell carcinoma, basal cell carcinoma, and poorly differentiated carcinoma.

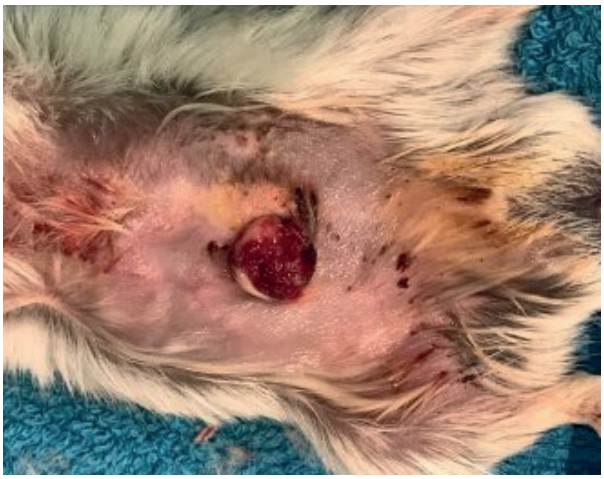


Figure 6.4 A more advanced growth in the ventral scent gland in a male gerbil. The owner noted blood on the substrate.

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Epitheliomas are not fully benign in gerbils as they show high mitotic rates and areas of necrosis histopathologically, but they have a low metastatic potential therefore complete surgical excision should be curative. *Adenocarcinomas may be locally invasive and have been reported to metastasise to regional lymph nodes and the lungs*.

Pre-operative definitive diagnosis via FNA is possible but considered unnecessary given that *regardless of neoplasm type, surgical removal is advised to prevent further self-trauma and infection*. Due to the high prevalence of malignancy, radiographing the thorax is advised to check for metastasis, and histopathology is advisable to give prognosis.

Surgery

Surgical resection should be performed using an elliptical incision, with *at least a 2 mm margin* of healthy skin advised. 0.7m (6/0) Vicryl on a needle is used to close skin with an intradermal suture pattern (Figure 6.5).

EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) can be applied to the wound after closing if a splash block has not been used. Analgesia should be continued for a week to ten days postoperatively. Provided the gland is fully resected, surgery is often curative unless metastasis has already occurred.

Aural cholesteatoma

Aural cholesteatomas are benign, keratinised, epithelial neoplasms which grow either from the tympanic membrane or the distal ear canal. The mass expands through the tympanic membrane and invades the inner ear. Further growth causes progressive erosion of the surrounding bone.

Clinical signs include head tilt, aural discomfort, facial paralysis, dysphagia, discharge from affected ear, and the mass may grow large enough to protrude from the ear. *The initial presentation may be similar to a case of otitis media but while otitis media is documented in gerbils, it is uncommon.*

Treatment is mainly palliative as the condition is progressive and due to the location is poorly accessible for surgical resection. Medical management with NSAIDS, multimodal analgesia and antibiotics if secondary bacterial infection present will improve quality of life.

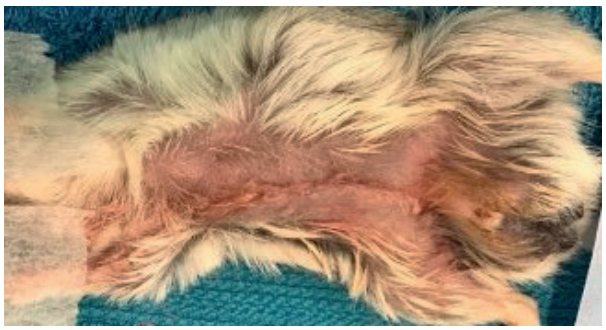


Figure 6.5 A postoperative Figure of the gerbil in Figure 6.3. The wound has been closed using an intradermal suture pattern.

Reproductive tract neoplasms

Ovarian neoplasia

Ovarian neoplasia is commonly seen in gerbils over two years of age (but can be seen in younger animals) with a reported incidence of between 8.4–26.5% depending on the study. Granulosa cell tumours are the most common ovarian neoplasm seen and one report indicated a metastatic rate of 18% with metastasis was common to the omentum and mesentery with metastasis to the lungs rarely seen. Theca cell tumours, luteal cell tumours, interstitial cell tumours, dysgerminomas, and teratomas have been reported. Concurrent ovarian cysts are common.

Clinical signs are similar to ovarian cysts, including apathy, reduced appetite or anorexia, abdominal distention, dyspnoea, reduced mobility, vaginal discharge (which may be haemorrhagic or purulent if secondary bacterial infection).

Diagnosis can be achieved by ultrasound examination of the ovary and the abdomen can be scanned to check for metastasis. Ovariohysterectomy is the treatment of choice.

Other reproductive tract neoplasms

Uterine tumours tend to be malignant in gerbils. Uterine hemangiopericytoma, leiomyoma, leiomyosarcoma, uterine adenocarcinoma, have all been documented. *Mammary tumours are rarely reported in gerbils*.

Testicular tumours are uncommon but are reported in gerbils over two years of age. Seminomas and teratomas are documented. Castration is the treatment of choice. Prostatic adenomas are reported.

Other neoplasms

Lymphoma and lymphosarcoma are uncommon compared to the other rodent species. Osteosarcoma, systemic mast cell disease, thyroid adenoma, salivary gland sarcoma have all been documented.

Reproductive diseases

Ovarian cysts

Ovarian cysts are very common in gerbils, mainly over 2 years of age but are seen in younger gerbils and can be uni- or bilateral. *The most common cause of bilateral, rounded, abdominal masses in female gerbils is cystic ovaries.*

Clinical signs are: apathy, reduced appetite or anorexia, abdominal distention, dyspnoea, reduced mobility, symmetrical bilateral flank alopecia (if cysts are hormonally active), and vaginal discharge (which may be haemorrhagic or purulent if secondary bacterial infection).

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Diagnosis is via ultrasound and the treatment of choice is ovariohysterectomy.

Reproductive organ neoplasia

See section on neoplasia.

Pyometra

Pyometra is recognised in gerbils with clinical signs similar to more familiar species. Diagnosis is via clinical signs, ultrasound, and cytology of vaginal discharge. Treatment of choice is ovariohysterectomy, supportive care, analgesia, and antibiotics.

Epilepsy

Seizures are recognised as an inherited condition in gerbils and are usually first seen at two months of age, they increase in frequency as the gerbil ages, although *most will outgrow the seizures by six months old*. The seizures may vary in intensity from full epileptiform seizures to some individuals only experiencing twitching of the vibrissae and pinnae.

Seizures are often triggered by a stressful incident such as handling, cage cleaning, and moving the enclosure. Medical treatment is not normally required. Minimising stress is important and gerbils may become habituated to certain previously stressful stimuli.

Renal disease

Renal disease is common in gerbils, both chronic interstitial nephritis and renal amyloidosis being documented in aged animals. Renal tumours are rare.

Clinical signs are the same as for other species: polydipsia, polyuria, dehydration, weight loss, decreased activity, cystitis, and reduced or loss of appetite.

Diagnostics advised are biochemistry and urinalysis. Gerbils will often pass urine when placed in a clean plastic container and that can be examined for specific gravity, dipstick and ideally microscopy.

Table 6.5 Urinalysis.

Gerbil Urinalysis Normal	
Colour	Variable depending on diet
Protein	+
Glucose	+
Bilirubin	+
Acetone	+
pH	7.4–8.2
Specific Gravity	Highly concentrated
Sediment Examination	Calcium carbonate and triple-phosphate crystals

Urine specific gravity consistently less than 1.015, plasma urea >5.2 mmol/l (>31.3 mg/dl) and crea >124 umol/l (>1.4mg/dl), in a dehydrated individual is highly suggestive of renal disease. Proteinuria and hypoalbuminaemia may be seen. Urine culture should be considered if bacterial involvement is of clinical concern (see Table 6.5 for normal urinalysis).

Treatment is as per rats.

Diabetes mellitus

Gerbils fed on muesli or seed-based diets are thought to be over represented due to their likelihood of obesity. Obese gerbils are prone to developing hyperglycaemia and diabetes mellitus.

Clinical signs are the same as in more familiar species: lethargy, weight loss, hyperglycaemia, polydipsia, polyuria, glycosuria, and cystitis. Diagnosis is via clinical signs and persistent hyperglycaemia and persistent glycosuria. *Gerbils can exhibit stress-induced glycosuria which should not be interpreted as diabetes*. Treatment requires dietary improvement, weight loss if obese, and increased exercise. Should blood glucose levels fail to normalise, glipizide 0.6m/kg (increase if required) may be effective.

Euthanasia

End of life conversations can be a difficult area to broach with some clients. Occasionally owners will wait for us to broach the topic as they are 'worried' if they do so we will think they are not dedicated enough. Reassuring clients that sometimes we need to love our pets enough to let them go when the time is right can be invaluable.

Many owners will want to stay with their gerbil during this procedure and the process should be fully explained including possible events such as sudden jumping, or gasping. I administer *a deep sedation* (e.g. medetomidine/butorphanol injected SC), once the pet has fully relaxed, I will use the kidney by choice, to inject the pentobarbitone, to allow the owners to stroke their pets head. *Intra-abdominal or intra-cardiac injections should never be administered in a conscious gerbil as it is considered extremely painful.*

Many owners will opt for private cremation with return of their pets' ashes and all options that your practice offers for dogs and cats should also be made available to your gerbil clients. I offer a lock of fur, footprints (coloured ink pad from craft shop and a card). I send sympathy cards if I know the patient and owner well.

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7 Chinchilla

Chinchillas (*Chinchilla lanigera*) have only been kept in captivity for around 100 years. They are hystricomorph rodents, their incisors and cheek teeth continuously grow, and the labial surface of the incisors have iron deposition within the enamel resulting in a yellow orange colouration. They are clean rodents with virtually no smell. The long gestation period is 111 days as they birth precocial young. Their average lifespan is 10–15 years of age but late teens or over 20 is not unusual. The maximum recorded lifespan for a chinchilla in the wild is 6 years old.

Chinchillas are native to the semi-arid mountainous Andes in South America, where they live in large groups of 100–500 individuals (called herds) at high elevations of 3,000–5,000 m. The natural habitat is rugged with rocky or sandy ground. They occupy tunnel systems dug between roots of cacti and/or succulents with a minority living in rock crevices and are extremely active and agile. *In the wild they are crepuscular and nocturnal with 70% of their food intake occurring during the night.*

Chinchillas have several physiological adaptations for limited water availability: they produce highly concentrated urine, very dry faeces, and they have lower evaporative water loss compared to other rodents.

Wild diet

They evolved to consume low-quality, fibrous plants, leaves, stems, and seeds that are found on the mountainous slopes. *Their diet varies seasonally, feeding on up to 24 different plant species.* Their mainstay diet is herbs and grasses, especially needle grass (*Nassella chilensis*), during the summer they mainly consume succulents (increases their water consumption) and they have a highly adapted gut which allows them to get adequate nutrition from a low-quality food source.

Companionship

No research has been performed on conspecific companionship in chinchillas, however, given the fact they are a prey species who evolved to live in single species groups of 100–500 (100 is the most common size), it is reasonable to assume they have an innate requirement for the company of another chinchilla. Common grouping sizes are 2 or 3 in captivity, either all female or mixed sex with a neutered male. Regardless of sex ratio, the chinchillas need to be introduced as youngsters to prevent fighting. Same sex pairs may fight when they reach sexual maturity (usually 8 months old but occasionally younger), especially if there is a lack of space.

Owners may need to be advised that they should not keep the opposite sexes in the same room, as although it is common sense to us not to do so, I recently had a case where a long standing bonded pair of entire male chinchillas started fighting. On questioning the owner further they insisted they kept single sex bonded chinchillas despite me finding out they had a cage with entire female chinchillas in the same room. The owner struggled to understand that the sight, sound, and smell of the females in season was disrupting the bond of the male chinchillas despite not actually being in the same cage and that they should not keep mixed entire sexes in the same air space. *It is important to not only treat any injuries of fighting but also to establish why the bond has broken down.*

Enclosure and enrichment

Chinchillas are extremely active and agile and can jump distances of 1.8 m (6 ft). They require large multilevel cages to meet their needs. Minimum size recommended is 2 m × 2 m × 2 m. *Many chinchilla cages are made of wire, including the floors, which enables excellent ventilation but may predispose to pododermatitis.* The mesh size must be 15 mm × 15 mm or smaller to reduce risk of injuries caused by foot entrapment. Larger mesh size commonly results in tibial fractures due to the hind foot becoming trapped. Galvanised steel is frequently used to make the cages. *Care should be taken with cheaply made cages which have visible beads of galvanised material as this presents a risk for zinc toxicity.*

If all the floors are mesh, it is important to ensure *some of the levels are solid floored* (at least 25% of the floor space) *to allow the chinchilla to choose to spend time off the mesh flooring. Pregnant chinchillas must be given solid flooring.*

Shelving and thick branches can be added which helps create a complex 3D environment allowing a more normal expression of behaviour. Some chinchillas like access to hammocks. It is important to *ensure there is no uninterrupted height of 60 cm or more to reduce risk of injuries from falls.*

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Recommended bedding includes dust extracted hay and shavings. *Łapiński et al. (2023) found that chinchillas that were given a larger cage, with solid floors and with a deep litter substrate of shavings were more active and less fearful of humans*. It could be interpreted that the substrate made the chinchillas feel more secure because it enabled them to move more easily as well as allowing normal behaviour such as digging and rolling.

Chinchillas are shy animals and need several options to hide. Wooden nest boxes should be provided the ideal size being 25 cm × 25 cm × 25 cm and lined with hay. Plumbing pipes 10–15 cm (4–6 inches) in diameter make excellent hides and can be hung in the enclosure. They should be monitored for signs of gnawing. Cardboard tubes can also be a good option encouraging their innate requirement to chew.

Safe woods to include as enrichment for chewing include elm, grapevines, maple, birch, and pieces of bark from apple, pear, peach, and ash trees.

Solid wheels can be provided for running. To prevent painful spinal conditions, it is important to provide a large enough wheel that they do not have to bend their back while running ideally at least 38 cm (15 inches) in diameter. They should be able to run with a straight back and without having to lift their head.

Supervised time out of their enclosure is important. Carpeted cat towers make good places for chinchillas to explore while out of their cage. Care should be taken to protect electrical cables to prevent electrocution if they are gnawed by the chinchilla.

Owing to their luxurious fur (which they evolved to cope with the cold), chinchillas are prone to heat stroke. The ideal temperature range is quoted to be 10–15°C (50–59°F). At temperatures over 25°C (77°F) chinchillas become distressed (especially if the humidity is too high) which will progress to fatal heat stroke if the temperature continues to rise.

Dust bath

Chinchillas require regular access to dust bathing to care for their fur. *Unless there are fresh traumatic injuries or surgical wounds, the dust bath should be offered for 20–30 minutes daily*. It is important that the dust bath is removed from the cage after the allocated time, as chinchillas are prone to over bathing leading to conjunctivitis due to the dust irritating the eyes. They may also use the dust bath as a latrine.

Play sand is not suitable for dust bathing. The most recommended substrates for dust baths are Fullers Earth, volcanic ash, and commercial chinchilla dust. The pan should be large enough for the chinchilla to roll around and the depth of the dust should be 3–5 cm.

Diet

Chinchillas evolved to consume low-quality, fibrous plants, leaves, stems, and seeds that are found on the mountainous slopes, which varies seasonally, feeding on up to 24 different plant species. They have a highly adapted gut that allows them to get adequate nutrition from a low-quality food source. They perform caecotrophy directly from anus, with approximately 40% of faeces reingested. *In captivity we should try to mimic this diet as closely as possible. Any dietary change should be implemented slowly to prevent bloat or diarrhoea*.

The mainstay of pet chinchillas' diet is ad lib high-quality hay and dried grasses supplemented with chinchilla pellets (25–50 g/chinchilla/day). If fed pellets ad lib they will preferentially consume pellets over hay, which leads to a decrease in water consumption as well as increasing the risk of dental disease.

Small amounts of vegetables (e.g. carrots) and low sugar fruits (e.g. blueberries or strawberries) can be offered as treats although brassicas can occasionally predispose to gas formation and bloat. *Chinchillas seem to prefer dried leaves to fresh ones*. Raisins are loved but they are high in sugar and will predispose to obesity and diabetes so they should be limited to no more than one a few times weekly. Nuts should be avoided, and seeds should be limited to the occasional pumpkin seed due the fat levels.

Muesli diets should be avoided as they allow selective feeding and are generally low in fibre and too high in carbohydrates. Diets too low in fibre and/or too high in carbohydrates cause imbalances in gut flora (which may cause diarrhoea), obesity, and poor dental attrition leading to coronal overgrowth and dental disease.

Although they can synthesise their own vitamin C, Johnson-Delaney (2006) recommended providing *150–200 mg vitamin C/day, to keep the periodontal tissues firm and promote healthy dentition*. Rose hips and kiwi are good sources of vitamin C.

Water

Hagen *et al.* (2014) found that chinchillas preferred to drink from an open bowl. Traditionally open bowls have been avoided due to the risk of matting of fur on the chin. One method may be to provide bottles which feed into a small open dish as opposed to a nozzle or nipple. Water intake is around 73 ml/kg/day on a hay and pellet diet (Hagen *et al.*, 2014).

Ultraviolet light

Rivas *et al.* (2014) demonstrated that chinchillas exposed to UVB light produced more of active vitamin D. *The fact that chinchillas have retained the dermal biosynthetic pathway for vitamin D highly suggests that they have a UVB exposure requirement*, however no studies have been performed to establish safe levels to date.

Vertebrates with hypovitaminosis D are susceptible to diseases that affect the musculoskeletal, cardiovascular, and immune systems. Acquired dental disease is so common in chinchillas it is possible that chronic hypovitaminosis D

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may play a role in nutritional osteodystrophy of the skull (Reiter, 2008).

Sexing

Male

Male chinchillas have a longer genital–anal distance (Figure 7.1). The prepuce is conical in shape and although there is no true scrotum, there are two movable sacs lateral to the anus (post-anal sacs) which the caudal epididymis can drop into. The penis can be manually extruded and is 1–2 cm when flaccid, when erect it can be up to 11 cm.

Female

Female chinchillas have a shorter genital–anal distance (Figure 7.2). The urethral process protrudes and may be confused as a prepuce. There is a well-developed clitoris that can be manually extruded through the urethral process and mistaken for a penis, however there will be no distinction between the clitoris and the process which there would be between a prepuce and penis. The vaginal opening sits between the urethral process and anus but will be covered by a membrane unless in oestrus. There are three pairs of nipples, two pairs laterally on the thorax and one pair inguinally. During oestrus the perineum changes colour from a dull flesh tone to a deep red colour but there is no vulval swelling.



Figure 7.1 A male chinchilla has a longer genital–anal distance.



Figure 7.2 A female chinchilla has a shorter genital anal distance and as the urethral process protrudes it may be confused as a prepuce.

Preventative health care

Regular weighing by owners may help pick up trends in subtle weight losses, indicating that the chinchilla may have an underlying condition. Regular veterinary check-ups are advisable due to their ability to hide pain, although *compared to the other rodents, chinchillas are often medically robust*.

Hospitalisation

With any species, but especially prey species, it is important to minimise stress during handling and while in the clinic. Stressed animals have a high sympathetic tone which can counteract effects of sedatives, predispose to vasoconstriction, excessive myocardial work, and arrhythmias.

Hospitalisation is similar to guinea pigs and care should be given not to overheat as they are prone to heat stroke if maintained in an environment warmer than 27°C (80.6°F), the recommended temperature for the ward is 19–21°C (66–70°F).

Venipuncture sites

Many texts on exotics describe anaesthetising the chinchilla and performing a cranial vena cava stick to draw blood. This carries the risk of death due to uncontrollable haemorrhage. The tendency in chinchillas to readily develop hepatic lipidosis further adds to the risk of haemorrhage. Laboratory vets and technicians have perfected this technique with very few bad outcomes, but they are performing this very regularly whereas clinical veterinary staff do not. Personally, I would not do this to one of my own pets therefore I will not do it to a client's.

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As a rule of thumb, in a healthy chinchilla 1% of the body weight (in grams) is the volume of blood that can be safely taken, in ill individuals it is recommended to take a smaller volume. Realistically, even if a blood sample is taken at 0.5% of the body weight, this still works out at 2.5 ml in a 500 g chinchilla, which is more than enough given we can achieve a biochemistry and a haematology on 1 ml. For blood glucose measurements, the pinnae vessels can be pricked to provide a drop of blood. See Tables 7.1 and 7.2 for reference ranges.

Chinchillas find restraint for venipuncture stressful, so consider an anaesthetic to reduce stress and risk of injuries from struggling. EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) can be applied to facilitate venipuncture.

My vein of choice is the lateral saphenous vein (Figure 7.3a), but other options include the jugular vein, cephalic vein (Figure 7.3b & c), and femoral vein (Figure 7.3d). The gingival vein may be used but is less reliable for sampling in chinchillas compared to other rodents.

Jugular venipuncture can be performed using a 25g needle. Restrain the chinchilla like a cat (head and neck held extended up and front legs held down at edge of table), the jugular is entered utilising a blind stick in the lateral neck (using the same landmarks as per more familiar species).

Lateral saphenous vein and cephalic vein are my preferred choices for intravenous catheter placement using 24–26g catheters (see Dehydration section).

Drugs contraindicated in chinchillas

Antibiotic induced dysbiosis and enterotoxaemia

Narrow spectrum antibiotics (especially ones with predominantly activity against Gram positive bacteria) such as: amoxicillin, ampicillin, cephalosporins, clindamycin, erythromycin, lincomycin, and penicillin should never be given orally to chinchillas. Oral administration causes an overgrowth of Gram negative bacteria, especially Clostridium, which results in an often fatal enterotoxaemia and haemorrhagic typhlitis. If in doubt about the oral safety of an antibiotic always check the formulary prior to administration. That being said, even supposedly 'safe' antibiotics can cause issues with the gut microbiome in some individuals.

Metronidazole should be used with caution.

Other drugs

Cisapride has not been found to have efficacy in chinchillas.

Tramadol has been found to have no efficacy in chinchillas and at high doses can cause transient neurological adverse effects therefore is not recommended for use in this species (Sadar & Mans, 2023).

Species-specific anatomy/behaviour

Chinchillas have virtually no odour, never scratch, and rarely bite.

Chinchillas are hystricomorphs with elodont dentition (elodont: continuously growing throughout life).

Table 7.1 Chinchilla biochemistry.

BIOCHEMISTRY (Heatley & Russell, 2020; *Carpenter & Marion, 2018)	REFERENCE RANGE (UK units)	REFERENCE RANGE (US units)
ALKP	3–47 IU/l	3–47 U/l
ALT	10–35 IU/l	10–35 U/l
AST	96 IU/l	96 U/l
GLU	3.3–6.7 mmol/l	60–120 mg/dl
CHOL	1.03–2.59 mmol/l	40–100 mg/dl
TBIL	7 µmol/l	0.4 mg/dl
UREA	3.6–8.9 mmol/l	10–25 mg/dl
CREA*	35.36–114.92 µmol/l	0.4–1.3 mg/dl
TP*	38–56 g/l	3.8–5.6 g/dl
ALB	24–42 g/l	2.4–4.2 g/dl
CA	1.4–3.02 mmol/l	5.6–12.1 mg/dl
PHOS	1.29–2.58 mmol/l	4–8 mg/dl
K	5–6.5 mmol/l	5–6.5 mEq/l
Na	130–155 mmol/l	130–155 mEq/l
FRUCTOSAMINE (Lagostomus maximus)	215 to 296 µmol/l	3.87–5.21 mg/dl

Table 7.2 Chinchilla haematology.

HAEMATOLOGY (Heatley & Russell, 2020; *Carpenter & Marion, 2018)	UNITS	REFERENCE RANGE
Red blood cells	10^{12}/l	5.2–10.7
Packed cell volume	%	27–54
Haemoglobin	mmol/l (g/l)	4.96–9.56 (80–154)
Mean corpuscular volume	fl	86.5–116.1
Mean corpuscular haemoglobin concentration	mmol/l (g/l)	20.17–23.71 (325–382)
Mean corpuscular haemoglobin	fmol/l	2.8–4.4
Platelets	10^{9}/l	254–740
White blood cells	10^{9}/l	4–11.5
Differential blood count (relative values)		
Monocytes*	%	0–5
Lymphocytes*	%	45–60
Neutrophils*	%	39–54
Eosinophils*	%	0–5
Basophils *	%	0–1



Figure 7.3a The lateral saphenous vein being used for venipuncture.

They have two incisors in the maxilla and two in the mandible which grow 5–7.5 cm/year in healthy individuals. Dental formula: $2 \times (I\ 1/1; C\ 0/0; P\ 1/1; M\ 3/3) = 20$.

The tight cardiac stomach sphincter prohibits vomiting or eructation. There is no need to starve for an anaesthetic.

They are hindgut fermenters with a large caecum.

The tympanic bullae are particularly large.

A palatal ostium makes intubation challenging.

The pupil is a vertical slit instead of round.



Figure 7.3b The cephalic vein.

Their fur is unusually thick with 60–90 individual hairs emerging from a single follicle. They do not tolerate dampness and will develop matted fur if kept in a warm humid environment.

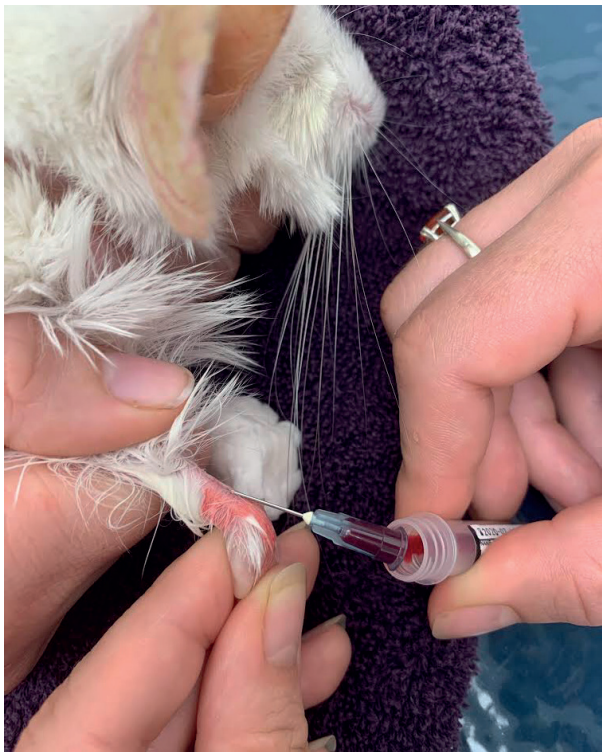


Figure 7.3c The cephalic vein being used for venipuncture. The needle size should be judged on the size of the vein in the individual.



Figure 7.3d The femoral vein.

Male chinchillas have open inguinal canals. Female chinchillas have two uterine horns and two cervices (externally will look like one cervix). They have very long gestation periods (111 days) and birth precocial young.

The tibia is longer than the femur and the fibula is virtually non-existent.

They have several adaptations to cope with water deprivation. They produce very concentrated urine. Their bowel is around twice the length of that of guinea pigs and their large intestine ensures maximum water absorption from faeces, producing small (average 8 mm × 3.9 mm), dry, dark, hard faecal pellets.

Flight is their main defence mechanism although they may bite or spray urine while standing up if they are not used to handling.

If they are frightened, chinchillas may shed a patch of fur (known as fur slip). Owners should be reassured the patch will grow back (takes 6–8 weeks) although it may be several months before it becomes indistinguishable. Chinchillas hide illness and pain.

They spend time resting on their hind legs.

Chinchillas can jump off the consulting table leading to injuries.

Pain in chinchillas

One of our jobs as veterinary professionals is to *actively look for pain*. In Chapter 1 we discussed that all vertebrates have the same anatomical and physiological pain pathways.

There is very little in the literature on specific studies performed in chinchillas investigating pain and analgesic efficacy. Doses are extrapolated from other rodents (I tend to use the same doses that I use in guinea pigs) and regular reassessment to ensure adequate control of pain is being achieved is vital.

Osteoarthritis in chinchillas

Osteoarthritis is a degenerative condition affecting the cartilage and bones of a joint. It is commonly referred to as the wear and tear arthritis. Any species with a skeleton can develop osteoarthritis, species that actively hide pain mean that we have to actively look for skeletal pain in these species. Osteoarthritis of the temporomandibular joints will cause secondary dental disease due to pain caused by chewing. Osteoarthritis of any joints may cause secondary dental disease due to a decrease in appetite, which as it happens slowly, will often not be noted by the owner.

Analgesia

Doses that I use are mainly extrapolated from guinea pigs (I generally start at the low end if ranges offered) and regular reassessment to ensure adequate control of pain is

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Table 7.3 Chinchilla analgesia.

Analgesic	Dose
Meloxicam	0.6–2 mg/kg q12hrs PO SC
Paracetamol (acetaminophen)	25–50 mg/kg q8–12hrs PO
Buprenorphine	0.2 mg/kg SC q4–6hrs
Gabapentin	10–30 mg/kg q8–12hrs PO
Maropitant	2 mg/kg SC
Methadone	0.5–2 mg/kg SC q4hrs dependant on pain scoring
Lidocaine	2 mg/kg (maximum 4 mg/kg)
Bupivacaine	1 mg/kg (maximum 2 mg/kg)
Acupuncture	

being achieved is vital. See the Analgesia section in Chapter 3. *Tramadol has been found to have no efficacy in chinchillas and at high doses can cause transient neurological adverse effects therefore is not recommended for use in this species* (Sadar & Mans, 2023). See Table 7.3 for suggested analgesic doses.

Clinical exam

See Table 7.4 for biological data. I always place a towel or blanket on the table to reduce the patient's stress. Prey species have evolved to be prey, therefore have an innate requirement to escape predation at a split-second notice. This is why they find a slippery surface so frightening, if they cannot find purchase, they cannot escape predation. Using the blanket or towel reduces the chance of them injuring themselves or you as their handler.

I will look at the chinchilla face on to check for facial symmetry.

Personally, I prefer to perform the rest of the examination with the chinchilla on my knee (if friendly and comfortable with handling) or on the table. I perform the same clinical exam, in the same order that I do for any other species (except that I perform oral examination of the molars last). See Chapter 3 for clinical exam in guinea pigs for more detailed information on clinical exam (but not the oral examination) (see Video 7.1 for skeletal pain examination).

Table 7.4 Biological data.

Parameter	Reference range
Average longevity	10–15+ years
Respiratory rate	45–80 bpm
Heart rate	200–300 bpm
Body temperature	38–39°C (100.4–102.2°F)
Maintenance fluid therapy	Quoted 36–90 ml/kg/day, I use 60 ml/kg/day
Sexual maturity	6–9 months
Gestation	111 days

Performing conscious oral clinical exam

After wrapping the chinchilla in a burrito, I will ask an assistant (which may be the owner) to hold the pet vertically with its back nestled against their breastbone supporting the thorax and its hindquarters. I use the medium metal otoscopic cone.

Restraint of the head is important. I use my non-dominant hand (for me that is my left hand) to restrain the head, I place the palm of my hand flat onto the skull and use my index (pointer) finger and thumb to hold onto the point of the zygomatic arch just distal to the eyes. I then pull gently caudally with my index and thumb. The maxillary incisors are approximately 0.6 cm and are squared at end, mandibular incisors are 0.9 cm and are more pointed at the ends. The incisors should be yellow/brown in colour. Patches of white or white incisors may be associated with a calcium or vitamin D deficiency.

> Chinchillas sometimes have food in their mouths, so this is completely normal. However, it does mean that the oral cavity must be swabbed out with cotton buds before the teeth can be visualised.

Chinchillas normally have a horizontal occlusal plane; the molars are cream coloured and should be approximately 1–2 mm above the gingiva in healthy animals.

It is possible to have coronal overgrowth without spike formation if the teeth are still working as functional units. Crossley (2001) reported that 35% of apparently healthy chinchillas in the UK have cheek tooth coronal elongation. In these cases, the teeth are symmetrical.

I place the otoscope cone into the right diastema just caudal to the upper incisors. Examine the soft tissues for any wounds, ulceration, or bleeding. Move around the chinchilla, without taking the scope out, and perform the rest of the clinical exam to include both the upper and lower arcades.

Maxillary molars flare towards the cheek when they overgrow. The lower arcades grow towards the tongue. Dental caries are brown black in colour and are seen more easily on the occlusal surface.

Many vets (including myself) used to focus on looking for spikes when performing this exam. *Now I advise looking for asymmetry (seeing spikes is a bonus)*. Asymmetry is often easier to appreciate. If they have pain on one side, they will preferentially chew on the other side which leads to asymmetrical wear of the continuously growing molars. If in doubt I advise full oral exam under sedation or general anaesthesia (once stabilised if anorexic) including skull radiographs. This is discussed fully in the section on dentistry.

The ears can be examined with a smaller speculum.

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Anorexia and gut stasis (ileus)

Anorexia is a true emergency in chinchillas that can lead to gut stasis, a negative energy balance and fatty liver syndrome (very prone to this especially if overweight) developing within 12–24 h. It is important to remember that *anorexia is a symptom* not a condition in chinchillas.

Unfortunately for us, anorexia is a nonspecific reaction to stress which can be physical (pain anywhere and illness) or psychological (separation from cage mate, change in environment). A good clinical history and thorough clinical exam will often help direct your diagnostic tests. I assume any anorexic chinchilla is in pain somewhere.

For example, if the chinchilla has a painful or bloated abdomen without an obvious discrete mass, I will recommend a full general health profile and a conscious dorsoventral radiograph to check for gas in the gastrointestinal tract. This is important to differentiate cases of anorexia which may require immediate surgical intervention from those that should be stabilised before any anaesthetic or surgical procedure is considered.

Stabilisation of the anorexic chinchilla

The mainstays of stabilisation are analgesia, assisted feeding, correction of dehydration, and prokinetics.

Dehydration

Severe dehydration will cause sunken eyes, a delayed capillary refill time, and a prolonged skin tent; however, milder cases of dehydration may have no obvious signs. Chapter 4, Dehydration section should be consulted for signs of dehydration in rodents.

> Oral rehydration is often underestimated, but in cases of mild dehydration, oral rehydration is highly successful.

Owing to chinchillas being from an arid environment and having adaptations to conserve water, their maintenance fluid therapy volume is lower than guinea pigs, and 36 ml/kg/day is recommended by Jepson (2009), while other authors recommend 45–90 ml/kg/day. *I tend to use 60 ml/kg/day as fluid maintenance volume in chinchillas*.

Fluids should be warmed to body temperature (38–39°C (100.4–102.2°F)) prior to administration. *I do not agree with subcutaneous fluid administration. It is incredibly painful* and dehydrated patients will not absorb fluids via this route as efficiently.

Intraperitoneal fluid administration can be given once (repeated administrations are not advisable). Maximum volume administered this route is 10 ml using the right caudal quadrant as per other rodents to minimise risk of entry into the gastrointestinal tract.

An intravenous catheter may be placed in the jugular, cephalic or lateral saphenous vein but it is challenging. Chinchillas find restraint for intravenous (IV) catheter placement stressful, and consideration should be given to a brief anaesthetic using isoflurane or sevoflurane to reduce stress. EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) application will help facilitate placement. Their veins require 24–26g catheters.

Intraosseous (IO) fluid administration is much easier. IV or IO administration of fluids should be given to any chinchilla considered to be >5% dehydrated.

IO CATHETER PLACEMENT

My preferred IO site is the proximal tibia, as the tibial crest is easy to find but an alternative site is the proximal femur. General anaesthesia (if not moribund) is required as placement is considered painful. After surgical preparation of the site, instil lidocaine (2 mg/kg) at the level of the periosteum. Using a 25g needle (surgical steel suture material (sterile) as a stylet, or replace the needle) is placed in a normograde fashion, a radiograph should be taken to ensure correct placement as inadvertent puncture of both cortices is a common complication in the tiny species. *IO placement is contraindicated in any patients that have sepsis, osteomyelitis, or metabolic bone disease*.

IO catheters can be connected to syringe drivers or boluses can be administered every 30–60 minutes. The bag of fluids, or syringes of fluids, can be maintained at the correct temperature using a baby bottle warmer. *Ongoing analgesia is important, IO catheter flushing and administration of fluids is painful in humans therefore should be considered painful in other species.*

Hydration status should be assessed throughout the day, enabling you to alter the rate and volume of fluids therapy as required. My intravenous fluid of choice in most cases is saline. The total fluids required can be calculated and divided into syringes to be administered via boluses.

Assisted feeding

The term assisted feeding is preferable to force feeding when discussing with owners.

There are several high-quality, purpose made foods available such as: Oxbow Critical Care, Supreme Recovery or EmerAid Herbivore. All three are high in fibre to promote gut motility, are palatable (although individuals may prefer one over another), and easy to prepare. Feeding syringes with widened nozzles are available from wholesalers made by Supreme Pet Foods and Oxbow.

Some chinchillas will require wrapping in a towel (burrito technique) for assisted feeding, with recommended volumes of 10–20 ml/kg/feed three to four times daily to support gut motility. Some individuals will tolerate smaller, more frequent feeds better.

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Dried grass, hay, and the chinchillas' normal diet should always be readily available to encourage the chinchilla to eat on its own. HEV'N (Holistic Exotic Veterinary Nutrition) make and supply a Gastrointestinal Mix, which is a mix of dried plants that have a prokinetic effect on the gastrointestinal tract, which as chinchillas favour dried leaves over fresh ones, may be more readily consumed.

PROKINETICS/GASTRIC PROTECTANTS

Drugs to help promote normal gastrointestinal motility can be administered.

Metoclopramide (0.5–1 mg/kg q6–8hrs, increases gastric emptying and small intestinal motility) is recommended and I find it useful. Cisapride (0.5 mg/kg q8hrs) is recommended as a prokinetic for rodents in many texts, but Mans *et al*. (2021) found that even at 10 mg/kg q12hrs there was not a clinically relevant effect on faecal output in chinchillas.

I will use metoclopramide until the chinchilla is eating and passing normal faeces.

Ranitidine (2–5 mg/kg q12hrs) is recommended and is very useful in rabbits due to its dual effect of being a prokinetic as well as lower gastric acid production and may be useful in chinchillas. At time of writing this drug is only available from compounding companies. Other viable options which are used in rabbits, are cimetidine (5–10 mg/kg q6–12hrs) or famotidine (0.5–1 mg/kg q12–24hrs).

I will add in a gut protectant if I am concerned about the presence of ulceration. Sucralfate 25–50 mg/kg q8–12hrs can be used in suspected cases of gastric or duodenal ulceration. Sucralfate can be sourced from compounding companies such as Summit or Bova (UK) or Doctors Foster and Smith (USA).

Some owners prefer grinding the tablets and mixing with water for administration. Alternatively, the pill can be administered via a pill giver or a 1 ml syringe with the nozzle and top cut off. Compounding companies make paste versions of many drugs.

Once stable (eating and passing faeces) some owners will decide against further investigation. Depending on your findings during the clinical exam this could be an acceptable approach. For example, if you found spinal or hip pain then continuing with long term analgesia without radiographs is absolutely fine if the animal is clinically stable.

> However, if dental disease is suspected then owners should be counselled this approach would be completely inappropriate, full investigation is required for prognosis, treatment, and management decisions to be taken.

Hepatic lipidosis

Hepatic lipidosis and ketosis is common secondary to anorexia in chinchillas and can occur quickly. *Hepatic lipidosis in anorexic chinchillas is one of the most common findings during post mortem examinations*.

Confusingly, the biochemistry is frequently within reference ranges, although the blood glucose level may be high. *It is important not to diagnose the chinchilla with diabetes mellitus (see Diabetes section) as severe cases of hepatic lipidosis can have high blood glucose, ketonuria, glycosuria, and low urinary pH*.

Careful handling of the chinchilla is advised as the liver may be more friable, leading to risk of organ rupture.

Treatment involves diagnosing and treating the underlying cause of the anorexia, correct fluid deficits, and give nutritional support through assisted feeding (20 ml/kg/feed, four times daily). *Monitoring the urine ketones and pH is a cost-effective way of monitoring response to treatment*.

General anaesthesia

Reducing risks associated with general anaesthesia starts before admission.

Chinchillas cannot vomit and need a near constant supply of fibre to maintain gut motility therefore they should not be starved before an anaesthetic. Owners are used to starving themselves or their dogs and cats prior to surgery, so we need to be clear when we book chinchillas in for surgery that they should not be starved. Advise owners to bring a 'packed lunch' of favourite and familiar foods.

Bonded cage mates should always be admitted, not only will this *reduce stress during the hospitalisation period, help protect the bond between conspecifics, it also has been shown to reduce healing times and increase coping ability for aversive or painful experiences* (Denommé and Mason, 2022).

Allow chinchillas at least an hour to acclimatise to the ward after travelling to the clinic to reduce stress levels prior to induction.

With only a few exceptions (bloat, or a foreign body), ill chinchillas should be fully stabilised (rehydrated, passing faeces and starting to eat) before an anaesthetic should be administered.

Pre-anaesthetic blood tests

The need for these is debated among vets. Personally, I advise they are an option but acknowledge in the young healthy individual the vast majority will be within reference ranges. The benefit is that it will give you a personalised reference range for this individual patient and as rodents are so good at hiding disease, occasionally we get unexpected results which may alter our plans. Blood can be taken from the cephalic vein or the lateral saphenous vein, but many chinchillas will require a GA for venipuncture. There is nothing wrong with aseptically dripping the blood from a needle into the collection tube. Biochemistry can be run 'in-house' using the same pre-op profiles used for dogs and cats. (See Table 7.1 for reference ranges.)

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Pre-emptive analgesia/prokinetics

Once admitted (and if not already on medications) administer meloxicam 0.6 mg/kg and/or another analgesic if appropriate depending on the surgical procedure, and metoclopramide 1 mg/kg. These can either be given orally or by subcutaneous (SC) injection.

Pre-medication

Whether I administer a pre-medication is dependent on the stress levels of patient and the surgical procedure being performed. *It is important to consider the stress of being handled and being injected. Rodents find injections very stressful and as they have small muscle masses IM injections are very painful and may need to be given in multiple sites depending on volume*. I think very carefully before I administer pre-medicants in rodents as I have to consider the type of surgery (and which analgesics will be required) and decide whether the stress and pain of the pre-med is worse than the mask induction. In my experience *mask induction while wrapped in a towel burrito results in a fast, seemingly minimal stress induction, whereas injections always result in vocalisations and obvious stress*. The drugs used for this are the same drugs we use in more familiar mammals. The doses can be found in Table 7.5.

Local anaesthesia for muscle splash blocks or infiltration

Chinchillas do not have an increased susceptibility to the toxic side effects of local anaesthesia, it is simply their small size that makes it much easier to accidentally overdose them.

Combining the drugs allows for greater effect with a lower recommended dose. Lidocaine has faster onset but a shorter duration of action (60–120 minutes) whereas bupivacaine has a slower onset of action but a longer duration (180–480 minutes). Mixing a maximum of 4 mg/kg of lidocaine with 2 mg/kg of bupivacaine in a syringe then diluting the mixture with saline to increase the volume of the solution and allow easier splash blocking (exactly what it sounds like). Drip the solution onto the wound in the muscles) or infiltrate an area. Pulling up the required dose and pre-diluting, prevents accidental overdose.

Induction

There are several anaesthetic protocols available for use in chinchillas. There is an argument to be made that the safest anaesthetic is the one you are used to.

My preferred inhalational gaseous anaesthetic is isoflurane. Reportedly, most rodents react more favourably to the smell of sevoflurane but in my experience, many dislike the smell just as much and in the UK, isoflurane is the only licensed gaseous inhalational anaesthetic for rodents.

Fox *et al.* (2016) found that *isoflurane resulted in a faster recovery and had no effect on appetite or faecal output whereas dexmedetomidine ketamine combination resulted in a self-limiting reduction of food intake and faecal output which lasted for up to 3 days*.

My preferred method of induction is to burrito the chinchilla and use a face mask. Have cotton buds to hand to swab to clear the mouth of food or saliva post-induction.

Allow four breaths of 100% oxygen (this is all that is required to fully saturate the blood with oxygen), then turn the vaporiser straight to 5% isoflurane. Initially the chinchilla may wriggle but it will quickly become induced (most are less than 40 seconds) with minimal stress. In the majority of cases the chinchilla will not have even passed urine or faeces (which frequently occurs due to stress and commonly passed when induction chambers are utilised).

Turn the vaporiser to 2.5% and assess anaesthetic depth (using the same reflexes we are used to in more familiar mammals, a moistened cotton bud can be used to check the blink reflex), adjusting the setting on the vaporiser as necessary.

The large cornea should be protected by applying an ocular lubricant.

Intubation

Intubation is challenging due to the palatal ostium but can be done visually using an otoscope or endoscope. I do not routinely intubate chinchillas.

Maintenance

I use a T-piece circuit, with an oxygen flow of 1.5 l/min. Isoflurane 2–4% as required.

Monitoring

Monitoring the anaesthetic is the same as for more familiar species. Multiparameter monitoring is possible. MAI Animal Health make Vetcorder Portable Monitor which is suitable for all species, including dogs and cats, for under £1,000. It can record: an electrocardiogram (ECG), heart rate, pulse oximetry (placed on tongue or paw), and rectal temperature. If recording an ECG rather than using

Table 7.5 Premedicant doses.

Drug	Dose and route	Reversal
Midazolam	1–2 mg/kg IM SC	Flumazenil 0.1 mg/kg SC
Midazolam + butorphanol	0.2–0.5 mg/kg + 0.2–0.5 mg/kg IM	Flumazenil 0.1 mg/kg SC
Medetomidine + butorphanol	0.05–0.1 mg/kg IM + 2 mg/kg IM	Atipamezole 0.5–1.5 mg/kg IM SC

Alligator clips (which can traumatise the skin), insert small gauge needles through the skin (25g) and attach the clips to the needles.

If that is outside the practice's budget, a soft, flexible tipped rectal thermometer can be used to monitor rectal temperature. Hypothermia is a major risk factor in anaesthetic deaths. A human foetal Doppler can be purchased online (I used eBay) for around £50, ensure you request the 8 mHz probe rather than the standard 2 mHz one. An infant stethoscope can be used to monitor heart rate if the Doppler is not an option. Respiratory rates can be counted and pulse oximeters can be used from other species but they may struggle to get an accurate reading due to the high heart rate (200–350 beats per minute in chinchillas).

Theatre

Chinchillas have a high surface area to volume ratio which means they lose heat rapidly. The resulting hypothermia can cause fatalities. Keeping the ambient temperature in theatre high, wrapping the extremities in bubble wrap, placing a towel on the operating table, and using a plastic drape (also allows visualisation of your patient) are all easy ways to help mitigate heat loss. Minimise the number of times the door is opened as each time it happens the ambient temperature in the room will drop. *Hypothermia leads to prolonged recovery and increased risk of death*.

Positioning

Chinchillas are diaphragmatic breathers. Their small thoracic cavity in comparison to their large abdominal cavity can make breathing more difficult due to the weight of the abdominal organs sitting against the diaphragm. Placing a folded drape or face cloth under the thorax elevates the thorax and allows the abdominal organs to move away from the diaphragm. If the chinchilla is not intubated, ensure the head and neck are straightened to allow for easier breathing.

Surgical preparation

To conserve body temperature, minimise the surgical clip. Only one agent is used for surgical scrub, either chlorhexidine or an iodine-based skin disinfectant depending on the surgeon's preference. We do not use a surgical spirit-based spray as during evaporation this causes loss of heat. Using warm water for the surgical scrub will help reduce heat loss.

Transparent, plastic surgical drapes are ideal as they allow visualisation of the patient while conserving body heat. The required surgical window can be cut into the drape and a surgical spray adhesive is used in lieu of towel clips.

EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) can be applied to the wound postoperatively if no other local anaesthetic infiltration has been performed in the skin. This helps with the immediate post-operative skin pain as most rodents show the most overt signs of pain 12 hours postoperatively. Since I have started using this technique, I have seen a faster return to eating on recovery.

Recovery

If used, medetomidine can be reversed with 0.5–1.5 mg/kg atipamezole SC, reversing the medetomidine will also reverse the analgesia provided by medetomidine but that is not reason enough for not reversing.

Maintain the chinchilla on oxygen, and if intubated, extubate when the jaw tone increases. Shivering thermogenesis is a sign of recovery from anaesthesia. Continue to monitor while in an incubator or a warm room (ideally around 26°C (78.8°F)) until up and moving then the temperature of the room can be reduced to 20°C (68°F).

Syringe feed (10–20 ml/kg Oxbow Recovery, Science Recovery or EmerAid Herbivore) once the chinchilla is fully conscious and monitor faecal output.

Advise the owner to monitor food intake and faecal output. If the owner is unable to syringe feed at home, then they must bring the chinchilla back to the practice as soon as possible for admission if they will not eat at home. In the vast majority of cases with good analgesia this will not be an issue (see Table 7.3).

Dental disease

Our current understanding of dental disease is that it is a *common condition in captive chinchillas* but studies have shown it is rare in wild chinchillas. *Captivity, most likely diet, is implicated in the development of dental disease*. Not all owners are aware that chinchillas' teeth grow continuously throughout life.

Clinical signs

Clinical signs associated with dental disease vary depending on the severity and can include: weight loss, decreased food intake or anorexia, change of food preference, reduction in faecal output or size, drooling (known as slobbers) causing a wet chin, poor fur quality, crusting around mouth or forepaws, epiphora (due to pain or impingement of the nasolacrimal duct (NLD)), flattening of fur periocularly, tooth grinding, secondary bacterial conjunctivitis or proptosis due to retrobulbar abscessation secondary to retrograde growth of maxillary molar apices.

Diet

The mainstay of the chinchillas' diet is hay. Lowland pasture hays contain higher energy, lower fibre, and lower phytolith (therefore less abrasive) contents compared to the low energy, high fibre, high phytolith, mountain vegetation which forms the diet chinchillas evolved to eat. Coupled with the fact that hay grown under favourable conditions is clean whereas wild diet is contaminated with

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soil dust that is highly abrasive which is why *35% of apparently healthy captive chinchillas have longer molars than their wild counterparts*.

Diet is believed to contribute to dental disease through inappropriate wear of the teeth, a wider vertical movement leading to abnormal pressures (pellets/nuggets), spikes forming in the mouth and over time abnormal pressures resulting in retrograde unerupted crown growth, being too high in phosphorus, and hypovitaminosis C. High-quality diet may also lead to full mineralisation of teeth, slowing their wear.

Time taken to educate owners on correct diet and husbandry at their routine check-ups can help reduce the risk of dental disease.

Examination of teeth

Careful examination of the incisors to assess the length, shape, occlusal surface and enamel quality, and colour is important. The normal colour of chinchilla incisors should be yellow/brown. White incisors may be associated with a diet deficient in calcium or vitamin D.

Incisor malocclusion is not as common in the chinchilla as it is in the rabbit and may be associated with trauma. Subtle signs such as diagonal wear or horizontal ribbing of the enamel should increase our index of suspicion for dental issues. Malocclusion can be obvious to us but missed by the owners.

Client discussion

An honest, open, and frank conversation is needed. *Not all owners will be emotionally, financially or time invested enough to proceed with treatment*. Owners need to be prepared to expect repeated dental procedures approximately every twelve weeks, with the second dental procedure occurring in a shorter time frame while the chinchilla becomes used to chewing in a more normal pattern.

In some cases, euthanasia is the best option for that individual. Euthanasia is not a welfare concern (a difficult concept for me to understand as a young vet), however *existing in chronic pain and substandard husbandry is a massive welfare issue.*

In the vast majority of cases there is no cure, management of this lifelong condition with repeated dental surgeries, long term analgesia and husbandry reviews will be required. This requires dedication and financial input from the owner. Some owners may not be able to cope with this.

Some early cases of dental disease without root changes may be curable after the first dental procedure, with dietary corrections so that they are mainly fed on: fresh growing grass, hay and dried grass and addressing of any underlying medical issues (e.g. pain). This type of diet encourages prolonged chewing in a natural chewing pattern. The high silicate phytoliths in the grass promotes normal wear of the teeth. *Any elderly chinchillas presenting with dental disease for the first time should be fully examined for skeletal pain*.

Once the chinchilla is stable enough (passing normal faeces but not necessarily eating on its own) I advise general anaesthesia, radiographs of the skull, full exploration of the oral cavity and dental surgery. *Radiographing the skull is compulsory if the owner wishes me to perform a dental*. Radiographs are required to stage the disease. Computed tomography (CT) scanning will give superior information and images compared to radiographs but not all owners are in the financial position to opt for CT scanning, so I work mainly on good quality radiographs.

I radiograph the first time and how frequently I repeat the radiographs depend entirely on the stage of the disease, mild 'root' changes I will repeat the radiograph in 6 months (then in a year if still requiring dental procedures but minimal 'root' changes), severe 'root' changes I recommend repeating the radiographs at 3 months then 6 months. Ongoing frequency is dependent on if there is progression of 'root' changes and clinical judgement is utilised.

Apical cheek tooth elongation (technical term for 'root' disease) causes pressure on nerve endings resulting in pain. Should the 'root' disease progress, the maxillary roots may grow into nasal cavity/sinus, occlude the NLD, and or grow into the orbital foramen. The mandibular roots may grow through the ventral mandibular cortex palpable as hard bumps on the ventral medial border of the mandible. *Without knowing the condition, length and shape of the maxillary and mandibular 'roots' we cannot give information on prognosis or know if long term analgesia is required*.

Without this information, chinchillas with moderate to severe 'root' disease will be in chronic pain, will not chew properly and will require much more frequent dentals. With appropriate analgesia, chinchillas will cope better with moderate/severe 'root' disease.

Dental radiographs

Once anaesthetised (see General anaesthesia section) I will take a right lateral and a dorsoventral skull radiograph as routine. *Good positioning is required for the radiographs to be of diagnostic use*. Personally, I do not often take oblique views unless I am worried about dental abscessation and need further assessment. If I am worried about dental disease being secondary to temporomandibular joint arthritis, I will take a cranio-caudal view of the skull (Figure 7.4)

Severe root disease is easy to see (Figure 7.5) but mild or subtle changes can be harder to appreciate if you do not spend all day looking at chinchilla skull radiographs. I utilise three lines on the lateral view and three lines on the dorsoventral view drawn onto the radiographs to help with assessing dental disease (Boehmer & Crossley, 2009) (See Figures 7.6a & b).

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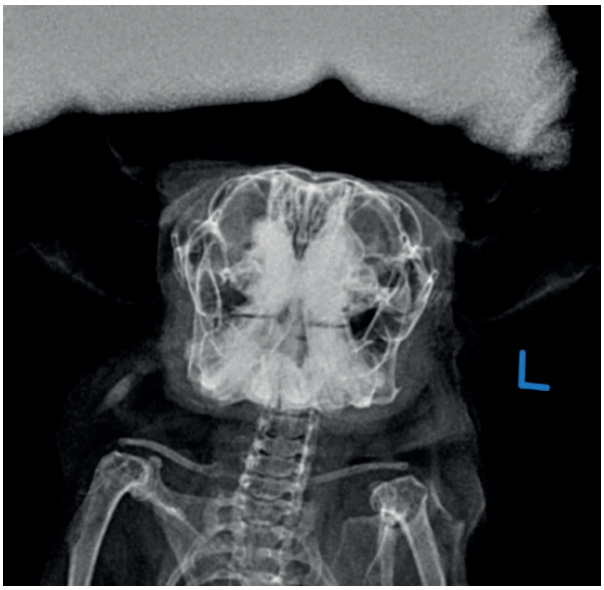


Figure 7.4 A cranio-caudal view of the skull. I use a sandbag behind the head to steady the skull.



Figure 7.5 A radiograph of a chinchilla with severe dental disease.

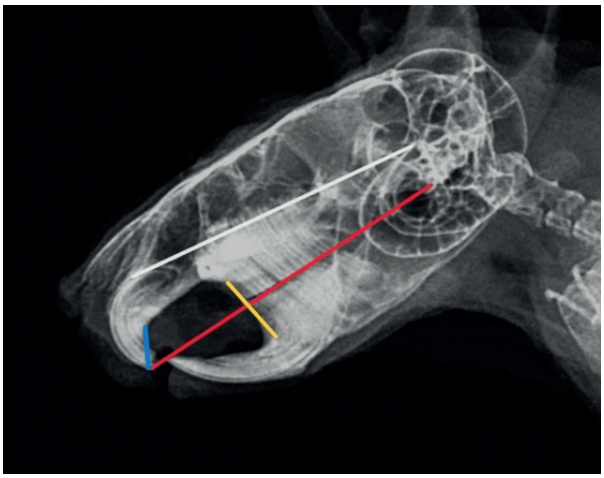


Figure 7.6a Close to normal lateral skull radiograph chinchilla with objective lines drawn.

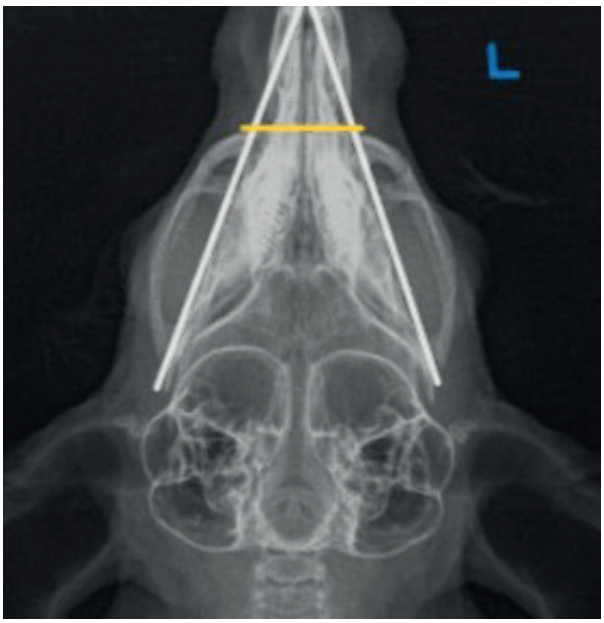


Figure 7.6b A dorsoventral skull radiograph with the objective lines drawn.

Lateral skull radiograph (Figure 7.6a)

Correct positioning is vital, in a true lateral skull radiograph both mandibles are overlying each other as are the tympanic bullae. Rotation of the skull, meaning the bodies of both mandibles are visible is not a lateral view, it is an oblique and the lines cannot be drawn on the radiograph.

Examine the supporting bone and periapical structures of each tooth. The periapical region of each tooth is where the germinal tissue sits. This area can be large and is radiolucent, surrounded by a thin radiopaque lamina dura which should be visible along the entire length of the reserve crown of all teeth. The apex of the maxillary incisors extends to two-thirds of the length of the diastema and the apex of the mandibular incisors extend to and lingual to cheek tooth two or three.

One line (drawn in white) is drawn from the dorsal aspect of the upper incisor to the notch of the tympanic bulla. There should be *minimal to no sign of 'roots' above this line in the normal chinchilla. Unfortunately, it is uncommon in pet chinchillas to see no abnormalities*.

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The blue line is drawn along the angle of the palatine bone holding the upper incisor to the cranial border of the upper incisor, this line shows where the tip of the upper incisors *should* be.

Another line (this time in red) is drawn from the tip of the upper incisor (end of the blue line), to about three-quarters of the height of the bulla (height measured from the base to the notch of the tympanic bulla). This red line runs straight along the occlusal line of the cheek teeth. The *occlusal line should be near horizontal and run parallel to the ventral border of the mandible and frontal bones*. The interproximal spaces are tight in the normal chinchilla.

A line drawn vertically (yellow line) on the rostral borders of the maxillary and mandibular arcades should be level with each other. *Mismatch of the cheek teeth arcades is a very reliable sign of significant dental disease in chinchillas*. There is often *caudal displacement of the mandibular arcade in cases of dental disease* (see Figures 7.7a & b).

Dorsoventral skull radiograph (Figure 7.6b)

The first line drawn (white lines), connects the medial border of the maxillary incisor to the most caudolateral part of the ipsilateral mandibular ramus. The second line is exactly the same but on the contralateral side. *No part of any tooth should be visible outside of these two lines*.

The third line (yellow) is drawn horizontally through the points where the first lines cross the maxillary zygomatic arches which shows the most rostral extent of clinically normal cheek tooth apices.

Figures 7.7a–c show the same lines drawn on radiographs of chinchillas with dental disease.

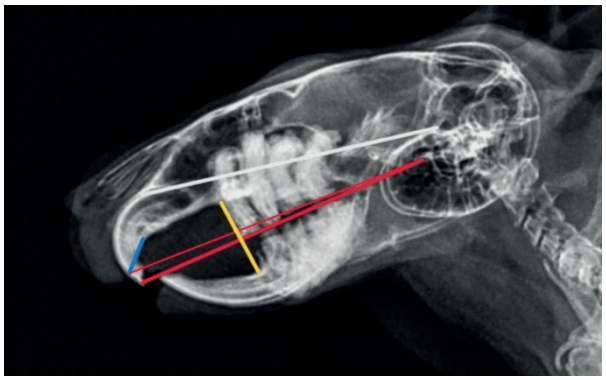


Figure 7.7a Lateral skull radiograph of a chinchilla with dental disease with the objective lines drawn. The thin red line is where the occlusal plane should be, the thick red line is drawn as an example of what can happen if we fail to take into account what length the upper incisors should be, that is, if we forget to draw the blue line.

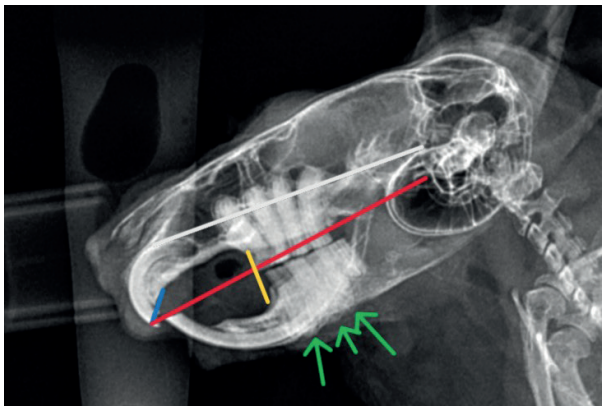


Figure 7.7b Another lateral radiograph of a different chinchilla with dental disease with the objective lines drawn. The green arrows are showing root penetration of the mandible.



Figure 7.7c Dorsoventral radiograph of a chinchilla with dental disease showing the objective lines drawn. Personally, I find the lateral more informative.

Oral examination under GA

Once I have radiographed, I will perform a full oral examination.

In the normal chinchilla:

The dental formula is: (2×) I 1/1 C 0/0 P1/1 M3/3

The maxillary incisors are 0.6 cm long and the mandibular approximately 0.9 cm. There should be a horizontal occlusal surface. The enamel is a yellow brown colour.

Cheek teeth: each mandibular tooth occludes with one maxillary tooth and works as a functional unit and forms a continuous grinding surface due to the lack of space between teeth. The healthy teeth are cream in colour.

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The mandible is wider than the maxilla and at rest the cheek teeth are not in occlusion. Chewing is performed mainly in a rostrocaudal movement with very little lateral movement.

All arcades should be 1 mm above gingival margin and the occlusal plane is almost horizontal. *The mouth is symmetrical.*

Chinchillas with dental disease are likely to have asymmetrical dental arcades (Figure 7.8). The maxillary molars flare towards the cheek and damage the buccal mucosa (painful) while the lower arcades grow lingually into the tongue.

Chinchillas will suffer from pain associated with retrograde root growth. This may be palpated as hard bumps on the ventral border of the mandible or in the pre-orbital fossa. Frequency between dentals could be as frequent as every twelve weeks but owners should be warned that the first few dentals may be closer together as the chinchillas' muscles adjust to chewing and adjust from being stretched.

The current belief is that dental disease in chinchillas is caused by improper wear due to altered chewing and inappropriate diet, calcium deficiency, vitamin D deficiency, vitamin C deficiency, and temporomandibular joint disease.

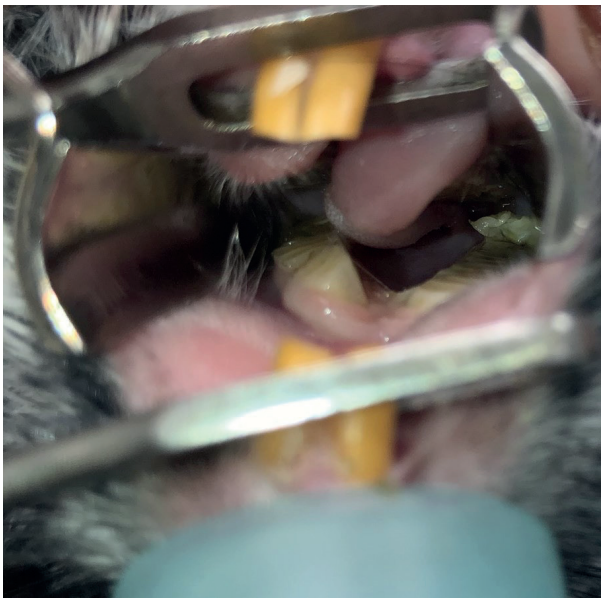


Figure 7.8 Showing the asymmetrical wear of the upper arcades in a chinchilla with dental disease.

The aim of dental surgery is to restore the correct shape, length, and symmetry of all the dental arcades therefore allowing normal functioning of the teeth.

Incisor malocclusion

Incisor malocclusion (is not as common in chinchillas as in rabbits) can be managed with repeated trimming of the teeth (this can be done consciously in many patients) using a diamond cutting disc (use of guard advised) or a dental drill bit with the water turned off. The normal incisor eruption rate is approximately 1.25 mm a week but this can increase if they are unopposed in malocclusion.

It should be remembered that chinchillas chew mainly in a rostrocaudal motion; if the incisor malocclusion leads to the upper incisors sitting behind the lower incisors it prevents the normal rostrocaudal chewing movement (Figure 7.9). Some individuals will find frequent trimming stressful, therefore if the incisors are non-functional, or are preventing normal chewing, extraction is recommended.



Figure 7.9 Although incisor malocclusion is not common in chinchillas, it is important to realise the incisor malocclusion may interfere with normal chewing movement.

Incisors should never be clipped with molar cutters or nail cutters. This practice is outdated and barbaric. Due to the conformation of the enamel, clipping causes the enamel to shatter leading to vertical fractures, pulpitis and potentially apical abscessation. There is no excuse for clipping incisors.

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Tooth resorption

Tooth resorption is common and can lead to the loss of clinical crowns, food and fur can become impacted within the widened interproximal space which will often then lead to periodontal disease. Periodontal disease can be present without tooth resorption.

Periodontal disease

Chinchillas are exceptionally prone to periodontal disease. Crossley (2003) found an incidence of 63% of the chinchillas examined had periodontal disease. *It is imperative that every chinchilla suspected of dental disease also have a full oral assessment for periodontal disease*. Some authors maintain oral endoscopy is the best way to fully examine the mouth even under anaesthesia.

Personally, I make a point of examining each tooth for mobility and probing for gingival pockets. *Left untreated, periodontal disease will progress to bone and tooth resorption, be a chronic source of oral pain, leading to decreased food intake, further weight loss and increase the risk of hepatic lipidosis or ketosis developing.*

There will be pockets filled with food, hair, and debris around the teeth (Figure 7.10). *It is important that all the material is removed*, I use small atraumatic forceps to grasp the hair/debris and pull it out. The pockets are then cleaned with dilute chlorhexidine or flushed with 2% hydrogen peroxide. Some authors prefer hydrogen peroxide because the foam is easier to contain and less risk of aspiration. Large or infected pockets benefit from being packed with Doxirobe Gel.

In some cases, systemic antibiotics will be required. Ideally antibiotic choice should be guided by bacteriology results but empirical choices include parental long-acting penicillin G benzathine/procaine combination 50,000 IU/kg SC q3–5days, or doxycycline 5 mg/kg q12hrs plus metronidazole 15–20 mg/kg q12hrs for 10–14 days. Other antibiotic choices that are safe in chinchillas and have good activity against anaerobic bacteria are: chloramphenicol and azithromycin.

Dental caries

Caries are extremely common in chinchillas (52% of chinchilla skulls examined; Crossley 2003) and are usually brown or black in colour caused by areas of bacterial decay (Figures 7.10 and 7.11). Sugary treats and low abrasive foods may lead to increased bacterial plaque formation and decreased saliva production (due to reduced time chewing) which is thought to predispose chinchillas to caries.

The diseased area should be removed using a ball tipped diamond bur or a rose bud rodent drill bit drilling until a healthy crown is evident. Wiping the teeth with cotton buds wet with diluted chlorhexidine can be used to clean tooth surfaces and remove plaque. Owners should be advised to stop feeding sugary treats and to increase the hay and dried grass component of the diet.



Figure 7.10 Note the discolouration of the molars which is due to carie development (unusual level) and the wide space between the lower right mandibular arcade molars which has a piece of hay protruding from it.



Figure 7.11 Caries are more often more visible on the occlusal surface such as seen in this photo. The level of caries seen in Figure 7.10 is unusual in my experience. Note the buccally flaring spike on the right upper arcade.

Gingival hyperplasia

Coronal overgrowth of the cheek teeth frequently stimulates gingival hyperplasia in chinchillas. This is problematic for two reasons: *maxillary molar buccal spikes may be masked*

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by the overgrown gingiva preventing proper treatment, and even reducing the corona length to 1 mm above gingival level may not be sufficient if the gingival level is artificially long.

It may be difficult to appreciate that there is gingival hyperplasia. *Sliding the dental spatula along the buccal aspect of the maxillary arcades* can help to ensure there are no spikes present. *Repeating the lateral skull radiograph after coronal reduction* will show whether sufficient length has been removed, if the cheek tooth length is still too long on the radiograph despite being reduced to 1 mm above the gingiva, two options are available: Owners can be advised that a repeat dental will be necessary in 4–6 weeks, which should give the gingiva time to naturally regress or electrocautery can be used to carefully resect the excessive gingiva, allowing further reduction in coronal length. The cheek teeth should not be taken down below gingival level as it is painful and will prevent chewing until the teeth grow into occlusion.

Dental surgery

Extraction of the Incisors

Owners worry that their pet cannot eat without their incisors. Counselling owners to consider incisors as scissors, that chinchillas hold their food in their front paws and longer term cutting of grass and fresh foods into bite sized portions is all that is required. *If incisor extraction is performed, all the incisors should be extracted*. Incisor extraction is performed using a pre-bent needle instead of a Crossley Luxator otherwise using the same technique as in guinea pigs (see Chapter 3).

Cheek teeth

RISKS ASSOCIATED

Branches of the facial artery are located in the buccal mucosa, lateral to the last maxillary molars. Trauma to the overlying soft tissue by handheld instruments can lead to death if bleeding is not quickly stopped. Owners should be counselled that while these complications are rare, a fatal bleed can quickly occur. Collaclot is a collagen sponge which will quickly lead to haemostasis. It is inexpensive, has a long shelf and in these situations can be lifesaving.

EQUIPMENT NEEDED

Straight dental handpiece with rodent drill bit, rose bud or bar tipped rodent drill bit, diagnostic pen torch with speculum (or head torch and an oral speculum), mouth gag, cheek pouch dilator (or Thudicum nasal speculum), curved tweezers, small dental probe, moistened cotton buds.

Owing to the small amount of available space in the oral cavity, I do not intubate for dental procedures.

I prefer to position chinchillas in dorsal recumbency for this procedure with their thorax raised on a towel. Positioning like this allows me better access and visualisation of all four arcades.

There are many different types of cheek pouch dilators on the market and in my experience, the springs of many of them are too strong, resulting in the instrument 'pinging' out of the mouth or overstretching the cheeks which will be painful. Care should be taken not to further damage any friable oral mucosa when placing the dilators.

It is important when placing the oral gag over the incisors that it is opened slowly and stop opening as soon as there is any tension felt. *Over opening the mouth will cause damage to the temporomandibular joint*.

I use a diagnostic pen torch with Perspex speculum (Eickemeyer) as both my light source and my speculum/soft tissue guard. The tapered tip of the speculum provides good illumination even to the caudal teeth (Figure 7.12).

Each tooth should be checked for mobility and probed for pockets or periodontal disease. Run the torch speculum along the buccal aspect of the maxillary arcades to check for hidden spikes. *Chinchillas often have a 'sneaky' spike on the most caudal mandibular molars which are sometimes felt rather than seen initially*. Periodontal diseases and caries should be treated as above.

Using the dental drill with a rodent drill bit and soft tissue guard, held in a pen grip (allows much greater control over the drill), to remove any spurs and to reduce the coronal length (if required) in all arcades. Any crowns that are very loose should be gently pulled out. Remove tooth dust

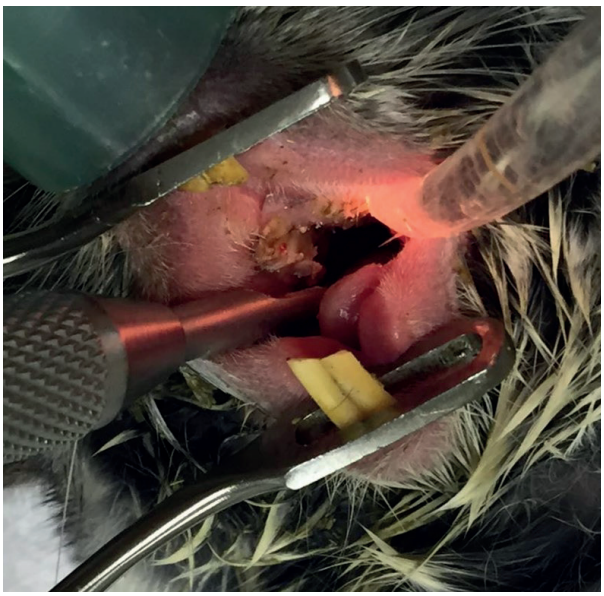


Figure 7.12 Use of the diagnostic pen torch to move soft tissue and as a light source while using the dental drill.

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with dilute chlorhexidine moistened cotton buds, ensure both upper arcades are symmetrical and that both lower arcades are symmetrical.

Hand rasping kits are still available. *Only the smoother, diamond rasp should ever be used*. Hand rasping takes much longer to perform and there is a higher risk of perforating the arteries. *Caution should be exercised as any kind of rasping can potentially lead to damage of the periodontal ligament, loosen the molars, and exacerbate dental disease.*

Repeat the lateral skull radiograph to ensure enough coronal reduction has been performed (see under Gingival hyperplasia).

Post-dental care

Assist feed on recovery from anaesthesia. Assisted feeding may be required for a few days (often longer than would be needed in rabbits), allowing the jaw muscles to readapt to closing the mouth and to allow occlusal surfaces of the cheek teeth to wear back into function. Monitor faecal output and urine can be tested to check pH and for ketones. Dipstick analysis of urine is a cost-effective way of ensuring adequate nutrition is being given.

Continue analgesia for at least a week after the procedure although cases with 'root' disease will require long term analgesia. Dietary assessment is required, and owners should be advised we aim to eventually reduce the time between dental surgeries to approximately three months. *More frequent dentals may indicate that our analgesia is inadequate, especially neurogenic pain that can be caused by apical elongation pressing on nerves*.

Dental abscesses

Periapical infections and dental related abscesses are an uncommon complication of dental disease in chinchilla although they can be frustrating and difficult to treat.

Not every facial abscess is related to dental disease, good quality radiographs of the skull and in an ideal world CT assessment would be utilised to gain the most information regarding the structures involved. Not every owner will be in the position to opt for CT scanning therefore I mainly work on good quality radiographs.

'Gold standard' is a term I never use. It can cause shame in owners if they cannot afford to give their beloved pet the 'gold standard' treatment. I use the term 'textbook standard' as it removes the monetary perception. For many people their go-to emotion when they are shamed or fearful (many owners fear our negative perception) is anger. This will increase our stress levels, the owners stress levels and ultimately everyone loses. Small changes in terminology can make a big difference to the entire outcome in many cases.

The site of the abscess will ultimately determine all the views taken but multiple views and oblique radiograph should be performed. Prognosis is variable depending on location and number of arcades involved. The chinchilla should be fully stabilised before surgery which is performed in a similar manner to rabbits (see Chapter 2, Abscess section).

Caseous lymphadenitis due to *Streptococcus equi subsp zooepidemicus* has been recorded in chinchillas, which on initial presentation could mimic a dental abscess. *Streptococcus equi subsp zooepidemicus* tends to form chains of Gram-positive cocci on cytology. The antibiotic in the case report was changed from azithromycin to trimethoprim-sulfamethoxazole (30 mg/kg q12hrs) on the basis of culture and sensitivity results and lack of clinical response to the azithromycin, which lead to the complete resolution of the abscess post-drainage (See Table 7.6 for antibiotics commonly used in chinchillas).

Table 7.6 Antibiotics commonly used in chinchillas.

Drug	Dose
Azithromycin	30 mg/kg PO q24hrs
Ciprofloxacin	10 mg/kg PO q12hrs
Chloramphenicol	30–50 mg/kg SC, IM, IV, PO q8–24hrs
Doxycycline	5 mg/kg PO q12h, long-acting formulation 50mg/kg q7days SC
Enrofloxacin	10–20 mg/kg PO SC q12hrs (IM painful and may result in sterile abscess/ muscle necrosis)
Gentamicin	5–8 mg/kg divided q8–24hrs SC, IM
Marbofloxacin	5 mg/kg PO q12–24hrs
Metronidazole (may cause a self limiting reduction in food intake in some chinchillas, monitor food intake and faecal output carefully whilst on treatment)	15–20 mg/kg PO q12–24hrs
Oxytetracycline	50 mg/ kg PO q12hrs
Penicillin G benzathine/ procaine long-acting combination	50,000 IU/kg SC q3–5 days *Do not use orally.*
Silver sulfadiazine cream	Topical q12–24hrs
Trimethoprim sulfamethoxazole	30 mg/kg PO q12hrs
Tylosin	10 mg/kg PO, SC q12hrs

Infection with *Streptococcus equi subsp zooepidemicus* should be included as a differential for chinchillas presenting with subcutaneous abscesses.

Heat stroke

Chinchillas are adapted to a low humidity (30–40%) environment and are better suited to lower temperatures. Environments with ambient temperatures of over 26.7°C (80°F), especially if they also concurrently have high relative humidity of >50% can rapidly result in heat stroke. Obesity may predispose to heat stroke.

The chinchilla may present recumbent or ataxic, with rapid breathing, bright red mucous membranes, and thick stringy saliva. The blood vessels on the pinnae will be prominent and there may be blood froth at the nose and mouth.

Treatment involves cooling of the animal with the use of fans and tap water, and IV fluids should be given. Unfortunately, most will deteriorate after an initial improvement so owners should be advised the prognosis is guarded to poor.

Respiratory disorders

Chinchillas are obligate nasal breathers and the respiratory rate (RR) is quoted as 45–80 breaths per minute. Their tendency to become highly stressed when observed is likely why there is such a wide range, *as under general anaesthesia the rate is usually lower than the lower reference range limit*. Diaphragmatic hernias have been recorded in chinchillas.

Clinical signs may frequently be nonspecific and include hunched posture, decreased activity levels, unkempt coat, and reduced appetite.

Upper respiratory disease

Nasal discharges are uncommon in chinchillas, but may be associated with dental disease, nasal foreign body, rhinitis, sinusitis, megaoesophagus (single case in literature), cleft palate, or lower respiratory disease (RD). Upper RD may be seen more commonly in young, stressed, and immunocompromised animals. Kits are particularly prone to developing foreign body rhinitis if cornstarch is added to their dust baths.

Predisposing factors are high humidity, overcrowding, poor ventilation, constant drafts, recent transport, contact with infected conspecifics, contact with dogs, cats or rabbits which may serve as carriers of pathogens (see below).

Clinical signs are similar to those in more familiar species: sneezing, dyspnoea, serous to mucopurulent nasal discharge, matting of fur on forelegs.

Upper respiratory infections may progress to the lungs. Subsequent septicaemia can develop, leading to systemic dissemination and secondary otitis media/interna, or meningoencephalitis.

Treatment

Treatment will vary depending on clinical presentation, see section on otitis media/interna for specific treatment advice.

Culture and sensitivity via deep nasal swabs are advisable due to the high risk of multi-drug resistance making empirical antibiotic choice challenging. Bromhexine can be used as a mucolytic, advising owners that initially their pet may sneeze more as the discharge becomes easier to remove. Nebulisation with either F10 RTU or appropriate antibiotics guided by culture can be carried out twice daily for a minimum 15 minutes. See Table 7.7 for nebulisation doses. Analgesia such as meloxicam to reduce inflammation and associated pain and if necessary correct husbandry.

Lower respiratory disease/bacterial pneumonia

Pneumonia is uncommon in chinchillas. Bacteria that are commonly isolated from cases of pneumonia (in no particular order) include *Staphylococcus aureus*, *Streptococcus pneumoniae*, *S. pyogenes*, *S. zooepidemicus*, *Klebsiella pneumoniae*, *Bordetella bronchiseptica*, *Pasteurella multocida*, and *Pseudomonas aeruginosa*.

Clinical signs include depression, tachypnoea, dyspnoea, and if severe, open mouth breathing, fever, anorexia, dehydration, lethargy, and sudden death. Peracute septicaemia is seen more commonly with Gram negative infections.

Acute forms of suppurative to fibrinous bronchopneumonia are often seen in conjunction with suppurative rhinitis, otitis media, and meningoencephalitis. In chronic cases, subcutaneous abscesses due to haematogenous spread have been described.

Table 7.7 Nebulisation doses.

Drug	Dose (most nebulisers will hold 10 ml)
Saline	10 ml good for hydration of respiratory tract
Acetylcysteine (mucolytic)	10–20 mg per 1 ml saline (higher doses may be associated with bronchospasm)
Bromhexine (mucolytic)	0.15 mg per 1 ml saline
F10	1:250 or ready to use (already prediluted)
Aminophylline	3 mg per 1 ml saline
Amikacin	5 mg per 1 ml saline
Doxycycline	13 mg per 1 ml saline
Enrofloxacin	10 mg per 1 ml saline
Gentamicin	5 mg per 1 ml saline

Cardiac disease should be ruled out first then systemic antibiotics, oxygen therapy and nebulisation of antibiotics can be initiated. The prognosis is poor in chronic cases.

Radiographs

Thoracic radiographs can help differentiate respiratory disorders of cardiogenic aetiology from those of primary RD. Good positioning is critical for interpretation which is the same in all species.

Standard views include right and left lateral, dorsoventral, and ventrodorsal.

On right lateral views the cardiac silhouette is often more oval; while it may appear relatively smaller on the left lateral view. On the lateral views, the large amount of intrathoracic fat that is often present will increase thoracic opacity.

The ventrodorsal view is indicated for evaluation of: cranial and caudal mediastinum, the caudal vena cava, accessory lung lobe, and pleural effusions.

Cardiac disorders

The resting heart rate of normal chinchillas is 200–300 bpm. Benign murmurs are common in chinchillas.

Dilated cardiomyopathy, congenital septal defects and valvular disease have all been reported in chinchillas.

Cardiac disease may present with nonspecific signs such as: general weakness, loss of activity, weight loss, and dyspnoea. These nonspecific signs of cardiac disease are common in more familiar species too.

The clinical exam should be performed gently and quietly as stress may cause decompensation and worsening of clinical signs. Consider administering midazolam 0.2–0.5 mg/kg IM to fractious animals. Mucous membrane colour is best evaluated either at the conjunctiva or the gingiva in the area of the incisors. The latter is also a good site for checking the capillary refill time.

Vertebral heart score

The vertebral heart score (VHS) is measured the same way as in dogs. The long axis (LA) is measured from the carina to the heart apex; the short axis (SA) is measured perpendicular to the LA and spans the width of the heart at its widest. Both axes are then independently measured against the thoracic vertebrae starting at the cranial border of T4, the sum of both axis measured in vertebrae provides the score.

The *reference range for VHS* in the chinchilla is: 7.5–10.2.

> Remember that normal size of the cardiac silhouette does not rule out cardiac disease as for example, hypertrophic cardiomyopathy often results in minimal alteration of the external diameters of the heart.

Treatment

There are no reports in the literature about treatment of heart disease in chinchillas. I have used the same drug doses as per guinea pigs successfully in the clinic.

Ophthalmology in chinchillas

The approach to ophthalmology in chinchillas is similar to that in dogs and cats, they are just eyes. The most common ophthalmic conditions seen in chinchillas are lens disease, corneal disease, and conjunctivitis.

Chinchillas have prominent eyes, a large cornea, and lack a third eyelid which may be why they are prone to corneal ulceration. They have a vertical slit shaped pupil which is semi-constricted in ambient lighting. The pupil responds poorly to attempts to dilate with drugs.

There is a medial caruncle present and 30% of healthy chinchillas have associated trichiasis which has been suggested as a normal variation as no clinical signs have been associated with it (Müller *et al.*, 2010).

All chinchillas (100%) have a pupillary light response but *97% of chinchillas lack a menace response*. They have a low blink rate (2–4 blinks per 10 minutes), which will increase when they are excited, and a low Schirmer Tear Test reading (1.07 mm/minute compare to rabbits which are 5.3 mm/minute). The normal intraocular pressure has been recorded as 17.71 +/– 4.17 mmHg. Of the bacterial flora in healthy chinchilla eyes 94% are Gram positive. *Flushing of the NLD is not feasible* in chinchillas due to the difficulty in visualising the entrance of the duct.

Conjunctivitis

Conjunctivitis is common in chinchillas and has various causes. Historically, most cases of conjunctivitis were attributed to (especially if there were no concurrent signs of RD): irritation from dust bathing (or inappropriate sand choice), poor cage ventilation leading to excessive levels of ammonia, or obstruction of the NLD due to dental disease. Fluorescein should be applied to check for concurrent corneal disease.

A full husbandry review will reveal inadequacies in ventilation or sand choice. Decreasing the frequency or duration of sand bathing may be appropriate and, in some cases, topical application of a lubricant may be beneficial. *If epiphora alone is present, radiography should be performed to look for underlying dental disease (increased tear production is a reflex response to mucosal or bone pain)*, although secondary bacterial infection may complicate the presentation. Some chinchillas may present with epiphora due to acute flare ups of chronic skeletal pain in my experience.

Bacterial conjunctivitis

Secondary bacterial conjunctivitis is commonly seen, with 62% of isolated bacteria being Gram negative. Of these isolates 50% were *Pseudomonas aeruginosa* and 27% were *Staphylococcus* sp. As mentioned above, the majority of

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cases of *P. aeruginosa* conjunctivitis had concurrent respiratory signs.

Primary bacterial conjunctivitis is uncommon in chinchillas but has been reported associated with *Staphylococcus aureus*, *Streptococcus* sp, and *Pseudomonas aeruginosa* (often has concurrent respiratory signs).

Chinchillas that present with acute conjunctivitis (duration 1–3 days) are much more commonly affected by Gram negative bacteria, and owners are more likely to present them sooner due to the severity of the clinical signs. Chinchillas affected by Gram positive bacteria tend to have milder clinical signs that will usually mean a longer duration before presentation by the owner. Owing to the longer duration, there is an increased incidence of periocular alopecia.

Although *P. aeruginosa* is the predominant bacterial species associated with bacterial conjunctivitis in chinchillas, culture and sensitivity is strongly recommended as most isolates of Pseudomonas display multi-drug resistance to commonly used antibiotics. Ozawa *et al*. (2017) found that 46.2% of Pseudomonas isolates were resistant to five or more antibiotics and only 36% of the *Pseudomonas* isolates were sensitive to enrofloxacin, whereas 77% isolates were sensitive to ciprofloxacin, and 88% of the isolates were sensitive to gentamicin.

Empirical treatment while awaiting culture and sensitivity results is strongly recommended in cases of suspect *Pseudomonas* due to the risk of *Pseudomonas* conjunctivitis developing into systemic illness and/or sepsis. *Pseudomonas* has been documented to cause enteritis, otitis, metritis, abortion, pneumonia, neurological signs, and sudden death. *Appropriate topical antibiotic choices are gentamicin and polymyxin B. If there are concurrent respiratory signs, systemic antibiotics are advised*.

Corneal disease

Corneal disease is common in captive chinchillas and should be investigated in a similar manner to more familiar species using ophthalmic exam and fluorescein should be applied with any lesions documented. Corneal lipid keratopathy is reported in chinchillas.

Corneal erosions/ulcers

The large, prominent corneal surface and lack of a protective third eyelid may predispose chinchillas to corneal trauma. *As for any species, owners should be advised that ulcers generally do one of three things: heal within a week, take months to heal, or lead to the loss of the eye*. It is important to manage owners' expectations during the initial consultation (Figure 7.13).

Corneal damage and keratitis secondary to trauma is commonly seen in chinchillas, and is often associated with blepharospasm, ocular discharge, and conjunctivitis. It is important to evaluate the palpebral reflex (use moistened cotton bud), and examine the ears, in all chinchillas with corneal damage as lack of the palpebral reflex secondary to facial nerve damage caused by otitis media has been reported. Exposure keratitis may be secondary to a retrobulbar abscess (secondary to dental disease) causing exophthalmos and an inability to fully close eyelids.

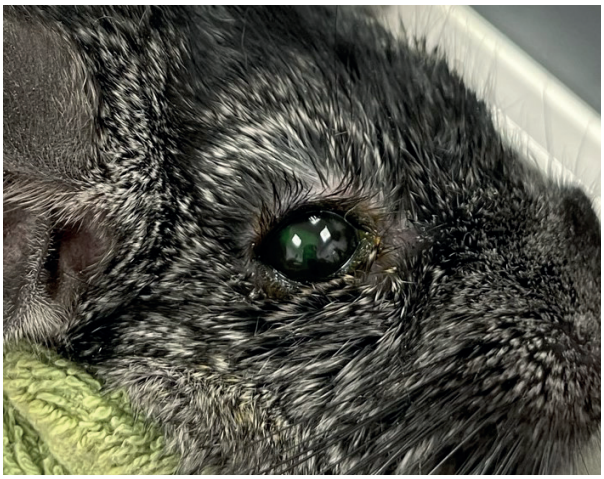


Figure 7.13 Fluorescein positive corneal erosion.

If secondary bacterial involvement is suspected (which is common) culture and sensitivity is recommended due to *Pseudomonas* sp. tendency to display multi-drug resistance to commonly used antibiotics.

Treatment

Analgesia such as meloxicam should be provided until the cornea heals. Humans find corneal ulceration excruciating.

Topical lubrication is also advisable q6–12hrs depending on the owner's ability to handle the chinchilla. As always *it is important to balance the potential stress caused by medicating with the benefit provided*.

Topical antibiotics using a broad-spectrum eye drop such as a triple antibiotic drop containing polymyxin b/neomycin/gramicidin, or as guided by culture and sensitivity. A Gram stain can be performed and if Gram negative rods or cocci are identified, ciprofloxacin or ofloxacin may be preferred and can be adjusted if necessary once culture results are obtained.

Deep or non-healing ulcers will require debridement (as is performed in dogs) once the infection has been controlled. EDTA drops may be used although in my experience they are rarely required.

Corneal stromal abscesses

Stromal abscesses secondary to trauma may be seen as a white plaque on the cornea. There are varying degrees of oedema and neovascularization. The neovascularization may obscure part of the abscess. This usually occurs when the

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epithelium grows over a traumatic defect in the cornea, trapping the bacterial contamination. Uveitis may also be present.

Treatment

Treatment is intense and may require surgical drainage of the abscess, which will also enable culture and sensitivity. The most frequently isolated bacteria are *Staphylococcus* spp. and *Pasteurella* spp. Frequent topical antibiotics and in some cases, systemic antibiotics are required. Topical and systemic anti-inflammatories are indicated.

Lens disorders

Nuclear sclerosis

Nuclear sclerosis is seen as a normal ageing change in chinchillas and as in more familiar species does not affect vision.

Cataracts

Cataracts are commonly seen in chinchillas and many, if not all, are associated with diabetes. *Blood glucose and serum fructosamine should be checked in every chinchilla presenting with cataracts* (Figure 7.14). They are usually bilateral but may present initially unilaterally and at different stages. The median age presented with cataracts is 8 years old (2–12 years).

Owing to many cases of diabetes being type 2, *if the owner has changed the diet, the chinchilla may present with cataracts and have normal blood glucose*. If the chinchilla is obese and/or has a diet too high in sugar, a full dietary review should be performed as many cases of diabetes can be managed nutritionally. HEV'N manufactures a diabetic herb mix which may help with reducing blood glucose (Diabetes).

Exophthalmos

The most common cause of exophthalmos in chinchillas is retrobulbar abscessation secondary to dental disease. Other less common causes include orbital neoplasia, sinus infection, foreign body, trauma, and brain tumours extending into orbital.

The globe is normal in size, is often visual, and progression of the exophthalmos may be slow. Retropulsion of the globe is not possible.

Diagnosis

Diagnosis is made from clinical signs. Radiographs are indicated to check for bone involvement and fully assess for dental disease. Careful oral examination under general anaesthetic should be performed.

Treatment

Treatment will vary slightly depending on aetiology. *Consideration should be given to euthanasia in cases with severe dental disease and/or osteomyelitis*. See Dental disease section for assessment and treatment options.

Enucleation should be performed transconjunctivally if there is no evidence of an abscess or neoplasia. Chinchillas have an orbital venous sinus and a large Harderian gland.

Abscessation or neoplasia will require exenteration to remove all the structures within the bony orbit.

Exenteration

It is important *to advise owners that once healed, the skin is likely to sink into the socket*. If owners have had previous experience of enucleation performed transconjunctivally, they will not be expecting the less cosmetic appearance of a sunken, empty eye socket.

Perform a lateral canthotomy and remove eyelid margins. Excise all the tissue within the orbit, working close to the bone to remove the whole sinus together with the globe. Ligate or use a clip to control haemorrhage. Collaclot (CambCol) can be used to control any haemorrhage if needed. The skin is then sutured closed.



Figure 7.14 Chinchillas have prominent veins on their pinnae. A handheld glucometer may be used to estimate blood glucose by using an ear prick.

Orthopaedics: fractures

Fracture repair principles used for dogs and cats are easily adapted to small mammals (it is just mammalian bone), and chinchillas have similar skeletal anatomy which allows texts aimed at repair of selected fractures in dogs and cats to be consulted for surgical approach.

Most fractures in chinchillas are due to trauma, either being dropped by the owner, trapping a leg in the cage or from a bite. *Owing to their legs being so close to their body (which makes lameness harder to detect), their amazing ability to hide pain, and today's busy lifestyle of many owners, it is not uncommon for a considerable delay to be present between the trauma occurring and presentation*. Occasionally, an old, healed fracture will be found as an incidental finding on radiographs with the owners being completely unaware of the previous trauma. The tibia is the most fractured bone, and fractures of the tibia are frequently either transverse, or a short spiral fracture.

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Pathological fractures can occur due to: osteopenia (lack of exercise, lack of UVb, diet too high in phosphorus/too low in calcium), osteomyelitis, and neoplasia.

Various options exist for fractures in chinchillas and many owners are keen to explore options. *Pain causes anorexia and gut stasis so prompt analgesia and supportive care is required.*

The main differences in chinchilla orthopaedics which we need to appreciate are:

- Chinchillas rest and move with their limbs very close to their body. This makes external coaptation more challenging to effectively apply – but not impossible.
- Dogs and cats find it easy to lift a painful limb while ambulating normally on the remaining three limbs – chinchillas find this very difficult. Chinchillas also rely on their front legs for eating and thorough grooming, and hind legs for balance when grooming perianal regions or consuming caecotrophs.
- Chinchillas spend a lot of time resting predominantly on their hind legs. The tibia is longer than the femur and the fibula is virtually non-existent.
- Chinchilla bones are thin and fragile which can make surgical repair challenging.

Supportive care (check for signs of shock and treat appropriately) should be given and owners should be advised that depending on the presentation, stabilisation may take several days, with the exception of spinal fractures, shock is more life threatening than a fracture is.

General anaesthesia is required to ensure diagnostic quality radiographs. Two orthogonal views should be taken, and I advise full body radiographs to assess for other skeletal injuries. Careful handling under general anaesthesia should be used if suspicious of a vertebral fracture as the epaxial muscles will not be supporting the vertebrae. If an open fracture is present, then a swab should be taken of the fracture to allow bacterial culture and sensitivity to be performed.

Regardless of fracture repair technique used, it is vitally important to adequately analgise (they will find this just as painful as you would), minimise stress, and restrict activity levels until healed.

Conservative management

Depending on fracture type, *strict cage rest in a single level enclosure and analgesia* may actually achieve good clinical resolution of fractures. Tibial fractures are unlikely to respond well to this kind of management technique due to the limited soft tissue covering. See Figure 7.15 a chinchilla with a femoral fracture and surgery was not an option for this owner. The femur healed well within eight weeks and the chinchilla has a fully functional leg.



Figure 7.15 This right femoral fracture was managed by cage rest and multimodal analgesia. The femur healed well within eight weeks and the chinchilla has a fully functional leg.

Surgical management

See intramedullary pinning, external skeletal fixators (ESF), and amputation in Chapter 3. Complications associated with surgical fixation include pin loosening and infection, non-union, necrosis of distal limb, and auto mutilation. *Chinchillas usually adapt well to hind limb amputation.*

Dermatology

Chinchillas have exquisitely fine fur, that is why they are valued for their pelt. Each follicle contains 50–60 woolly hairs around a central guard hair and the first moult in an adult chinchilla can occur at any time. Patchy to generalised hair loss is seen with many conditions including fur slip, fur chewing, nutritional deficiencies, dermatophytosis, trauma, and bacterial infections.

Fur slip

Fur slip is a method of predator avoidance but can be extremely distressing to owners, especially if they are not aware it can happen. If frightened during handling, or if fur is grasped roughly, a large patch can become easily epilated. Owners should be reassured the patch will grow back (takes 6–8 weeks) although it may be several months before it becomes indistinguishable.

Fur chewing

Fur chewing is an exaggerated form of overgrooming where the fur is chewed (rather than groomed) halfway down the

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length, most often at their hips and sides. In severe cases there may be a 'lion's mane' appearance due to the inability to chew around their own head. There is increased adrenocortical activity (higher plasma cortisol levels and adrenocortical hyperplasia) associated with fur chewing and it is considered a manifestation of physiological stress in chinchillas. In other words, *fur chewing is a maladaptive displacement behaviour triggered by stress in stress sensitive individuals*.

There is a female sex bias and good evidence exists to suggest chronic pain may be a trigger (e.g. 21–30% of dental disease cases in one study demonstrated fur chewing). Other known triggers for fur chewing include poor environmental conditions, stressors (e.g. loud noises), boredom, overcrowding, lack of access to regular dust baths, and low fibre diets. *Chronic, inescapable boredom is a welfare concern for any species*.

Once the behaviour has started it can be difficult to fully resolve and relapse is common. A full husbandry review should be carried out with environmental enrichment and dietary requirements in mind. Increasing the amount of solid flooring with substrate on it and increasing the frequency of changing the dust bath, has been shown to reduce fur chewing in farmed chinchillas.

Husbandry changes should be implemented specifically to reduce stress that may include reducing handling, reducing light intensity or duration, decrease noise disturbances, and give enrichment to redirect behaviour. It is important to monitor enrichment to ensure it does not add to the stress of the chinchilla. Fluoxetine has been suggested as a therapy, however the one study performed where it was administered at 10 mg/kg q24hrs it had limited success. This may be due to the fact concurrent husbandry changes were not performed.

Matted fur

Like most mammals, chinchillas produce natural sebaceous oils. Chinchillas use dust bathing to help absorb these oils. If they are deprived of frequent dust baths, matted fur can develop from a build-up of these natural oils. Being kept in an environment that is too warm and humid (>24°C (>75.2°F) and > 50% humidity) will also result in matted fur.

Therefore, a full husbandry review is required to correct the situation.

Pododermatitis

Pododermatitis is less common in chinchillas than in guinea pigs, and is associated with a lack of mobility, constant exposure to wire or abrasive flooring, obesity, and poor sanitation. The constant trauma to the foot causes: erythema, swelling, ulceration, and crusting of the soft foot pads. The hind feet are mainly affected. Hyperkeratosis and erythema are more common. Severe cases, with open lesions and osteomyelitis are much less common.

Open lesions should be swabbed for culture and sensitivity, but typically *Staphylococcus* spp. or *Streptococcus* spp. are isolated.

Treatment varies depending on severity from husbandry adjustments only in the mildest cases, application of petroleum jelly to areas of hyperkeratosis without open lesions, weight management in cases of obesity, through to surgical debridement for the more severe cases. Treatment protocols are similar to those for guinea pigs in surgical cases.

Oxyurid infection

Oxyurid infection of unknown species was diagnosed in a chinchilla presenting with intense perianal pruritus. The chinchilla was seen vigorously rubbing her bum along the bottom of the enclosure. A tape test of the anus revealed the classical oxyurid egg shape (thick walled, oval, flattened lateral symmetrically) and fenbendazole 20 mg/kg q24hrs for 5 days was curative.

Dermatophytosis

Dermatophytosis is rarely seen in pet chinchillas. If diagnosed, consideration should be given to the potential immunosuppression of the chinchilla and co-morbidities or stressors should be investigated. Owners should be advised that dermatophytosis is zoonotic.

The most common fungal isolate is *Trichophyton mentagrophytes* (which does not fluoresce under a Wood's Lamp) but *Microsporum canis* and *M. gypseum* have been described.

Clinical signs can include patchy, scaling alopecia, affecting primarily the nose, feet, ears, and tail (although the lesions can be anywhere). There are variable levels of erythema.

Diagnosis is via skin scrapings and *hair plucks from the edge of lesions* for fungal culture and/or microscopic examination.

Treatment is systemic therapy with either itraconazole 5–10 mg/kg q24hrs or terbinafine 20–40 mg/kg q24hrs, and continued until two negative fungal cultures are obtained 1 month apart. Not all owners will be able or willing to pay for repeated fungal cultures. Treatment should be for a minimum of three weeks and if owners cannot or will not repeat the cultures this should be recorded in the notes and owners should be made aware that without the tests, we cannot guarantee full resolution just because the obvious clinical signs have resolved. Terbinafine is preferred as it has been shown to be more effective in guinea pigs.

Disinfection of the environment with a suitable agent such as sodium hypochlorite or F10 is an important part of management. Wooden enclosures or shelves may be difficult to eradicate the dermatophytes from.

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Ectoparasites

Ectoparasites are uncommon due to chinchillas' extremely dense coat, but anecdotal reports exist for *Cheyletiella* infestations.

Fleas

In houses with dogs and cats, they may contract fleas. Heavy infestations of fleas may result in a dull coat, intense pruritus, and patchy alopecia. Diagnosis is achieved via careful clinical examinations for fleas and flea dirt. Treatment is topical selamectin 15–25 mg/kg, environmental control, and treatment of the dog or cat.

Nutritional disorders

Owing to the ready availability of good quality chinchilla concentrates, nutritional disorders should not be a problem. It is worthwhile being aware of the potential of these conditions, especially if owners feed mainly a homemade diet.

Excess protein

A diet higher in protein than 15–18% results in the growth of gently coiled or wavy hair known as cotton fur.

Deficient fatty acids and zinc deficiency

Deficiency in fatty acids can cause reduced hair growth, dry scaly skin, and hair loss. Zinc deficiency leads to alopecia and parakeratotic hyperkeratosis.

Neoplastic skin disease

Neoplastic disorders are considered rare in chinchillas. Otrocka-Domagała *et al*. (2022) found during their retrospective study (2014–2021) that although neoplasms were rare in chinchillas, *malignant tumours represented the predominant group of neoplasms of cutaneous tumours* in chinchillas (as well as in rats, African pygmy hedgehogs, and degus).

There are only three cases of squamous cell carcinomas (SCC) (all were on limbs) and one case of mammary adenocarcinoma in the literature.

Squamous cell carcinomas

In all three reported cases of SCC the chinchillas were over 15 years old. The SCC were located on a limb, 2–5 cm in diameter, and had results in self-mutilation. All three cases were euthanised within 2 months of the initial diagnosis.

Ear diseases

Chinchillas are less prone to ear diseases than other rodents. They have a very large tympanic bullae, a large tympanic membrane, and a short, simple ear canal.

An otoscopic examination is recommended in all chinchillas. The canal is wide and there should be no discharge. The tympanic membrane should be visible and appears semi-transparent and avascular. Any changes to this including discharge, vascularity of the tympanic membrane, or bulging of the tympanic membrane are indicative of inflammation of the middle ear, which will require further investigation.

Clinical signs vary and include external ear canal discharge, head shaking, mild head tilt, facial paralysis, through to severe neurological deficits.

Primary otitis externa is rare. Most cases of otitis externa are secondary to otitis media with perforation of the tympanic membrane, although neoplasms and polyps of the external ear canal causing secondary otitis media/interna have been reported. A 5 month old female chinchilla presenting with respiratory distress, head tilt and suppurative otitis externa, was found to have inflammatory polyps in the auditory canal as the primary lesion.

If a purulent ear discharge is present, diagnostic imaging is recommended to check for middle ear disease. Increased radiopacity of the tympanic bullae can be detected on dorsoventral radiographs, although CT scans are much more sensitive and will give more information.

Pseudomonas is the most common isolate, and due to the high risk of multidrug resistance to antibiosis, culture and sensitivity is vital. Azithromycin 30 mg/kg q24hrs has been shown to reach high enough tissue levels in the middle ear to be effective against susceptible bacteria, and can be used empirically while waiting on results.

Sterile sampling of the middle ear

The thin-walled nature of the bone of the tympanic bullae lends itself to minimally invasive sterile sampling for culture and sensitivity in cases of otitis media.

Performed under a general anaesthetic, surgically prepare a small area over the dorsal aspect of the affected tympanic bullae. Make a small incision in the skin over the dorsal aspect of the tympanic bullae and use a 18–20g needle to perforate the bone. Once access has been achieved, a sterile 20–22g IV catheter without the stylet can be inserted to aspirate samples for cytology, culture, and sensitivity. The skin wound can be sutured or tissue glue can be applied (Figures 7.16 a–d).

Prognosis for full resolution of otitis media can be poor due to the difficulty of fully eradicating infection, especially if *Pseudomonas* is involved.

Pseudomonas aeruginosa infections

Chinchillas are prone to *Pseudomonas aeruginosa* infections, which can be localised to a single organ, but can also progress to multiorgan disease and sepsis.

Conditions associated with *P. aeruginosa* include conjunctivitis, enteritis, otitis, metritis, balanoposthitis, rhinitis, and pneumonia. If the infection spreads systemically, septicaemia, neurological signs, abortion, and death, can occur acutely. *P. aeruginosa* can be isolated from the

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Figure 7.16a Surgically prepare a small area over the dorsal aspect of the affected tympanic bullae. The tympanic bullae is easily palpated.



Figure 7.16b A small incision is made in the skin over the dorsal aspect of the tympanic bullae.

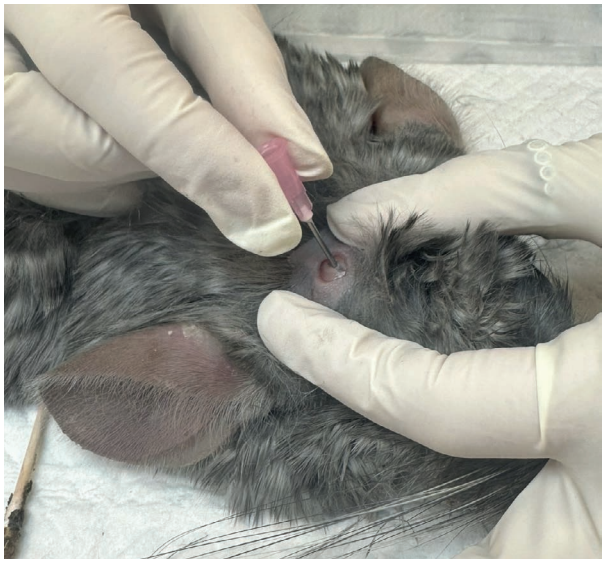


Figure 7.16c A 18–20g needle is used to perforate the bone.



Figure 7.16d Then a sterile 20–22g IV catheter without the stylet, can be inserted to aspirate samples for cytology, culture, and sensitivity.

mouth and faeces of apparently healthy chinchillas, but not from the conjunctiva.

Clinical signs associated with systemic pseudomoniasis are non-specific and include anorexia, decreased faecal output, lethargy, and depression. Clinical signs of *Pseudomonas* infections in specific organs are attributable to that body system.

Treatment

Empirical treatment with antibiotics pending culture results is always recommended in cases of suspected *Pseudomonas* infections due to the risk of systemic spread and associated morbidity/mortality.

Culture and sensitivity are strongly recommended as *P. aeruginosa* frequently displays multi-drug resistance,

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Table 7.8 Chinchilla urinalysis normal.

Colour	Variable depending on diet
Protein	+ (may be false positive if dip stick used)
pH	8.5 or higher
Specific gravity	>1.045 (may be >1.060)
Sediment examination	Scant calcium carbonate, triple-phosphate crystals, and amorphous crystals

especially to the few antibiotics that are considered safe to use in chinchillas.

Prevention

Regular cleaning and then disinfection of water bowls, taps, and bottles is critical as these can be a source of *Pseudomonas* colonisation.

Urinalysis

Normal urine can vary dramatically in colour as it can be affected by diet. The urine specific gravity (USG) is typically over 1.045 and may be over 1.060. The USG can be measured without spinning the urine. Dipsticks can be used to check for blood, ketones, and the pH, however *the high USG in chinchillas results in false positive protein on dipstick test, therefore dipsticks should not be relied upon for urinary protein measurement in this species*. The normal pH is often 8.5 on the dipstick but may be higher as that is the limit of the test! *Alkaline urine can also be associated with false positives for protein using dipsticks*.

Ketoacidosis may occur readily, secondary to anorexia, in chinchillas and urine dip sticks are a non-invasive, reliable, and cost-effective way of checking metabolic status.

Normal chinchilla urine will contain amorphous crystals, but *calcium carbonate crystals should be scant if present as they predominantly excrete excess calcium via the faeces*.

Urolithiasis in chinchillas

Chinchillas *absorb all calcium from ingested food and the vast majority of excess levels are excreted via the faeces*, which is completely different from rabbits and guinea pigs which excrete excess calcium via the urine.

The most common urinary tract disease in chinchillas is urolithiasis and it is seen *predominantly in male chinchillas, the median age is 30 months* (range 11–132 months). Most calculi are calcium carbonate and semen matrix calculi have been reported. The risk factors for development of calculi in chinchillas are currently unknown. It has been suggested that the wider, shorter urethra in female chinchillas, allows them to pass urinary calculi.

Clinical signs

Owners may report vocalisation during toileting. The pain caused by the posture required to urinate and defecate can result in vocalisation during these behaviours and so we need to also check for joint/skeletal pain during the clinical exam.

The most common clinical signs are haematuria, increased frequency of small volume urination, stranguria, pain on abdominal palpation, and the calculi may be palpable. Less specific signs include hunched posture, tooth grinding, reduced appetite/anorexia, weight loss, lethargy, and continuous straining in cases of blocked urethra.

In cases of ureteral or renal uroliths the only clinical signs present may be depression, anorexia, and weight loss, with no obvious changes in urination patterns.

The chinchilla may have a full, painful bladder, and may spontaneously urinate on the consulting table during bladder palpation. Alternatively, place the animal in a clean plastic tub as many will spontaneously urinate. Collection of the urine will allow a dipstick examination (to check for blood, glucose, ketones, and pH just as with more familiar species), and potentially a microscopic examination of the sediment. Chinchillas can also have porphyrin in their urine which may visually mimic blood.

Blood in the urine and painful bladder palpation is highly suspicious of bladder calculi, but uroliths (there may be more than one!) can be present anywhere along the urinary tract including: kidneys (uncommon), ureters, bladder, or the urethra. The prognosis is better for calculi in the bladder.

Diagnosis

Chinchilla uroliths are radio dense and radiography under sedation/general anaesthesia is diagnostic. Care should be taken during positioning to ensure the whole length of the urethra is included, especially in males (Figures 7.17a & b).

Dietary dissolution of uroliths is not possible in chinchillas and surgical intervention is required. Recurrence after surgery is common and is quoted to be as high as 50% recurrence rate. Unfortunately, as the risk factors are unknown, it is difficult to give advice to prevent recurrence, but there are some sensible considerations. Ensuring appropriate analgesia is given in cases with degenerative joint disease would not only increase welfare, but potentially prevent super concentration of the urine if they are too painful to posture correctly when emptying their bladder. Promoting natural diuresis by providing cooled dandelion or nettle teas as drinking water will promote further fluid intake, increasing flushing out of the bladder and hopefully reducing the risk of recurrence.

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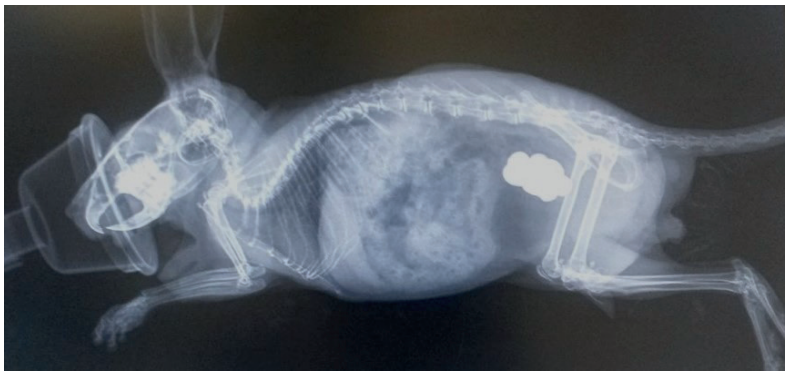


Figure 7.17a A lateral radiograph of a bladder stone in a chinchilla. This chinchilla presented with signs of cystitis.



Figure 7.17b A lateral radiograph of a different chinchilla with multiple uroliths. One looks to be in a ureter and two are in the neck of the bladder. The radiograph is poorly collimated and potentially uroliths in the urethra would be missed as the whole length of the urethra has not been included.

Sterile collection of urine

The rough texture of the uroliths will cause trauma to the mucosa of the bladder (or other structures) and secondary infection is common.

Ideally perform a culture and sensitivity prior to antibiosis, and this can be achieved by passing a sterile urinary catheter while under sedation. For most chinchillas this can be achieved using a 3–5 Fr urinary catheter or nasogastric tubes, and fingertip pressure should be used to pass the catheter to avoid iatrogenic damage to the urethra. As the male chinchilla penis is S shaped, *extending the penis caudally facilitates passing the catheter through the pelvic flexure of the urethra*. Female chinchillas rarely require urinary catheterisation, but are easy to perform due to the distinct separate urethral papillae.

Trimethoprim sulfamethoxazole is a sensible first line choice of antibiotic while waiting on culture results along with analgesia. I recommend starting medications for at least five days prior to surgery to minimise the secondary infection.

Urethral stones

Urethral catheterisation as above and retropulsion of the stone into the bladder can be attempted. The *stone may be embedded* into the urethral mucosa which may prevent movement. Diluted lidocaine may be instilled via the catheter to alleviate urethro spasm, which may facilitate retropulsion. In one study, 75% of the chinchillas presenting with urethral stones were euthanised within 24 hours of diagnosis (Martel-Arquette & Mans, 2016).

Cystotomy

Cystotomy in a chinchilla is very similar to in more familiar species, just on a much smaller scale. A urinary catheter can be placed as above.

My preference is to incise the skin directly over the bladder and extend the wound as needed.

Once the abdomen is open, taking care to fully tent the muscles and prevent iatrogenic damage to the caecum, I will exteriorise the bladder and pack around the bladder with moistened sterile swabs.

My preference is to ventro flex bladder and then stay sutures can be placed as per more familiar species. The bladder can then be incised near the cranial pole, in an avascular area. *Ensuring the bladder incision is nearer to the cranial pole of the bladder should prevent iatrogenic damage to the ureters* (ureters enter the bladder in the trigone area of the neck, on the dorsal aspect). My rationale behind the dorsal incision is the hope that gravity will help healing, as the urine will initially make more contact with the ventral aspect of the bladder.

The texture of the stones can be extremely rough, this can cause the stone to adhere tightly to the mucosa of the bladder. Use sterile forceps or a blunt curette to remove the stones (Figure 7.18), flushing the urethra and bladder neck with sterile saline will reduce the risk of leaving

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Figure 7.18 Removal of a urolith.

calculi behind. I also advise owners that I have experienced cases where calcified plaques have been present in the bladder mucosa rather than only discrete stones, therefore if you have flushed the bladder and yet cannot find all the calculi seen on the radiograph, examine the mucosa. If required, a small sliver of bladder mucosa can be removed for culture.

The bladder may be closed in one or two layers depending on the wall thickness. Most cases in my experience require two layers: 1.5 metric (4/0) Poliglecaprone 25 (Monocryl, Ethicon) with a swaged-on needle can be used to perform either two continuous inverting suture layers, or one inverting followed by a simple continuous, depending on individual surgeon's choice and experience. It is recommended to repair without penetrating the mucosa as it is thought that the suture may act as a nidus for further calculi development in susceptible individuals. Remove the swabs and stay sutures, flush the abdomen out with warmed saline and repair the muscle layer with 1.5 metric (4/0) Vicryl with a swaged-on needle use a cruciate pattern, repair the subcutaneous layer using a simple continuous and the skin layer may be repaired either with intradermal sutures or surgical staples. The stone can be sent for analysis. Analgesia should be continued for at least 10 days, or if osteoarthritis is present, then the owner should be counselled the pet may be on lifelong analgesia.

Diabetes mellitus

Hystricomorph rodents have a different insulin structure with lower physiologic activity (only 1–10% active) compared to other mammals. They compensate to control blood glucose levels by having a higher insulin concentration, a slower insulin degradation, and a higher number of insulin receptors compared to other mammals. *In other words, chinchillas cannot regulate blood glucose concentrations as tightly as other mammals*.

Both type 1 and type 2 diabetes mellitus occur in chinchillas, although type 2 is more common, especially in obese chinchillas.

Clinical signs are very similar to more familiar species: cataracts, polydipsia, polyuria, highly elevated blood glucose, glycosuria, ketonuria, low urinary pH, alongside more nonspecific clinical signs such as anorexia and lethargy.

> Hepatic lipidosis and ketonuria occur readily in chinchillas. If they have a reduced appetite for any reason, stress-induced hyperglycaemia being very common, and if the hyperglycaemia crosses the renal threshold, glycosuria will occur in stressed chinchillas. Therefore, diabetes mellitus should never be diagnosed on a single day's test results.

Diagnosis

A full clinical examination for comorbidities should be performed.

Full biochemistry and haematology are recommended (see Venipuncture) as well as serum fructosamine measurement. A fructosamine level has not been established in chinchillas, but has been in established in a close relative of the chinchilla, the free ranging plains viscacha (Lagostomus maximus): reference range 215–296 μmol/l (Wenker *et al.*, 2007).

Blood glucose levels should be checked on several occasions, which can be performed via an ear prick and a glucometer (Figure 7.14).

Urinalysis should also be performed (see Table 7.8). If blood is present, then sediment examination for evidence of a secondary bacterial urinary tract infection is advisable.

Treatment

Perform a full dietary review. Obese chinchillas are more likely to have type 2 diabetes, which will most likely

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respond to dietary management and weight loss. Stop all sugary treats, e.g. raisins or dried fruit.

The urine can be monitored at home with twice daily dip sticks to monitor glycosuria and ketone production. Should the glycosuria or ketonuria fail to resolve, glipizide starting at 0.6 mg/kg q12hrs and titrate upwards can be trialled, although there is scant data on the recommended dose.

Alternatively, insulin can be started, and owners should be advised on clinical signs associated with hypoglycaemia. Starting dose is recommended as 1 IU/kg q12hrs with incremental increases of 0.1 IU/kg. Owners may find volumes easier if it is worked out in millilitres rather than international units for ease of pulling up and injecting. Owners should monitor at home using urinary laboratory sticks. Glucose curves can be performed in the clinic; however, the stress of hospitalisation may make them difficult to interpret due to stress-induced hyperglycaemia.

I have successfully managed a couple of chinchillas on insulin injections, although most cases resolved with dietary adjustment.

Seizures

Seizures are uncommon but can be caused by encephalitis, septicaemia, toxicosis, dietary deficiencies, hypocalcaemia, hypoglycaemia, hepatic insufficiency, renal insufficiency, heat stroke (see section on heat stroke), and idiopathic epilepsy.

Diazepam can be administered per rectum to stop the seizure and blood should be taken for a full biochemistry and haematology. Treatment varies depending on aetiology.

Lead toxicosis

Although uncommon, it can occur especially if the owner has an old house and the chinchilla has been chewing painted woodwork, leaded window panes, or lead weights. Clinical signs associated with lead poisoning include seizures and blindness.

Diagnosis is achieved by checking blood lead levels. Radiographs should also be performed to check for metal fragment presence in the GI tract. Blood lead levels over 25 μg/dl (0.25 ppm) are suggestive of lead poisoning.

Treatment includes diazepam to control the seizure and chelating therapy with calcium disodium edetate 30 mg/kg SC q6–12hrs. Consider surgery or endoscopy to remove large fragments of metal from the stomach.

Zinc toxicosis

Zinc toxicosis is also uncommon, and although does not generally result in seizures, can cause ataxia which some owners may mistake for seizures. Generally, it will be caused by gnawing on items such as wine bottle tops or poor-quality galvanised mesh.

Clinical signs include depression, lethargy, GI stasis, and ataxia.

Diagnosis is achieved by checking blood lead levels. Radiographs should also be performed to check for metal fragment presence in the GI tract.

Treatment includes diazepam to control the seizure and chelating therapy with calcium disodium edetate 30 mg/kg SC q6–12hrs. Radiographs should also be performed to check for metal fragment presence in the GI tract.

Toxoplasmosis

Toxoplasmosis gondii is reported to cause sudden death, depression, hunched posture with drooping of ears and head, tachypnoea, ataxia, and seizures.

Reproductive disorders in chinchillas

Female chinchilla

Female chinchillas are seasonally polyoestrous, which in the northern hemisphere, occurs between November and May. They are sexually mature from 6–9 months of age and gestation is 111 days. Pregnancy is palpable from day 90. During oestrus the vaginal membrane is absent, the perineum changes colour from a dull flesh tone to a deep red colour, there is no vulval swelling, but a mucoid vaginal discharge develops. The vagina is situated between the urethral papillae and the anus and is normally obscured by a membrane. Absence of the vaginal membrane occurs during oestrus, parturition, or uterine disease.

Parturition usually occurs early in the morning, typically before 8 am, and is usually complete within 4 h. The litter size is between one and four precocial kits and the female will consume the placentas. Owners may become concerned if they see blood on the nose or paws of the chinchilla but should be reassured that this is normal to see immediately post-consumption of the placenta.

Dystocia

Dystocia is very uncommon in pet chinchillas and occurs for similar reasons as per more familiar species, including uterine inertia, malpresentation, single oversized foetus, dam obesity, disturbance during parturition, or previous trauma causing narrowed pelvis.

Clinical signs include restlessness, frequent vocalisation, constant attention to the genital region and a widened vaginal opening. Unproductive straining may be obvious. *If the labour has lasted over 4 h or there is sustained non-productive straining, consider intervention.*

Radiographs are indicated to check number, size, presentation, and for malformation of the pelvis.

Treatment of uncomplicated dystocia, i.e. uterine inertia, is the same as more familiar species: oxytocin 0.5–1 IU/kg SC and calcium gluconate 25–50 mg/kg, diluted with saline and given SC.

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Caesarean section is indicated if medical management fails or in cases of oversized or malpresented kits. Isoflurane or sevoflurane induction is performed without pre-medication to prevent the kits being adversely affected. Caesarean section is performed in a similar manner as in more familiar species and a survival rate of 67% was reported in one study.

Endometritis and pyometra

Endometritis and pyometra are uncommon but are increasingly being reported in pet chinchillas. Stump pyometra secondary to incomplete ovariectomy has been reported.

Clinical signs include acute onset depression and anorexia, or mild lethargy, or behavioural changes, open vulva and a vaginal discharge is seen in most cases (but closed pyometras can occur). Vaginal discharge varies in appearance from mucoid, to mucopurulent, to haemorrhagic, and can cause fur staining. It is important to *remember that a small amount of mucoid vaginal discharge is normal during oestrus*; clinical signs and cytology can be used to differentiate between oestrus discharge and pyometra.

Uterine enlargement may be palpable during clinical exam and palpation of the uterus may elicit more vaginal discharge.

Diagnosis is achieved via clinical signs and ultrasound examination of the uterus. Ideally perform urine dipstick analysis to check for ketosis. Hepatic lipidosis, ketosis, and sepsis may be present and complicate the presentation.

Treatment of choice includes stabilisation, systemic antibiotics, analgesia, and ovariohysterectomy (OVH). If the chinchilla is not stable enough for surgery or the owner does not want surgical intervention then medical management (as per guinea pigs) can be attempted with three SC injections of aglepristone (Alizin 30 mg/ml, Virbac Limited) 10 mg/kg given on days 1, 2, and 8, and stabilisation, systemic antibiotics, and analgesia.

Cystic ovaries

Cystic ovarian disease is rare in chinchillas.

Haemorrhagic vaginal discharge

If a female chinchilla which is housed with an entire male, presents with a haemorrhagic vaginal discharge, consider pyometra (see above), abortion, foetal retention, or placental retention.

Uterine neoplasia is increasingly reported with leiomyosarcoma, leiomyoma, fibroma, and haemangioma all being cited. Uterine masses are often palpable and there may be a haemorrhagic vaginal discharge. OVH is the treatment of choice.

Mammary glands

There are three pairs of mammary glands: two pairs are lateral thoracic and one pair are inguinal. Mammary neoplasia is very rare but metastasis have been reported (pulmonary, hepatic, and renal) and thoracic radiographs are advised prior to surgical resection of any mass.

Ovariohysterectomy

Chinchillas grossly have one cervix but internally there are actually two. From a surgical point of view this does not actually matter. OVH is similar to guinea pigs as chinchillas also have short ovarian ligaments. Care should be taken upon entry to the abdomen because of the risk of damage to the large gastrointestinal tract.

Male chinchilla

Male chinchillas are sexually mature from 8–9 months of age. *Penile disorders are very common therefore the prepuce and the glans penis should be carefully examined in every male chinchilla*. Fur rings, phimosis, balanoposthitis, preputial abscesses, and paraphimosis have all been described.

Fur rings

Fur rings consist of a ring of constricting fur, wrapped around the penis, under the prepuce. This can lead to paraphimosis, stranguria, excessive grooming, or self-trauma. Carefully extrude penis to diagnose.

Treatment requires removal of the fur ring either by lubrication and cleaning, or by cutting the ring. In many cases this can be performed conscious but in fractious animals a sedation or general anaesthetic is more appropriate.

Paraphimosis

Fur rings may cause (see above). Over excitement or exhaustion may also result in paraphimosis which self corrects with time.

Phimosis and balanoposthitis

Extensive adhesions between glans penis and penis lead to phimosis. Secondary accumulation of debris or smegma results in bacterial balanoposthitis. *Septicaemia can develop if untreated*.

Ulcerative and suppurative balanoposthitis is one of the manifestations of *Pseudomonas* and with chronicity, peri genital abscessation may occur.

Clinical signs vary with severity but may include none, penile discharge, dysuria, straining to urinate, retention of urine in the prepuce (secondary to adhesions), anuria, excessive grooming, breeding failure, post-renal azotaemia, urethral constriction, and urinary scald.

Treatment is multifactorial. Analgesia such as meloxicam should be given and the effect assessed. Adhesions must be broken down between the glans penis and the prepuce, which may require surgical intervention if they are extensive. Ideally a swab should be taken for culture and sensitivity, while waiting on results trimethoprim sulfadiazine 30 mg/kg q12hrs can be started empirically.

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The owner must be able to extrude penis daily to repeatedly break down further adhesions, clean the area gently with 0.125% chlorhexidine, and apply a topical steroid cream.

Preputial abscesses

Preputial abscesses should be surgically explored and drained.

Castration

The fascia surrounding the vaginal tunic is very closely attached to other tissues *which makes closed castration difficult to perform* without blunt dissecting through the vaginal tunic. It is *easier and less traumatic* to perform an open castration as per other species and then ligate the tunic as close to the inguinal ring as possible to avoid the risk of herniation.

Abdominal castration

I am not a fan of abdominal castration as I believe that entry into the abdomen is *associated with more pain* than scrotal or pre-scrotal castration. Saunders (2013) also pointed out that abdominal castrations potentially are associated with higher risk of adhesion formation, peritonitis, and that severe complications may arise from any wound interference compared to traditional castrations.

Gastrointestinal disease

Oesophageal 'choke'

Occasionally, food items (raisins, fruit, nuts) or bedding material can become stuck in the oropharynx or upper oesophagus. This may be more common in chinchillas with dental disease as they may be more prone to swallowing treats whole due to the pain associated with fully chewing.

Clinical signs are sudden, and onset can be dramatic: drooling (may be green tinged from food), retching, and possibly dyspnoea.

Treatment is to remove the foreign material; this may be possible by stroking up the oesophagus to hopefully push the material back into the mouth. Midazolam may be required if the chinchilla is panicked.

If unsuccessful, an anaesthetic is required, and the chinchilla should be maintained with the head lower than the body to try and prevent aspiration. Swab out the mouth immediately upon induction to remove the saliva which is often mixed with the food and visualise the back of the oropharynx using an otoscope with the long speculum. Crocodile forceps can be used to grasp material for removal if required, or a crop tube/Sproul's needle on a 5 ml syringe can be used to provide suction. Endoscopy can be used if available.

Analgesia should be given.

What are normal faecal pellets like?

Normal faeces in chinchillas are smaller than expected for an animal of this size. Each faecal pellet is the size of a plump grain of rice (8.6 mm × 3.9 mm), is dark brown – black in colour, dry, and is slightly shiny when first passed (Figure 7.19).

Diarrhoea/soft faeces

Conditions leading to soft faeces or diarrhoea are less common in chinchillas compared to guinea pigs, however, any systemic disease, stressful, or painful condition can result in secondary gastrointestinal problems. These can have nonspecific clinical signs such as: decreased food intake or anorexia, decreased faecal output, and lethargy.

In pet chinchillas, sudden dietary changes, excessive amounts of fresh foods, or inappropriate oral antibiotic therapy are the most frequent cause of clinical signs.

Initially the only issue the owners may notice is faecal smearing on the cage floor, or perianal fur matted with faeces and the chinchilla may otherwise look and act completely normal. In chronic or severe cases, the chinchilla may be anorexic, dehydrated, and depressed.



Figure 7.19 Normal faeces in a chinchilla (bigger faeces) beside faeces from the same animal prior to treatment. The chinchilla had reduced appetite due to dental pain.

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A full history will establish if there have been any changes to the diet or oral antibiotics.

Diarrhoea due to diet

Stop the new food or fresh foods. Give a hay-based diet and fluids if needed.

Antibiotic induced dysbiosis and enterotoxaemia

Narrow spectrum antibiotics (especially ones with predominantly activity against Gram positive bacteria) such as amoxicillin, ampicillin, cephalosporins, clindamycin, erythromycin, lincomycin, and penicillins should never be given orally to chinchillas. Oral administration causes an overgrowth of Gram negative bacteria, especially *Clostridium*, which results in an often fatal enterotoxaemia and haemorrhagic typhlitis. If in doubt about the oral safety of an antibiotic always check the formulary prior to administration. That being said, even supposedly 'safe' antibiotics can cause issues with the gut microbiome in some individuals.

Treatment

Stop the oral antibiotics immediately. Assess hydration status and give IV fluids if necessary. Providing high-quality hay-based diet, and assisted feeding should be given in cases of anorexia. Collect faeces from the cage mates and add them to a syringe feed to help repopulate the gut. Prognosis is likely to be poor.

Intestinal intussusception

Intestinal intussusception usually involves the *descending colon* but can involve any part of the intestinal tract, and is secondary to anything that alters the motility of the bowel, e.g. dysbacteriosis, enteritis, constipation, or diarrhoea.

Clinical signs are sudden onset anorexia, refusal to dust bathe, distended abdomen, hypothermia, and abnormal posture (due to acutely painful abdomen).

Tenesmus is not normally seen unless the intussusception prolapses rectally. Abdominal palpation may reveal a turgid, cylindrical mass (as in more familiar species).

Treatment

Surgical intervention is critical, but the underlying condition must also be diagnosed and treated. The length of intestine involved may be extensive, and the affected portion is often congested and cyanotic. Resection and anastomosis are required in many cases, which is exactly the same procedure as in more familiar species – just on a smaller scale.

Owners should be aware that recurrence and rapid deterioration is a known risk, as intussusception is an acute complication of an often chronic underlying primary problem.

Rectal prolapse

Rectal prolapse and intestinal intussusception frequently occur together (see above). If the rectal prolapse is uncomplicated, manage it as in more familiar species under GA or deep sedation: reduce tissue swelling with hypertonic saline or sugar solution, lubricate, and replace. Place a purse string suture, taking care not to occlude the urethral papillae.

Investigation for the underlying cause of the prolapse is necessary.

Infectious causes of diarrhoea

Giardia

Giardia in chinchilla is species specific and disease as a result is uncommon. It is considered an opportunistic pathogen rather than a primary disease.

Chinchillas can also contract *G. duodenalis*, which is zoonotic. Juvenile animals or show animals are more likely to be positive for this species of *Giardia*. There is a confirmed case of transmission to a human after a young child consumed faeces from an infected chinchilla.

Diagnosis is via faecal smear or analysis.

Treatment is oral metronidazole 15–20 mg/kg q12–24hrs. *Metronidazole can cause a self-limiting reduction in food intake in some chinchillas, therefore owners should be advised to monitor food intake and faecal output carefully while on treatment*. Normal appetite should resume within 24 h of stopping the metronidazole.

Tinidazole is used as the treatment of choice for *Giardia* in people. Although tinidazole has shown that it does not affect the appetite in chinchillas (when dosed 20 mg/kg q12–24hrs), there is no data on the efficacy against *Giardia* in chinchillas yet. It is worth considering its use in cases refractory to treatment with metronidazole or if a particular individual is sensitive to metronidazole appetite suppressive effects.

Helminth infection

Captive chinchillas reportedly have a low incidence, but no studies have been performed in wild chinchillas. Previous, isolated reports of infection with *Haemonchus contortus*, *Trichostrongylus colubriformis*, and *Ostertagia ostertagi* have all been attributed to consumption of contaminated forage.

Oxyurid infection

Oxyurid infection of unknown species was diagnosed in a chinchilla presenting with intense perianal pruritus. The chinchilla was seen vigorously rubbing her bum along the bottom of the enclosure. A tape test of the anus revealed the classical oxyurid egg shape (thick walled, oval, flattened lateral symmetrically) and fenbendazole 20 mg/kg q24hrs for 5 days was curative.

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Cestode infection

Rodentolepis nana has been documented, and as transmission is via the faecal–oral route, in a species that performs coprophagy, superinfection with high numbers is possible.

Clinical signs include none (mild infections), anorexia, diarrhoea, weight loss, dehydration, and death.

Diagnosis is via faecal flotation and treatment is oral or SC praziquantel 5–10 mg/kg.

Taenia spp. and *Echinococcus* spp. have been documented but are considered rare.

Colonic foreign body

Whereas rabbits will typically present with a duodenal foreign body, chinchillas tend to develop colonic foreign bodies. In both species the foreign body is commonly an impacted oval of matted hair. Gut transit time in chinchillas is 12–15 h.

Clinical signs are mild depression, anorexia, *and a complete lack of faecal pellets*.

> Any chinchilla presenting with a complete lack of faecal pellets should raise the suspicion of luminal obstruction.

Clinical exam may elicit abdominal pain and distended loops of bowel may be felt.

Diagnosis

Radiographs demonstrate dilatation of the bowel by gas or fluid proximal to the foreign body. *Abdominal ultrasound is a very useful modality to pick up colonic foreign bodies*. The foreign body shows as an oval shaped hyperechoic object with strong acoustic shadowing and anechoic fluid dilates the bowel proximal to the foreign body.

Early biochemical changes include hyperglycaemia (pain), increased ALKP, hypokalaemia, increased BUN (pre-renal), hyperproteinaemia, which later becomes hypoalbuminaemia. *Severe hypoalbuminaemia carries a higher risk of mortality*. One study showed a perioperative survival rate of 71% and the acute postoperative deaths were attributable to severe hypoalbuminaemia, which led to hypotension, ascites, peripheral oedema, and gastrointestinal ileus.

Treatment

Some cases may respond to medical therapy. Good analgesia and supportive care with IV fluids and assisted feeding, but prokinetics should not be administered at this stage. Failure to respond to medical therapy requires surgical intervention, and in a retrospective study (Koizumi, 2022), all seven chinchillas diagnosed with a colonic foreign body required surgical intervention.

Exploratory laparotomy reveals an area of severely dilated bowel proximal to the foreign body with lumen collapse distally. Exteriorise the affected bowel and pack abdomen with moistened swabs. If possible, gently milk the foreign body distally or proximally to an area of healthy bowel before incising. Close the bowel with 0.7m (6/0) swaged-on Polydioxanone (PDS, Ethicon) interrupted sutures and check for leaks. Rinse abdomen out with warm sterile saline and close the muscle with 1.5m (4/0) swaged-on polyglactin 910 (Vicryl, Ethicon) in a cruciate pattern. The skin is closed using 1.5m (4/0) swaged-on polyglactin 910 (Vicryl, Ethicon) using an intradermal pattern. Apply EMLA 5% Cream (2.5% lidocaine, 2.5% prilocaine, Aspen) to the wound.

Continue on analgesics for 10 days, prokinetics and supportive care until eating and passing faeces. The pathogenesis is unknown at this time.

Tympany

Tympany is less common in chinchillas than in rabbits or guinea pigs and is seen secondary to rapid change in diet (fast fermentation of food material), over consumption of fresh clover, gastroenteritis, dysbacteriosis, ileus, luminal obstruction (see foreign body), or, very rarely, due to intestinal torsion.

Clinical signs include sudden onset anorexia, no faeces, a distended, tense abdomen, abdominal pain, hypovolaemic shock, tachycardia, tachypnoea, pale tacky mucous membranes, slow capillary refill time, hypothermia, and collapse.

Prognosis and treatment depend on the underlying diagnosis. Severe cases or cases that have been delayed prior to presentation have a much poorer prognosis.

Tympany due to gastric outflow obstruction, requires surgical intervention. Obstruction may be due to matted fur, carpet, wood chip, wallpaper, soft wood, rubber toys, mural abscess, or neoplasia. Owing to the continuous production of saliva, large volumes of stomach fluid, and the fact the stomach always contains ingesta, outflow obstruction will rapidly lead to gastric dilatation.

Surgical intervention

Once anaesthetised, the stomach should be decompressed using a nasogastric or orogastric tube. *Percutaneous drainage of the stomach via a needle is not advised* due to the risk of tearing or rupture of the stomach. If the chinchilla is breathing well, the stomach may be decompressed via a needle once the laparotomy has been performed which allows any tearing to be visualised and repaired. If the obstruction is a foreign body in the duodenum, attempt to milk the blockage back into the stomach for removal.

Gastrotomy is performed as per more familiar species, utilising stay sutures, incising in an avascular region between the curvatures, and is closed in two layers, with an inverting suture, using 1.5 – 2m (4/0 – 3/0) synthetic, absorbable, monofilament, with a swaged-on needle.

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The rest of the abdomen should be examined, including the rest of the intestinal tract (in case there are more foreign bodies). Inspissated free floating purulent material is not an uncommon finding, due to previous areas of localised abdominal inflammation.

Analgesics, IV fluids, supportive care including nutrition, and prokinetics should be continued post-operatively.

Constipation

Although not commonly seen, the faecal pellets are misshapen, darker, drier, and may be coated with blood (due to straining).

The main two causes of constipation are chronic low levels of dehydration and insufficient dietary fibre. Lack of exercise and obesity also are complicating factors. Lack of dietary fibre may be due to an increased consumption of pellets, either because of what the owner offers, or due to underlying dental disease causing altered consumption of food types.

Treatment is symptomatic with oral or systemic fluids (depending on severity). Laxatives (lactulose or a small number of raisins – unless diabetic) can be given. Analgesics are advised and prokinetics may be of benefit providing there is no obstruction.

A full dietary and water provision review and correction, if required, should be carried out. As there may be an underlying health concern, e.g. dental disease, a full clinical exam to diagnose and treat the primary condition is necessary.

Euthanasia

End of life conversations can be a difficult area to broach with some clients. Occasionally owners will wait for us to broach the topic as they are 'worried' if they do so we will think they are not dedicated enough. Reassuring clients that sometimes we need to love our pets enough to let them go when the time is right can be invaluable.

Many owners will want to stay with their chinchilla during this procedure and the process should be fully explained including possible events such as sudden jumping, the need to change veins or gasping. I administer a pre-med (e.g. medetomidine/butorphanol or midazolam/ketamine (maintains venous pressure) injected SC or IM), once the chinchilla has fully relaxed, I will use the lateral saphenous vein by choice, to inject the pentobarbitone, to allow the owners to stroke the animals head.

I advise allowing the bonded cage mate to see its companion after euthanasia. I advise clients that I believe it allows the cage mate to accept that its companion will not return. Over the years, many owners have reported to me they felt the cage mate was looking for its companion constantly after losing them and certainly anecdotally this is reduced when they get the opportunity to 'say goodbye'. Whether this is purely anthropomorphic or not is up for debate, however it demonstrates to your owners that you care, and you listen to their concerns.

Many owners will opt for private cremation with return of their pets' ashes and all options that your practice offers for dogs and cats should also be made available to your chinchilla clients. I offer a lock of fur, footprints (coloured ink pad from craft shop and a card). I send sympathy cards if I know the patient and owner well.

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8

Degu

Degus (*Octodon degus*) were brought into the UK in 1950 as a research animal for various human conditions (e.g. social behaviour, brain function, diabetes mellitus, cataracts) and have become popular pets over the last 30 years. They are hystricomorph rodents resembling large gerbils with a brush on the end of their tail, their incisors and cheek teeth continuously grow and the labial surface of the incisors have iron deposition within the enamel resulting in a yellow orange colouration. The cheek teeth are shaped like a figure 8, which is why the Latin name *octodon* (octo = 8, don (t) = teeth) was given to the species. *Inadequate husbandry and nutrition are the leading cause of illness in this charming species*.

They are clean rodents with virtually no smell. The long gestation period is 87–93 days as they birth precocial young and communally nurse their young. Their average lifespan is 5–8 years but a maximum age of 10 years old has been recorded. The maximum recorded lifespan for a degu in the wild is 1–2 years old. The normal body weight is 170–300 g.

Degus are social, burrowing, diurnal rodents, evolved to live in the semi-arid, savanna and scrub environments of the western slopes of the Andes Mountains in north and central Chile, from sea level up to elevations of 3,500 m although most are found at or below 1,200 m. Degus prefer open, dry, partially rocky areas without too dense vegetation. They are also seen occupying desert edges, or habitat that has been cleared of bush or trees by humans.

During the hot summer months, they may change their activity levels to be primarily crepuscular to escape the high temperatures. In the wild they are never active at night and during the day they will retire to their burrows once the ambient temperatures rise above 25°C (77°F) to escape the heat as a behavioural adaptation to regulate core body temperature.

They are an extremely social species, forming family groups of between two to five females with one to two males in the wild. Colonies can have up to 259 (average 40–80) individuals who share a feeding range and a system of tunnels. Tunnels are preferentially dug during the winter months as the soil is softer due to the higher rainfall. Degus will form digging chains with other degus to form tunnels at a depth of 15–60 cm below the ground level, the tunnel diameter is 8–10 cm and they are often a total of at least 2 m in length with widened chambers used as sleeping areas and larders. Nesting material is accumulated in the sleeping areas. There are multiple entrances and exits hidden beneath a scrub or behind a rock.

Studies suggest that degu sociality is non-kin based. *They spend extended periods of time (5–10 minutes at a time) allogrooming* and have been shown to perform allonursing of young in captivity.

They have large home territories of 16,700 m^2 per six degus. Compare that to our average room size of 9 m^2. The centre of the home territory will have a raised mound consisting of stones, twigs, earth, and faeces, which is used as a vantage point. They have favoured sites to perform sand bathing which also serves as an area of territory marking.

Wild diet

Degus spend on average 5–6 h per day foraging for food (accounting for 46% of time spent outside of the burrow), mainly at ground level but they will climb branches and shrubs to forage. They evolved to consume fibrous plants, leaves, stems, and seeds that are found on the mountainous slopes and their *wild diet is naturally high in fibre and low in sugars and starch*. Their *diet varies seasonally*, feeding on up to 66 different plant species.

Their mainstay diet is grasses and herbs, during the summer they consume more succulents (increases their water consumption) and they have a highly adapted gut that allows them to get adequate nutrition from low-quality food sources. *Degus perform coprophagy, eating approximately 38% of faeces produced in a 24 h period, although the majority (87%) of this occurs at night*.

Adaptations for water conservation

Degus (like chinchillas) have several physiological adaptations for limited water availability. They produce highly concentrated urine, very dry faeces, and they have lower evaporative water loss compared to other rodents

Companionship

Degus are a distinctly social species and have an innate requirement for conspecific companionship and will spend 5–10 minutes at a time grooming each other. *If kept without social interaction or physical stimulation they may become aggressive or self-mutilate, such is their requirement for companionship*.

They display a diverse range of communication techniques, including up to 15 unique vocalisations (which humans can hear), olfactory cues (urine and scent glands; females are better at perceiving individual odours), and body language. The ventral fur of the body is paler and reflects UV light and may be used for communication. Their urine also reflects UV light and is therefore used for communication and territory both by olfaction and vision.

A minimum of two degus should be kept together, but groupings of three to five degus have been suggested as a more natural grouping size. Groups of either same sex, or mixed sexes can be kept but should be introduced when they are young (unless from same litter) to minimise risk of fighting. Males in mixed sex groupings should be castrated.

Degus enjoy human interaction (see Figure 8.1) but many do not enjoy being handled.

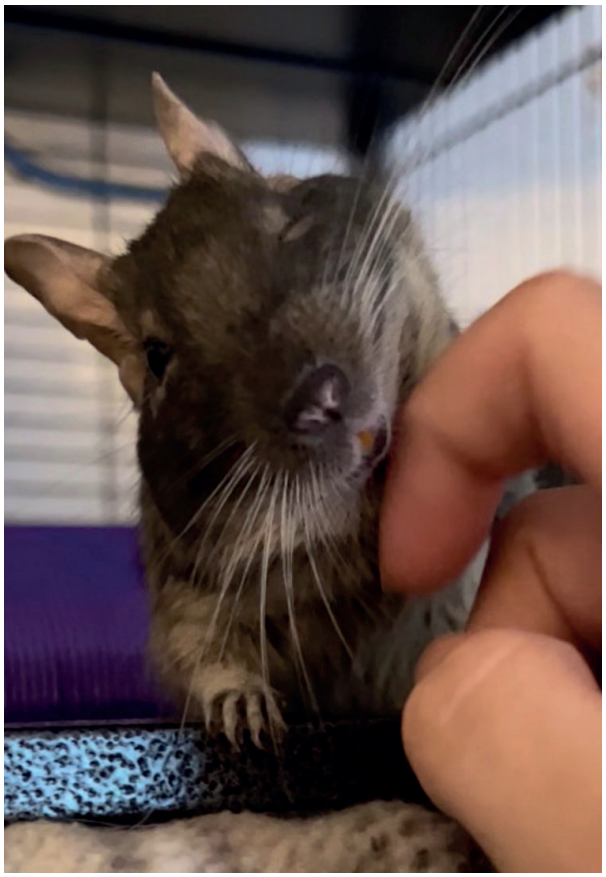


Figure 8.1 This degu is enjoying a face rub from his owner.

Enclosure and enrichment

Degus are extremely active and agile. They require large multilevel cages to meet their needs. Minimum size recommended is 1 m × 0.85 m × 1 m for two degus, with a larger enclosure required for more animals. *Ideally the cage should be much bigger than this and if only the minimum is able to be provided, the degus must be given at least 1 h daily of exercise outside of the cage*. To maximise the benefit of the exercise time, the ideal times to allow out of the cage include morning or early evening to mimic natural peaks in activity.

Chinchilla cages are often used for degus and *it is important that shelves, floors, and levels provided are solid rather than mesh floored, to prevent pododermatitis or injury*. Some websites provide deep based large enclosures that are suitable for degus see https://www.degutopia.co.uk/ for further cage options or ideas for homemade enclosures.

Ropes and thick branches (with leaves still attached) can be added to create a more complex 3D environment allowing a more normal expression of climbing behaviour. Safe species for branches include apple, hazel, hawthorn, kiln dried pine, pear, and linden (lime).

Degus like access to hammocks, which can be purchased or readily made from a triangle of fabric, loops sewn at each corner, and attached to the cage with wire. It is *important to ensure there is no uninterrupted height of 60 cm or more to reduce risk of injuries from falls*.

Wild degus sleep in widened chambers in their tunnel network. Cardboard boxes or wooden boxes can be used to mimic these chambers, allowing a hidden, safe place to rest. Both options are likely to be chewed. Alternatively, a large, open terracotta plant pot placed on its side can be used as one of the resting hides. The resting sites can be lined with hay, straw, or shredded paper, although the degus may decide to redecorate to their preference. Cardboard tubes can be given to provide another outlet for their innate requirement to chew, and to help mimic tunnels. Cardboard tubes are often free and provide good enrichment.

Recommended bedding includes dust extracted shavings and shredded newspaper, which should be at least 15 cm deep to allow digging. Whole sheets of kitchen roll will provide entertainment and enrichment, allowing the degus to shred to their preferred size and line their sleeping area.

Solid wheels can be provided for running. *To prevent painful spinal conditions, it is important to provide a large enough wheel that they do not have to bend their back while running ideally at least 30 cm in diameter*. They should be able to run with a straight back and without having to lift their head. Degus will on average run 4.5 km per day on a wheel! If owners are having trouble sourcing a large enough wheel, https://www.degutopia.co.uk/ provides information on how to make their own metal wheel. *Degus will generate a lot of body heat running, and they will invert their circadian rhythm to become nocturnal if the ambient temperature during the day is too warm for running. If*



Figure 8.2 One of my client's degu cage set inside a home designed playpen. The box with the pink lid is a dig box which is opened when the degu is out of the cage.

owners report their degu is spending much of the night running in their wheel, it is necessary to move the enclosure to a cooler room during the day.

Degus have *higher cognitive capacity than is reported in other rodents*, and can be trained to use tools, recognise colours, and learn new behaviours, *therefore it is even more vital they are provided with enrichment* in the form of toys, puzzle feeders, and opportunities to climb.

Parrot toys, ladders, jingle balls made from sisal, toys made from sisal or loofah, or homemade toys from cardboard are all good options to provide enrichment. Empty coconut shells can be hung in the enclosure to provide opportunities to gnaw. A plastic tub can be filled with hay to give an opportunity to play and forage.

Supervised time out of their enclosure is important, and playpens (see Figure 8.2) with dig boxes can be provided or attached to their main enclosure. Dig boxes allow expression of natural behaviour such as digging and tunnel construction. They are large, deep boxes with solid bases, filled with a mixture of organic soil and sand. Large marbles can be given during out of cage play time, many degus will enjoy chasing them across the floor. Feather sticks that are popular with cats are also popular with degus, providing an interactive toy for owners to play with their pet.

> Exercise balls should not be used. Degus are a prey species and although humans enjoy 'zorbing' I am quite sure we would not feel the same if the activity was performed in a real life Jurassic Park! Yet we expect a prey species to run happily in a ball they cannot escape from and have no ability to hide in. The degu does not know the family cat cannot eat it in the ball, or the family dog who is chasing the ball or the young child who kicks the ball is not going to eat it.

Degus should be maintained with a 12:12 h day:night schedule.

Dust bath

Degus require regular access to dust bathing to care for their fur and remove the naturally secreted oils. *Unless there are fresh traumatic injuries or surgical wounds, the dust bath should be offered for 20–30 minutes twice weekly.* It is important that the dust bath is removed from the cage after the

allocated time, as they may also use the dust bath as a latrine.

Play sand is not suitable for dust bathing. The most recommended substrates for dust baths are: Fullers Earth, volcanic ash, and commercial chinchilla dust. The pan should be large enough for the degu to roll around and the depth of the dust should be 3–5 cm.

Diet

Degus evolved to consume low-quality, fibrous plants, leaves, stems, and seeds that are found on the mountainous slopes, which varies seasonally, feeding on up to 66 different plant species. They have a highly adapted gut which allows them to get adequate nutrition from a low-quality food sources. They perform caecotrophy directly from anus, with approximately 38% of faeces reingested. *In captivity we should try to mimic this diet as closely as possible*. Proprietary mixes of seeds to grow are available online to allow owners to grow their own diet to mimic as close to a natural diet as possible. *Any dietary change should be implemented slowly to prevent bloat or diarrhoea*.

The mainstay of pet degus' diet is ad lib high-quality hay and dried grasses supplemented with ideally degu pellets (10 g/degu/day), and small amounts of low sugar vegetables/green leaves can be given three times weekly to satisfy their requirement for essential fatty acids. Degus cannot survive on a hay only diet. For ease of feeding, most 25 ml shot glasses will hold 10 g of pelleted diets. If fed pellets ad lib they will preferentially consume pellets over hay which leads to a decrease in water consumption as well as increasing the risk of dental disease.

Nuts should be avoided, and seeds should be limited to the occasional pumpkin seed due the fat levels. Once a month an in-shell hazelnut may be given to provide environmental enrichment as an opportunity to gnaw and forage.

Muesli diets should be avoided as they allow selective feeding and are generally low in fibre and too high in carbohydrates. Diets too low in fibre and/or too high in carbohydrates cause imbalances in gut flora (which may cause diarrhoea), obesity, and poor dental attrition leading to coronal overgrowth and dental disease (periodontitis and caries formation). Degus are also predisposed to developing diabetes mellitus if fed a diet too high in carbohydrates.

Barbering due to inappropriate husbandry

Degus are known to self-mutilate by barbering as a primary behavioural disorder due to anxiety. Known triggers for barbering are insufficient cage size, lack of exercise, lack of burrowing substrate, being kept as a solitary animal, inappropriate light dark cycle, or a high calorie low fibre diet.

Water

Hagen *et al*. (2014) found that degus had no preference to how water was presented and drank equal amounts via bowl and bottle. Any restriction in water intake is likely to decrease food intake, therefore ad lib water should be available at all times.

Water intake is around 100–160 ml/kg/day on a hay and pellet diet, with lab degus averaging 23 ml/animal/day. Water intake will increase with increased fibre intake, boredom, pain, or existing diseases, e.g. diabetes mellitus.

Ultraviolet light

Ultraviolet (UV) B lighting is rarely provided for pet degus but is commonly used for laboratory animals. *Given the fact that degus can see into the UV spectrum, evolved to live in an area of high UV exposure, will sunbathe in the wild, and UVB can help with calcium homeostasis, we should consider the provision of UV lighting for pet degus*. Work still needs to be done to establish exact guidelines, but it may be sensible to follow advice given for guinea pigs for UV lighting.

Vertebrates with hypovitaminosis D are susceptible to diseases that affect the musculoskeletal, cardiovascular, and immune systems. *Acquired dental disease is so common in degus it is possible that chronic hypovitaminosis D may play a role in nutritional osteodystrophy of the skull*.

Sexing

Male

Male degus have a longer genital–anal distance (Figure 8.3). The prepuce is conical in shape and although there is no true scrotum, there are two movable sacs lateral to the anus (post-anal sacs) which the caudal epididymis can drop into. The penis can be manually extruded, is 16 mm long and has spicules. The spicules are testosterone dependent, appearing at 2.5 months old and becoming fully developed by 3.5 months of age. There is an os penis.

Female

Female degus have a shorter genital–anal distance (Figure 8.4). The urethral process protrudes and may



Figure 8.3 Male degu, note the longer genital anal distance. There is no true scrotum in this species.

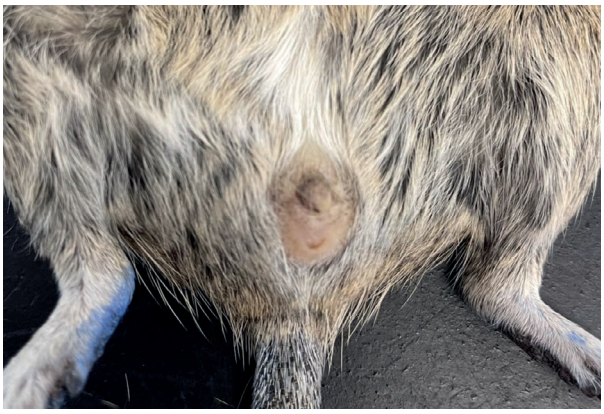


Figure 8.4 Female degu, note the shorter genital–anal distance. The urethral process protrudes and may be confused as a prepuce.

be confused as a prepuce. The vaginal opening sits between the urethral process and anus but will be covered by a membrane unless in oestrus. There are four pairs of nipples, three pairs laterally on the thorax and one pair inguinally. During oestrus the perineum swells and changes colour from a dull flesh tone to a deep red colour but there is no vulval swelling, the level varies greatly between individuals. *A whitish vaginal discharge can be seen during oestrus and should not be confused with a pyometra*.

Contrary to several authors, *the degu does indeed have a regular cyclical oestrus occurring every 17–21 days and they are spontaneous ovulators*. Young degus have shorter cycles of 17.5 days compared to 21.2 days to older degus. Degus show increased levels of activity including wheel running, when in oestrus (Mahoney *et al*., 2011).

Preventative health care

Regular weighing by owners may help pick up trends in subtle weight losses, indicating that the degu may have an underlying condition. Regular veterinary check-ups are advisable due to their ability to hide pain, although compared to the other rodents, degus are often medically robust apart from dental disease.

Hospitalisation

With any species, but especially prey species, *it is important to minimise stress during handling and while in the clinic. Stressed animals have a high sympathetic tone which can counteract effects of sedatives, predispose to vasoconstriction, excessive myocardial work, and arrhythmias*.

Hospitalisation is similar to guinea pigs and care should be given not to overheat as they are prone to heat stroke if maintained in an environment warmer than 27°C (80.6°F), the recommended temperature for the ward is 19–21°C (66–70°F).

Venipuncture sites

Many exotics texts describe anaesthetising the degu and performing a cranial vena cava stick to draw blood. This carries the risk of death due to uncontrollable haemorrhage. Laboratory vets and technicians have perfected this technique with very few bad outcomes but they are performing this very regularly whereas clinical veterinary staff do not. Personally, I would not do this to one of my own pets therefore I will not do it to a client's.

As a rule of thumb, in a healthy degu 1% of the body weight (in grams) is the volume of blood that can be safely taken, in ill individuals it is recommended to take a smaller volume. Biochemistry may be run in-house using dog or cat panels. See Tables 8.1 and 8.2 for degu reference ranges.

Degus find restraint for venipuncture very stressful and may bite, so consider an anaesthetic to reduce stress and risk of injuries from struggling. EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) can be applied to facilitate venipuncture.

My vein of choice is the lateral saphenous vein, but other options include the jugular vein, femoral vein, and cephalic vein. The gingival vein may also be used.

Lateral saphenous vein is my preferred choice for intravenous catheter placement using 24–26g catheters. Alternatively, an intraosseous (IO) catheter may be placed under anaesthesia (see Dehydration section).

Drugs contraindicated in degus

Antibiotic induced dysbiosis and enterotoxaemia

Narrow spectrum antibiotics (especially ones with predominantly activity against Gram positive bacteria) such as: amoxicillin, ampicillin, cephalosporins, clindamycin, erythromycin, lincomycin, and penicillin should never be given orally. Oral administration causes an overgrowth of Gram negative bacteria, especially *Clostridium*, which results in an often fatal enterotoxaemia and haemorrhagic typhlitis. If in doubt about the oral safety of an antibiotic always check the formulary prior to administration. That being said, even supposedly 'safe' antibiotics can cause issues with the gut microbiome in some individuals. See Table 8.3 for commonly used antibiotics in degus.

Steroids should be used with caution, if at all.

Species-specific anatomy/behaviour

Degus have virtually no odour, never scratch, and rarely bite.

Male degus are approximately 10% bigger than females.

Degus may become aggressive or self-mutilate unless provided with conspecifics and physical stimulation.

Degus are hystricomorphs with elodont dentition (elodont: continuously growing throughout life). They have two incisors in the maxilla and two in the mandible and

Table 8.1 Degu biochemistry.

BIOCHEMISTRY (Heatley & Russell, 2020)	REFERENCE RANGE (UK units)	REFERENCE RANGE (US units)
ALKP	299.4–471.86 IU/l	299.4–471.86 U/l
ALT	27.55–40.12 IU/l	27.55–40.12 U/l
AST	48.5–73.65 IU/l	48.5–73.65 U/l
AMYL	788.62–996.41 IU/l	788.62–996.41 U/l
GGT	1.8–3 IU/l	1.8–3 U/l
LDH	562.87–795.21 IU/l	562.87–795.21 U/l
CK	752.69–2627.55 IU/l	752.69–2627.55 U/l
LIPASE	35.93–50.9 IU/l	35.93–50.9 U/l
GLU	8.79–9.97 mmol/l	158.38–179.64 mg/dl
CHOL	2.27–2.76 mmol/l	87.64–106.56 mg/dl
TRIGYLCERIDES	1.47–2.16 mmol/l	130.09–191.15 mg/dl
BA	8.94–18.04 µmol/l	0.527–1.08 mg/dl
TBIL	1.91–2.86 mmol/l	0.11–0.17 mg/dl
UREA	14.16–15.96 mmol/l	39.66–44.71 mg/dl
CREA	51.94–60.96 µmol/l	0.59–0.69 mg/dl
TP	52.68–56.74 g/l	5.268–5.674 g/dl
ALB	32.97–34.97 g/l	3.297–3.497 g/dl
GLOB	19.05–24.36 g/l	1.905–2.436 g/dl
CA	3.075–3.26 mmol/l	12.3–13.07 mg/dl
PHOS	1.97–2.37 mmol/l	6.1–7.34 mg/dl
K	4.1–4.8 mmol/l	4.1–4.8 mEq/l
Na	140.4–144.2 mmol/l	140.4–144.2 mEq/l

Table 8.2 Degu haematology.

HAEMATOLOGY (Heatley & Russell, 2020)	UNITS	REFERENCE RANGE
Red blood cells	10^{12}/l	4.2–13.8
Packed cell volume	%	42–44
Haemoglobin	mmol/l (g/dl)	4.47–9.31 (7.2–15)
Mean corpuscular volume		37.5–54.7
Mean corpuscular haemoglobin concentration		286–422
Mean corpuscular haemoglobin		13.6–17.2
Platelets	10^{9}/l	53–475
White blood cells	10^{9}/l	3.2–19.9
Differential blood count (relative values)		
Monocytes	%	0–8
Lymphocytes	%	9–86
Neutrophils	%	0–5
Eosinophils	%	0–6 m, 0–18 f
Basophils	%	0–10 m, 0–2 f

Table 8.3 Commonly used antibiotics in degu.

Drug	Dose
Azithromycin	30 mg/kg PO q24hrs
Ciprofloxacin	10 mg/kg PO q12hrs
Chloramphenicol	30–50 mg/kg SC, IM, IV, PO q8–24hrs
Doxycycline	5 mg/kg PO (no dairy) q12hrs, long-acting formulation 50 mg/kg q7days SC
Enrofloxacin	10–20 mg/kg PO SC q12hrs (IM painful and may result in sterile abscess/muscle necrosis)
Gentamicin	5–8 mg/kg divided q8–24hrs SC, IM
Marbofloxacin	5 mg/kg PO q12–24hrs
Metronidazole	15–20 mg/kg PO q12–24hrs
Oxytetracycline	50 mg/ kg PO q12hrs
Silver sulfadiazine cream	Topical q12–24hrs
Trimethoprim sulfamethoxazole	30 mg/kg PO q12hrs

impressively grow approximately 36.5 cm/year in healthy individuals. Dental formula: 2 × (I 1/1; C 0/0; P 1/1; M 3/3) = 20. Degus can develop elondontomas associated with the maxillary incisor apice.

The tight cardiac stomach sphincter prohibits vomiting or eructation. There is no need to starve for an anaesthetic.

They are hindgut fermenters.

It is only the cervical section of the thymus that persists into adulthood, is large and bilobed.

The pupil is a vertical slit instead of round.

They have five toes on each foot, with the medial toe having a nail instead of a claw.

The proximal ends of the tibia and fibula are fused.

Male degus have open inguinal canals. Female degus have two uterine horns and two cervices (externally will look like one cervix). They have long gestation periods (87–93 days), birth precocial young and will communally nurse young.

They have several adaptations to cope with water deprivation. They produce very concentrated urine. They produce small, dry, dark, hard faecal pellets. They can reabsorb water from expired air via adaptations to nasal cartilages.

Flight is their main defence mechanism although they may bite if they are not used to handling.

The skin on the tail is loosely attached and tail slip injuries can occur if the tail is grasped.

> Degus hide illness and pain.

Degus can jump off the consulting table leading to injuries.

The spleen has a glandular appearance on histopathology.

They have relatively large kidneys, and their adrenal glands (round to ovoid) are large compared to other rodents of a similar size.

They have a divergent insulin structure and are very intolerant to sugars as a result.

Pain in degus

One of our jobs as veterinary professionals is to *actively look for pain*. In Chapter 1 we discussed that all vertebrates have the same anatomical and physiological pain pathways.

There is very little in the literature on specific studies performed in degus investigating pain and analgesic efficacy. Doses are extrapolated from other rodents (I tend to use the same doses that I use in guinea pigs) and regular reassessment to ensure adequate control of pain is being achieved is vital. Most degus have open round eyes and sleek coats. Pain may result in reduced grooming and squinting of the eyes. See Figure 8.5.

Osteoarthritis in degus

Osteoarthritis is a degenerative condition affecting the cartilage and bones of a joint. It is commonly referred to as the wear and tear arthritis. Any species with a skeleton can develop osteoarthritis, *species that actively hide pain mean that we have to actively look for skeletal pain in these species*.

Osteoarthritis of the temporomandibular joints will cause secondary dental disease due to pain caused by chewing. Osteoarthritis of any joints may cause secondary dental disease due to a decrease in appetite, which as it happens slowly, will often not be noted by the owner.



Figure 8.5 The degu on the right is squinting its eyes and has a poor appearance to the coat indicating lack of grooming compared to the degu on the left. The righthand degu was suffering from severe dental disease.



Figure 8.6a Radiograph showing osteoarthritic change in the right shoulder and bilateral elbow changes.



Figure 8.6b Radiograph showing osteoarthritic changes in both stifles.

Radiography can be used as in more familiar species. See Figures 8.6a & b.

Analgesia

Doses that I use are extrapolated from guinea pigs (start at the low end if ranges offered) and regular reassessment to ensure adequate control of pain is being achieved is vital. See the section on analgesia in Chapter 3 and Table 8.4. Acupuncture works well as part of a multimodal approach to pain (Figure 8.7).

Clinical exam

I always place a towel or blanket on the table to reduce the patient's stress. *Prey species have evolved to be prey, therefore have an innate requirement to escape predation at a split-second notice. This is why they find a slippery surface so frightening, if they cannot find purchase, they cannot escape predation. Using the blanket or towel reduces the chance of them injuring themselves or you as their handler.*

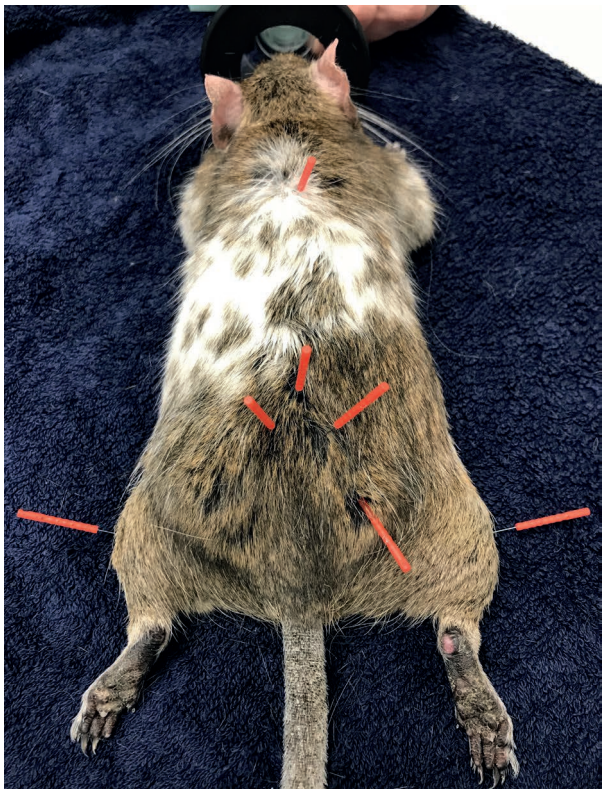


Figure 8.7 Acupuncture works well as part of a multimodal approach to pain.

Table 8.4 Degu analgesia.

Analgesic	Dose
Meloxicam	0.5–1.5 mg/kg q12hrs PO SC
Paracetamol (acetaminophen)	25–50 mg/kg q 8–12hrs PO
Buprenorphine	0.05 mg/kg SC q8hrs
Gabapentin	10–30 mg/kg q8–12hrs PO
Maropitant	2 mg/kg SC
Methadone	0.5–2 mg/kg SC q4hrs dependant on pain scoring
Tramadol	5–20 mg/kg q6–12hrs
Lidocaine	2 mg/kg (maximum 4 mg/kg)
Bupivacaine	1 mg/kg (maximum 2 mg/kg)
Acupuncture	

I will look at the degu face on to check for facial symmetry.

Personally, I prefer to perform the rest of the examination with the degu on my knee (if friendly and comfortable with handling) or on the table. I perform the same clinical exam, in the same order that I do for any other species (except that I perform oral examination of the molars last). See Chapter 3 for clinical exam in guinea pigs for more detailed information on clinical exam (but not the oral examination). See video 8.1 for skeletal pain examination.

Table 8.5 Degu biological data.

Parameter	Reference range
Average longevity	5–8 years
Body weight	170–300 g
Respiratory rate	45–80 bpm
Heart rate (*higher heart rates are found in younger animals of 1 year and the heart decreases with advancing age*)	100–340 bpm
Body temperature	38–39°C (100.4–102.2°F)
Maintenance fluid therapy	100 ml/kg/day
Sexual maturity	3–4 months
Gestation	87–93 days

Performing conscious oral clinical exam

After wrapping the degu in a burrito, I will ask an assistant (which may be the owner) to hold the pet vertically with its back nestled against their breastbone supporting the thorax and its hindquarters. I use the medium metal otoscopic cone.

Restraint of the head is important. I use my non-dominant hand (for me that is my left hand) to restrain the head, I place the palm of my hand flat onto the back of the skull and use my index (pointer) finger and thumb to hold onto the point of the zygomatic arch just distal to the eyes. I then pull gently caudally with my index and thumb.

The maxillary incisors are shorter than the mandibular and are squared at the end, mandibular incisors are approximately 1.5–2 times the length of maxillary incisors. The incisors should be yellow/brown in colour by the time they are 6 months old, new born degus have white incisors and the colour gradually changes with age. *Patches of white or white incisors may be associated with a calcium deficiency or a diet with an inverse or inappropriate calcium phosphorus ratio (should be 2:1).*

> Degus sometimes have food in their mouths, which is completely normal. However, it does mean that the oral cavity must be swabbed out with cotton buds before the teeth can be visualised.

Degus normally have an almost horizontal occlusal plane; the molars are cream coloured and should be approximately 2 mm above the gingiva in healthy animals and the teeth are symmetrical.

I place the otoscope cone into the right diastema just caudal to the upper incisors. Examine the soft tissues for any wounds, ulceration, or bleeding. Move around the degu, without taking the scope out, and perform the rest of the clinical exam to include the both upper and lower arcades.



Figure 8.8 The lower right premolar is overgrowing and flaring lingually. A Thudicum nasal speculum is being used as a cheek dilator.

Maxillary molars flare towards the cheek when they overgrow. The lower arcades grow towards the tongue, although in my experience can also grow vertically (see Figure 8.8). Dental caries are brown to black in colour and are seen more easily on the occlusal surface.

Many vets (including myself) used to focus on looking for spikes when performing this exam. Now *I advise looking for asymmetry (seeing spikes is a bonus)*. Asymmetry is often easier to appreciate. If they have pain on one side, they will preferentially chew on the other side which leads to asymmetrical wear of the continuously growing molars. If in doubt I advise full oral exam under sedation or general anaesthesia (once stabilised if anorexic) including skull radiographs. This is discussed fully in the section on dentistry.

The ears can be examined with a smaller speculum.

Anorexia and gut stasis (ileus)

Anorexia is a true emergency in degus which can lead to gut stasis, a negative energy balance and fatty liver syndrome (very prone to this especially if overweight) developing within 12–24 h. It is important to remember that anorexia is a symptom not a condition in degus.

Unfortunately for us, *anorexia is a nonspecific reaction to stress which can be physical (pain anywhere and illness) or psychological* (separation from cage mate, change in environment). A good clinical history and thorough clinical exam will often help direct your diagnostic tests. *I assume any anorexic degu is in pain somewhere.*

Stabilisation of the anorexic degu

The mainstays of stabilisation are analgesia, assisted feeding, correction of dehydration, and prokinetics and diagnosis of initiating cause.

Dehydration

Severe dehydration will cause sunken eyes, a delayed capillary refill time, and a prolonged skin tent; however, milder cases of dehydration may have no obvious signs. Chapter 4, Dehydration section should be consulted for signs of dehydration in rodents.

> Oral rehydration is often underestimated, but in cases of mild dehydration, oral rehydration is highly successful.

I use 100 ml/kg/day as fluid maintenance volume in degus.

Fluids should be warmed to body temperature (38–39°C (100.4–102.2°F)) prior to administration. *I do not agree with subcutaneous (SC) fluid administration. It is incredibly painful* and dehydrated patients will not absorb fluids via this route as efficiently.

Intraperitoneal fluid administration can be given once (repeated administrations are not advisable). Maximum volume administered this route is 5 ml using the right caudal quadrant as per other rodents to minimise risk of entry into the gastrointestinal tract.

An intravenous catheter may be placed in the jugular, or lateral saphenous vein but it is challenging. Degus find restraint for intravenous (IV) catheter placement stressful and consideration should be given to a brief anaesthetic using isoflurane or sevoflurane to reduce stress. EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) application will help facilitate placement. Their veins require 24–26g catheters.

IO fluid administration is much easier. IV or IO administration of fluids should be given to any degu considered to be >5% dehydrated.

IO catheter placement

My preferred IO site is the proximal tibia, as the tibial crest is easy to find but an alternative site is the proximal femur. *General anaesthesia (if not moribund) is required as placement is considered painful.* After surgical preparation of the site, instil lidocaine (2 mg/kg) at the level of the periosteum. Using a 25g needle (surgical steel suture material (sterile) as a stylet, or replace the needle) is placed in a normograde fashion, a radiograph should be taken to ensure correct placement as inadvertent puncture of both cortices is a common complication in the tiny species. *IO placement is contraindicated in any patients that have sepsis, osteomyelitis, or metabolic bone disease.*

IO catheters can be connected to syringe drivers or boluses can be administered every 30–60 minutes. The bag of fluids, or syringes of fluids, can be maintained at the correct temperature using a baby bottle warmer. *Ongoing analgesia is important, IO catheter flushing and administration of fluids is painful in humans therefore should be considered painful in other species.*

Hydration status should be assessed throughout the day, which enables you to alter the rate and volume of fluids therapy as required. My intravenous fluid of choice in most cases is saline. The total fluids required can be calculated and divided into syringes to be administered via boluses.

Assisted feeding

The term assisted feeding is preferable to force feeding when discussing with owners. There are several high-quality, purpose made foods available such as: Oxbow Critical Care, Supreme Recovery or EmerAid Herbivore. All three are high in fibre to promote gut motility, are palatable (although individuals may prefer one over another), and easy to prepare. Feeding syringes with widened nozzles are available from wholesalers made by Supreme Pet Foods and Oxbow.

Some degus will require wrapping in a towel (burrito technique) for assisted feeding, with recommended volumes of 10–20 ml/kg/feed three to four times daily to support gut motility. Some individuals will tolerate smaller, more frequent feeds better.

Dried grass, hay and the degus' normal diet should always be readily available to encourage the degu to eat on its own. HEV'N (Holistic Exotic Veterinary Nutrition) makes and supplies a Gastrointestinal Mix, which is a mix of dried plants that have a prokinetic effect on the gastrointestinal tract.

Prokinetics/gastric protectants

Drugs to help promote normal gastrointestinal motility can be administered.

Metoclopramide (0.5–1 mg/kg q6–8hrs, increases gastric emptying and small intestinal motility) is recommended and I find it useful. Cisapride (0.5 mg/kg q8hrs) is recommended as a prokinetic for rodents in many texts.

I will use metoclopramide until the degu is eating and passing normal faeces.

Ranitidine (2–5 mg/kg q12hrs) is recommended and is very useful in rabbits due to its dual effect of being a prokinetic as well as lower gastric acid production and may be useful in degus. At time of writing this drug is only available from compounding companies. Other viable options which are used in rabbits, are cimetidine (5–10 mg/kg q6–12hrs) or famotidine (0.5–1 mg/kg q12–24hrs).

I will add in a gut protectant if I am concerned about the presence of ulceration. Sucralfate 25–50 mg/kg q8–12hrs can be used in suspected cases of gastric or duodenal ulceration. Sucralfate can be sourced from compounding companies such as Summit or Bova (UK) or Doctors Foster and Smith (USA).

Some owners prefer grinding the tablets and mixing with water for administration. Alternatively, the pill can be administered via a pill giver or a 1 ml syringe with the

nozzle and top cut off. Compounding companies make palatable paste versions of many drugs.

Once stable (eating and passing faeces) some owners will decide against further investigation. Depending on your findings during the clinical exam this could be an acceptable approach. For example, if you found spinal or hip pain then continuing on with long term analgesia without radiographs is absolutely fine if the animal is clinically stable.

> However, if dental disease is suspected then owners should be counselled this approach would be completely inappropriate, full investigation is required.

General anaesthesia

Reducing risks associated with general anaesthesia starts before admission.

Degus cannot vomit and need a near constant supply of fibre to maintain gut motility therefore they should not be starved before an anaesthetic. Owners are used to starving themselves or their dogs and cats prior to surgery, so we need to be clear when we book degus in for surgery that they should not be starved. Advise owners to bring a 'packed lunch' of favourite and familiar foods.

Bonded cage mates should always be admitted, not only will this *reduce stress during the hospitalisation period, help protect the bond between conspecifics, it also has been shown to reduce healing times and increase coping ability for aversive or painful experiences* (Denommé and Mason, 2022).

Allow degus at least an hour to acclimatise to the ward after travelling to the clinic to reduce stress levels prior to induction.

With only a few exceptions (bloat, or a foreign body), ill degus should be fully stabilised (rehydrated, passing faeces and starting to eat) before an anaesthetic should be administered.

Pre-anaesthetic blood tests

The need for pre-anaesthetic blood tests is debated amongst vets. Personally, I advise they are an option but acknowledge in the young healthy individual the vast majority will be within reference ranges. The benefit is that it will give you a personalised reference range for this individual patient and as rodents are so good at hiding disease, occasionally we get unexpected results which may alter our plans. Blood can be taken from the cephalic vein, femoral vein, or the lateral saphenous vein, but many degus will require a general anaesthetic (GA) for venipuncture. There is nothing wrong with aseptically dripping the blood from a needle into the collection tube. Biochemistry can be run 'in-house' using the same pre-op profiles used for dogs and cats (see Tables 8.1 & 8.2 for reference ranges).

Pre-emptive analgesia/prokinetics

Once admitted (and if not already on medications) administer meloxicam 0.5–1.5 mg/kg and/or another analgesic if appropriate depending on the surgical procedure, and metoclopramide 1 mg/kg. These can either be given orally or by SC injection but my preference is oral.

Pre-medication

Whether I administer a pre-medication is dependent on the stress levels of patient and the surgical procedure being performed. *It is important to consider the stress of being handled and being injected. Rodents find injections very stressful and as they have small muscle masses intramuscular (IM) injections are very painful and may need to be given in multiple sites depending on volume*. I think very carefully before I administer pre-medicants in rodents as I have to consider the type of surgery (and which analgesics will be required) and decide whether the stress and pain of the pre-med is worse than the mask induction. In my experience mask induction while wrapped in a towel burrito results in a fast, seemingly minimal stress induction, whereas injections always result in vocalisations and obvious stress. The drugs used for this are the same drugs we use in more familiar mammals. The doses can be found in Table 8.6.

Local anaesthesia for muscle splash blocks or infiltration

Degus do not have an increased susceptibility to the toxic side effects of local anaesthesia, it is simply their small size that makes it much easier to accidentally overdose them.

Combining the drugs allows for greater effect with a lower recommended dose. Lidocaine has faster onset but a shorter duration of action (60–120 minutes) whereas bupivacaine has a slower onset of action but a longer duration (180–480 minutes). Mixing a maximum of 4 mg/kg of lidocaine with 2 mg/kg of bupivacaine in a syringe then

Table 8.6 Premedicant dose.

Drug	Dose and route	Reversal
Midazolam	0.4–2 mg/kg IM SC	Flumazenil 0.1 mg/kg SC
Midazolam + butorphanol	0.2–0.8 mg/kg + 0.3–0.5 mg/kg IM	Flumazenil 0.1 mg/kg SC
Medetomidine + butorphanol	0.05–0.1 mg/kg IM + 0.3–0.5 mg/kg IM	Atipamezole 0.5–1.5 mg/kg IM SC

diluting the mixture with saline to increase the volume of the solution and allow easier splash blocking (exactly what it sounds like). Drip the solution onto the wound in the muscles) or infiltrate an area. Pulling up the required dose and pre-diluting, prevents accidental overdose.

Induction

There are several anaesthetic protocols available for use in degus. There is an argument to be made that the safest anaesthetic is the one you are used to. Personally, I find that gaseous induction has less stress associated with it due to reduced handling and no prior injection.

My preferred inhalational gaseous anaesthetic is isoflurane. Reportedly, most rodents react more favourably to the smell of sevoflurane but in my experience, many dislike the smell just as much and in the UK, isoflurane is the only licensed gaseous inhalational anaesthetic for rodents.

My preferred method of induction is to burrito the degu and use a face mask. Have cotton buds to hand to swab the mouth clear of food or saliva post-induction. Alternatively, use a large dog mask as a small induction chamber.

Allow four breaths of 100% oxygen (this is all that is required to fully saturate the blood with oxygen), then turn the vaporiser straight to 5% isoflurane. Initially the degu may wriggle but it will quickly become induced (most are less than 40 seconds) with minimal stress. In the vast majority of cases the degu will not have passed urine or faeces (which frequently occurs due to stress and commonly passed when induction chambers are utilised.).

Turn the vaporiser to 2.5% and assess anaesthetic depth (using the same reflexes we are used to in more familiar mammals, a moistened cotton bud can be used to check the blink reflex), adjusting the setting on the vaporiser as necessary.

The cornea should be protected by applying an ocular lubricant.

Intubation

Intubation is challenging due to the palatal ostium but can be done visually using an otoscope or endoscope and a 16 or 18g IV catheter with the stylet removed. I do not routinely intubate degus.

Maintenance

I use a T-piece circuit, with an oxygen flow of 1.5 l/minute Isoflurane 2–4% as required.

Monitoring

Monitoring the anaesthetic is the same as for more familiar species. Multiparameter monitoring is possible. MAI Animal Health make Vetcorder Portable Monitor which is suitable for all species, including dogs and cats, for under £1,000. It can record: an electrocardiogram (ECG), heart rate, pulse oximetry (placed on tongue), and rectal temperature. If recording an ECG rather than using Alligator clips (which can traumatise the skin), insert small gauge needles through the skin (25g) and attach the clips to the needles.

If that is outside the practice's budget, a soft, flexible tipped rectal thermometer can be used to monitor rectal temperature. *Hypothermia is a major risk factor in anaesthetic deaths*. A human foetal Doppler can be purchased online (I used eBay) for around £50, ensure you request the 8 mHz probe rather than the standard 2 mHz one. An infant stethoscope can be used to monitor heart rate if the Doppler is not an option. Respiratory rates can be counted and pulse oximeters can be used from other species but they may struggle to get an accurate reading due to the high heart rate *(100–340 beats per minute in degus, the higher heart rates are found in younger animals of 1 year and the heart rate decreases with advancing age)*.

Theatre

Degus have a high surface area to volume ratio which means they *lose heat rapidly*. The resulting hypothermia can cause fatalities. Keeping the ambient temperature in the theatre high, placing a towel on the operating table and using a plastic drape (also allows visualisation of your patient!) are all easy ways to help mitigate heat loss. Minimise the number of times the door is opened as each time it happens the ambient temperature in the room will drop. *Hypothermia leads to prolonged recovery and increased risk of death*.

Positioning

Degus are diaphragmatic breathers. Their small thoracic cavity in comparison to their large abdominal cavity can make breathing more difficult due to the weight of the abdominal organs sitting against the diaphragm. Placing a folded drape or face cloth under the thorax elevates the thorax and allows the abdominal organs to move away from the diaphragm. Ensure the head and neck are straightened to allow for easier breathing.

Surgical preparation

To conserve body temperature, minimise the surgical clip. Only one agent is used for surgical scrub, either chlorhexidine or an iodine-based skin disinfectant depending on the surgeon's preference. We do not use a surgical spirit-based spray as during evaporation this causes loss of heat. Using warm water for the surgical scrub will help reduce heat loss.

Transparent, plastic surgical drapes are ideal as they allow visualisation of the patient while conserving body heat. The required surgical window can be cut into the drape and a surgical spray adhesive is used in lieu of towel clips.

EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) can be applied to the wound postoperatively if no other local anaesthetic infiltration has been performed in the skin. This helps with the immediate post-operative skin pain as most rodents show the most overt signs of pain 12 h

post-operatively. Since I have started using this technique, I have seen a faster return to eating on recovery.

Recovery

Maintain the degu on oxygen until it shows signs of moving. Shivering thermogenesis is a sign of recovery from anaesthesia. Continue to monitor while in an incubator or a warm room (ideally around 26°C (78.8°F)) until up and moving then the temperature of the room can be reduced to 20°C (68°F).

Syringe feed (10–20 ml/kg Oxbow Recovery, Science Recovery or EmerAid Herbivore) once the degu is fully conscious and monitor faecal output.

Advise the owner to monitor food intake and faecal output. If the owner is unable to syringe feed at home, then they must bring the degu back to the practice as soon as possible for admission if they will not eat at home. In the vast majority of cases with good analgesia this will not be an issue.

Dental disease

Our current understanding of dental disease is that it is an *extremely common condition in captive degus* with one study finding 60% of degus suffered from dental disease which when broken down into age groups raised to 75% of degus aged 2 years and above. *Captivity, most likely diet is implicated in the development of dental disease*, however given that its *occurrence seems to increase with age, should we consider that painful conditions such as osteoarthritis may also be implicated*, as failure to adequately control any painful condition is likely to reduce food intake and contribute to secondary dental disease. Not all owners are aware that degus' teeth grow continuously throughout life.

Clinical signs associated with dental disease vary depending on the severity and can include: weight loss, decreased food intake or anorexia, change of food preference, reduction in faecal output or size, drooling (known as slobbers) causing a wet chin, poor fur quality, crusting around mouth or forepaws, pawing at mouth, epiphora (due to pain or impingement of the nasolacrimal duct (NLD)), flattening of fur peri-ocularly, tooth grinding, secondary bacterial conjunctivitis or proptosis due to retrobulbar abscessation secondary to retrograde growth of maxillary molars, entry of the maxillary cheek teeth into the nasal cavity is common which results in signs of rhinitis and dyspnoea.

Degus are also prone to the development of elondontomas which can cause rhinitis and dyspnoea (see Elondontoma). The *ventral border of the mandible should be palpated* as it is extremely common for the teeth to extend through the mandibular cortex.

> It is common for degus suffering from dental disease to be presented with dyspnoea.

Diet

The mainstay of the captive degus' diet is hay but they cannot survive on hay alone. Lowland pasture hays contain higher energy, lower fibre, and lower phytolith (therefore less abrasive) contents compared to the low energy, high fibre, high phytolith, mountain vegetation which forms the diet degus evolved to eat. Coupled with the fact that hay grown under favourable conditions is clean whereas wild diet is contaminated with soil dust which is highly abrasive this may be why in one study 60% of captive degus have dental disease (Jekl *et al*., 2011).

Diet is believed to contribute to dental disease by: inappropriate wear of the teeth, a wider vertical movement leading to abnormal pressures (pellets/nuggets), spikes form in the mouth and over time abnormal pressures result in retrograde unerupted crown growth, and being too high in phosphorus (which causes inverse Ca:P).

Time taken to educate owners on correct diet and husbandry at their routine check-ups can help reduce the risk of dental disease.

Careful examination of the incisors to assess the length, shape, occlusal surface and enamel quality and colour is important. The normal colour of adult degu incisors should be yellow/brown or orange/brown. White incisors may be associated with a diet deficient in calcium, vitamin D, inappropriate Ca:P, or repeated trauma (e.g. bar pinging).

Incisor malocclusion is not as common in the degu as it is in the rabbit and is virtually always associated with molar overgrowth, although some cases may be caused by trauma. Subtle signs such as diagonal wear or horizontal ribbing of the enamel should increase our index of suspicion for dental issues. Malocclusion can be obvious to us but missed by the owners.

An honest, open, and frank conversation is needed. *Not all owners will be emotionally, financially, or time invested enough to proceed with treatment*. Owners need to be prepared to expect repeated dental procedures approximately every 12 weeks, with the second dental procedure occurring in a shorter time frame while the degu becomes used to chewing in a more normal pattern.

In some cases, euthanasia is the best option for that individual. Euthanasia is not a welfare concern (a difficult concept for me to understand as a young vet), however *existing in chronic pain and substandard husbandry is a massive welfare issue*.

In the vast majority of cases there is no cure, management of this lifelong condition with repeated dental surgeries, long term analgesia and husbandry reviews will be required. This requires dedication and financial input from the owner. *Some owners may not be able to cope with this*.

Some early cases of dental disease without root changes may be curable after the first dental procedure, with dietary

corrections so that they are mainly fed on: fresh growing grass, hay and dried grass and *addressing of any underlying medical issues (e.g. pain)*. This type of diet encourages prolonged chewing in a natural chewing pattern. The high silicate phytoliths in the grass promotes normal wear of the teeth. That being said, *it is rare in degus to achieve cure, as it is common for root changes to occur prior to clinically apparent oral changes in this species (Bolhuis* et al.*, 2017) meaning that by the time it is clinically apparent, the root changes are permanent*.

Dental radiographs

Once the degu is stable enough (passing normal faeces but not necessarily eating on its own) I advise general anaesthesia, radiographs of the skull, full exploration of the oral cavity and dental surgery. *Radiographing the skull is compulsory if the owner wishes me to perform a dental*. Radiographs are required to stage the disease. Computed tomography (CT) scanning will give superior information and images compared to radiographs but not all owners are in the financial position to opt for CT scanning, so I work mainly on good quality radiographs.

I radiograph the first time and how frequently I repeat the radiographs depends entirely on the stage of the disease, mild 'root' changes I will repeat the radiograph in 6 months (then in a year if still requiring dental procedures but minimal 'root' changes), severe 'root' changes I recommend repeating the radiographs at 3 months then again at 6 months. Ongoing frequency is dependent on if there is progression of 'root' changes and clinical judgement is utilised.

Apical cheek tooth elongation (technical term for 'root' disease) causes pressure on nerve endings resulting in pain (consider a neurogenic analgesia). Should the 'root' disease progress, the maxillary 'roots' may grow into nasal cavity / sinus, occlude the NLD, and or grow into the orbital foramen. I have seen one case where a 'root' had grown into the brain! (see Figure 8.9).

The mandibular 'roots' may grow through the ventral mandibular cortex *palpable as hard bumps on the ventral medial border of the mandible*. Without knowing the condition, length, and shape of the maxillary and mandibular 'roots' we cannot give information on prognosis or know if long term analgesia is required.

Without this information, degus with moderate to severe 'root' disease will be in chronic pain, will not chew properly and will require much more frequent dentals. With appropriate analgesia, degus will cope even with radiographically spectacular 'root' disease (Figure 8.10).

Once anaesthetised (see General anaesthesia section) I will take a right lateral, dorsoventral, and a cranio-caudal skull radiographs as routine. *Good positioning is required for the radiographs to be of diagnostic use*. True lateral radiographs then the mandible bodies will be overlying as will the tympanic bullae.

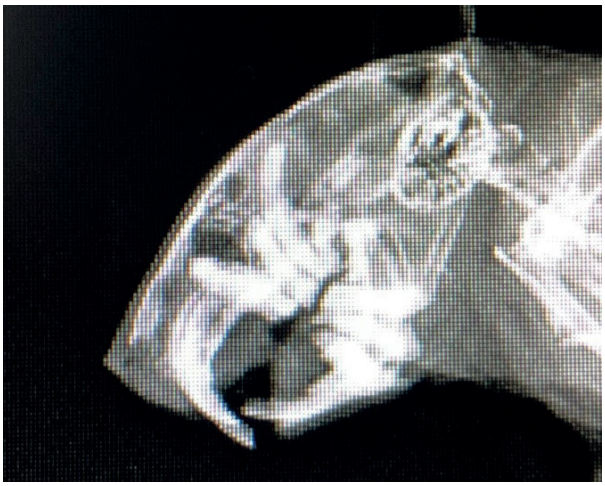


Figure 8.9 Lateral radiograph of a degu skull showing severe dental disease. Note the maxillary roots growing into the nasal sinus and the root growing into the brain. The mandibular roots are through the mandibular cortex.



Figure 8.10 Lateral radiograph of a degu with severe root disease.

Lateral radiograph will provide the most information on the length of the apices of all arcades and narrowing of the nasal cavity. *Degus have a much higher number of nasal cartilages than other rodents, with narrow lumina (they use these to condense and absorb water from their expired air) which is why even minor nasal cavity obstruction can cause severe dyspnoea*

and open mouth breathing. The increased water loss associated with open mouth breathing and the difficulty of drinking when forced to breath in this manner can quickly lead to severe dehydration.

Carefully examine the apices of the maxillary incisors to check for elondontoma formation, and the apices of the maxillary cheek teeth for pseudo odontoma formation.

The cranio-caudal radiograph will allow visualisation of the temporomandibular joint to look for pathology, e.g. arthritis, which dental disease may be secondary to. This view will also provide information on the mandibular symphysis, the occlusal plane of the cheek teeth, the presence of abnormal spikes, tympanic bullae, and may provide information on nasal disorders.

The dorsoventral view provides information on the incisors and the tympanic bullae.

Severe root disease is easy to see (see Figure 8.10) but mild or subtle changes can be harder to appreciate if you do not spend all day looking at degu skull radiographs. Capello and Lennox's (2013) *Clinical radiology of exotic companion mammals* is a useful textbook.

Elondontoma and pseudo ondontoma

Elondontoma formation is seen on radiographs as a radiopaque mass associated with the apice of a maxillary incisor, and may grow large enough to completely block the nasal cavity resulting in severe dyspnoea, serous (or purulent if secondary bacterial infection) nasal discharge, reduced air flow down the affected side's nostril and potentially aerophagia (Figure 8.11a–c).

The exact cause of elondontoma formation is not yet known but is suspected to be due to dental disease and or repeated trauma (e.g. bar pinging).

Elondontoma treatment

Treatment of elondontoma can be difficult. All of the incisors should be surgically removed, which will involve lateral or dorsal rhinotomy on the side of the affected maxillary incisor apice to remove or at least debulk the elondontoma. The surgical wound should be left open to heal by secondary intention and repeated scab removal will be needed. Good analgesia and supportive care are required. Pseudo odontoma is similar to elondontoma but the nasal cavity is invaded by elongation of the maxillary cheek teeth apices.

Other intranasal tumours can occur in degus, which are not associated with apices of teeth, but will give similar signs of elondontoma and due to the high prevalence of dental disease in this species, may occur concurrently to dental disease. These will be covered in the Respiratory section.



Figure 8.11a Lateral radiograph of a degu with early elondontoma.

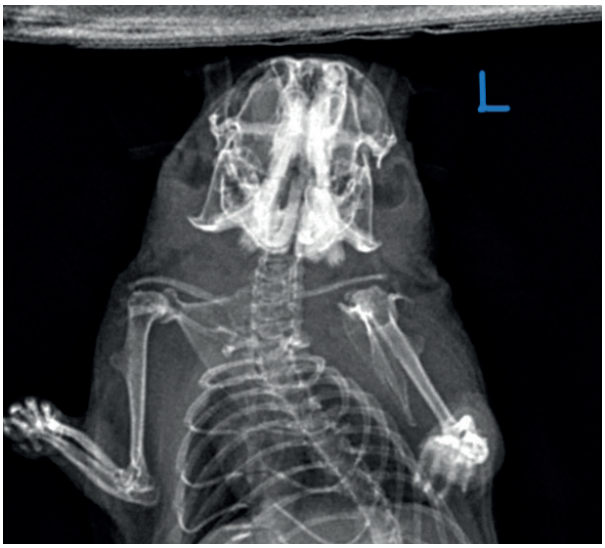


Figure 8.11b Rostrocaudal skull radiograph. Note the left nasal cavity has been completely blocked by an pseudo ondontoma.



Figure 8.11c Lateral skull radiograph. Note the nasal cavity has been completely blocked by an pseudo ondontoma caused by the premolar apice.

Oral examination

Once I have radiographed, I will perform a full oral examination.

In the normal degu the dental formula is: (2×) I 1/1 C 0/0 P1/1 M3/3.

The maxillary incisors are 0.5 cm long and the mandibular approximately 0.8 cm. There should be a horizontal occlusal surface. The enamel is a yellow brown colour from 6 months of age.

Cheek teeth: each mandibular tooth occludes with one maxillary tooth and works as a functional unit and forms a continuous grinding surface due to the lack of space between teeth, i.e. the clinical crowns are all the same height and the arcades are symmetrical. The healthy teeth are cream in colour and each tooth resembles a figure of 8.

The mandible is wider than the maxilla and at rest the cheek teeth are not in occlusion.

All arcades should be 2 mm above gingival margin and occlusal plane is almost horizontal. *The mouth is symmetrical.*

Degus with dental disease are likely to have asymmetrical dental arcades. The maxillary molars flare towards the cheek and damage the buccal mucosa (painful) while the lower arcades grow lingually into the tongue, grow vertically, or the mandibular premolar can bridge the tongue (Figure 8.8).

Degus *will suffer from pain associated with retrograde root growth*. This is readily palpated as hard bumps on the ventral border of the mandible. Compared to some other species (e.g. guinea pigs) with appropriate analgesia, degus can cope with truly spectacular 'root' disease. The period between dentals could be as frequent as every 12 weeks but owners should be warned that the first few dentals may be closer together as the degus' muscles adjust to chewing and adjust from being stretched.

> The aim of dental surgery is to restore the correct shape, length, and symmetry of all the dental arcades therefore allowing normal functioning of the teeth.

Incisor malocclusion

Incisor malocclusion *(is not as common in degus as in rabbits, and is usually secondary to cheek teeth elongation)* can be managed with repeated trimming of the teeth (this can be done conscious in many patients) using a diamond cutting disc (use of guard advised) or a dental drill bit with the water turned off. The normal incisor eruption rate is approximately 1 mm per day but this can increase if they are unopposed in malocclusion.

Some individuals will find frequent trimming stressful, therefore if the incisors are non-functional, or are preventing normal chewing, extraction is recommended.

> Incisors should never be clipped with molar cutters or nail cutters. This practice is outdated and barbaric. Due to the conformation of the enamel, clipping causes the enamel to shatter leading to vertical fractures, pulpitis and potentially apical abscessation. There is no excuse for clipping incisors.

Tooth resorption

Tooth resorption can occur and can lead to the loss of clinical crowns, food and fur can become impacted within the widened interproximal space which will often then lead to periodontal disease. Periodontal disease can be present without tooth resorption.

Periodontal disease

Degus can be prone to periodontal disease. Perform a full oral assessment for periodontal disease.

Personally, I make a point of examining each tooth for mobility and probing for gingival pockets. *Left untreated, periodontal disease will progress to bone and tooth resorption, be a chronic source of oral pain, leading to decreased food intake, further weight loss and increase the risk of hepatic lipidosis or ketosis developing*.

There may be pockets filled with food, hair, and debris around the teeth. *It is important that all the material is removed*, I use small atraumatic forceps to grasp the hair/debris and pull it out. The pockets are then cleaned with dilute chlorhexidine or flushed with 2% hydrogen peroxide. Some authors prefer hydrogen peroxide because the foam is easier to contain and less risk of aspiration. Unlike chinchillas, degus uncommonly have severe infections accompanying periodontal disease.

Dental caries

Caries are common in degus and are usually brown or black in colour caused by areas of bacterial decay. Sugary treats increase the risk.

The diseased area should be removed using a ball tipped diamond bur or a rose bud rodent drill bit drilling until a healthy crown is evident. Wiping the teeth with cotton buds wet with diluted chlorhexidine can be used to clean tooth surfaces and remove plaque. Owners should be advised to stop feeding sugary treats and to increase the hay and dried grass component of the diet.

Dental surgery

Extraction of the incisors

Extraction of the incisors is rarely indicated in degus. Overgrowth can be managed by trimming with a diamond disc or dental bur, and treating the underlying molar coronal overgrowth by performing a dental.

Incisor extraction should only be reserved for cases that have severe malocclusion, periapical infection, or elondontoma formation. Owners worry that their pet cannot eat without their incisors. Counselling owners to consider incisors as scissors, that degus hold their food in their front paws and longer term cutting of grass and fresh foods into bite sized portions that the degu can pick up and push into its mouth is all that is required. *If incisor extraction is performed, all the incisors should be extracted.* Incisor extraction is performed using a pre-bent 24g needle instead of a Crossley Luxator otherwise using the same technique as in guinea pigs (Chapter 3).

Cheek teeth

RISKS ASSOCIATED

Branches of the facial artery are located in the buccal mucosa, lateral to the last maxillary molars. Trauma to the overlying soft tissue by handheld instruments can lead to death if bleeding is not quickly stopped. Owners should be counselled that while these complications are rare, a fatal bleed can quickly occur. Collaclot is a collagen sponge which will quickly lead to haemostasis. It is inexpensive, has a long shelf and in these situations can be lifesaving.

EQUIPMENT NEEDED

Straight dental handpiece with rodent drill bit, rose bud or bar tipped rodent drill bit, diagnostic pen torch with speculum (or head torch and an oral speculum), two lengths of bandage, cheek pouch dilator (or Thudicum nasal speculum), curved tweezers, small dental probe, moistened cotton buds.

Performing dental surgery

Owing to the small amount of available space in the oral cavity, I do not intubate for dental procedures.

I prefer to position degus in dorsal recumbency for this procedure with their thorax raised on a towel. Positioning like this allows me better access and visualisation of all four arcades.

There are many different types of cheek pouch dilators on the market and in my experience, the springs of many of them are too strong, resulting in the instrument 'pinging' out of the mouth or overstretching the cheeks which will be painful. Care should be taken not to further damage any friable oral mucosa when placing the dilators. I will sometimes use Thudicum nasal speculum, sometimes I will not use any cheek pouch dilators.

Instead of using the oral gag over the incisors, I use two lengths of bandage material that is inserted over the incisors and used to slowly open the mouth, stopping as soon as there is any tension felt (Figure 8.12). *Over opening the mouth will cause damage to the temporomandibular joint.*

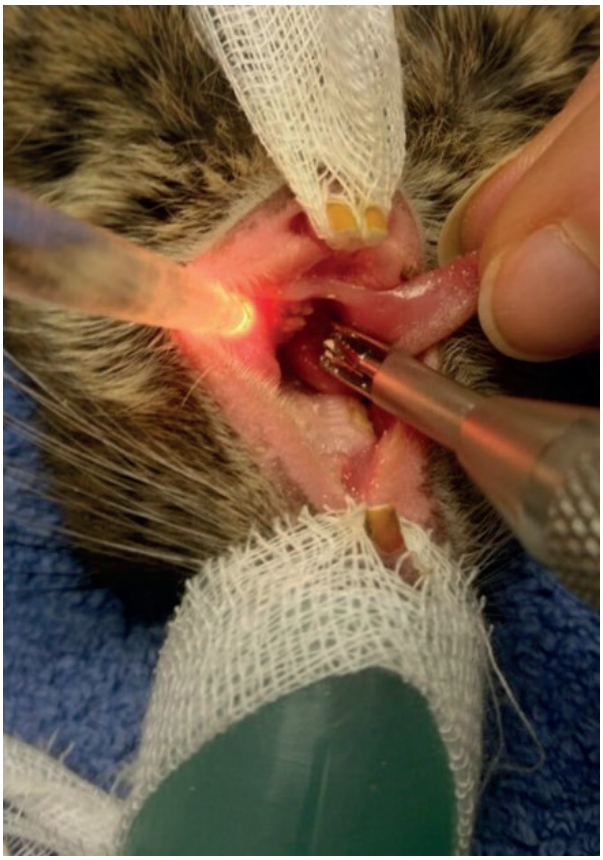


Figure 8.12 Bandages being used as a mouth gag while the dental is being performed. I tie the bandage placed on the maxillary incisors onto the anaesthetic circuit which helps keep the degus nose in. My diagnostic pen torch is used as a spatula and light source.

I use a diagnostic pen torch with Perspex speculum (Eickemeyer) as both my light source and my speculum/soft tissue guard. The tapered tip of the speculum provides good illumination even to the caudal teeth.

Each tooth should be checked for mobility and probed for pockets or periodontal disease. Run the torch speculum along the buccal aspect of the maxillary arcades to check for hidden spikes

Using the dental drill with a rodent drill bit and soft tissue guard, held in a pen grip (allows much greater control over the drill), to remove any spurs and to reduce the coronal length (if required) in all arcades. Any crowns that are very loose should be gently pulled out. Remove tooth dust with dilute chlorhexidine moistened cotton buds, ensure both upper arcades are symmetrical and that both lower arcades are symmetrical.

Hand rasping kits are still available. Only the smoother, diamond rasp should ever be used. Hand rasping takes much longer to perform and there is a higher risk of perforating the arteries. *Caution should be exercised as any kind of rasping can potentially lead to damage of the periodontal ligament, loosen the molars, and exacerbate dental disease.*

Post-dental care

Assist feed on recovery from anaesthesia. Assisted feeding may be required for a few days (often longer than would be needed in rabbits), allowing the jaw muscles to readapt to closing the mouth and to allow the occlusal surfaces of the cheek teeth to wear back into function. Monitor faecal output and *urine can be tested to check pH and for ketones. Dipstick analysis of urine is a cost-effective way of ensuring adequate nutrition is being given*.

Continue analgesia for at least a week after the procedure although cases with 'root' disease will require long term analgesia. Dietary assessment is required, and owners should be advised we aim to eventually reduce the time between dental surgeries to approximately three to four months. More frequent dentals may indicate that our analgesia is inadequate, especially neurogenic pain that can be caused by apical elongation pressing on nerves. In my experience, with correct analgesia, degus cope remarkably well with spectacular 'root' disease, *provided there is no narrowing of the nasal passage*.

Dental abscesses

Periapical infections and dental related abscesses are rare in degus. If present, treatment options are the same as for chinchillas.

Heat stroke

Degus are adapted to a low humidity (30–40%) environment and are better suited to lower temperatures as they utilise their deep burrow system to escape high ambient temperatures. Environments with ambient temperatures of over 28°C (82.4°F), especially if they also concurrently have high relative humidity of >50% can rapidly result in heat stroke. Obesity may predispose to heat stroke.

Clinical signs

The degu may present recumbent or ataxic, with rapid breathing, bright red mucous membranes, and thick stringy saliva. There may be blood froth at the nose and mouth.

Treatment

Treatment involves cooling of the animal with the use of fans and tap water, and IV/IO fluids should be given. Unfortunately, most will deteriorate after an initial improvement so owners should be advised the prognosis is guarded to poor.

Respiratory disorders

Degus are obligate nasal breathers and the RR is 45 – 80 breaths per minute (Ardiaca García *et al*., 2021), although some texts state much higher respiratory rates.

Primary infectious causes of respiratory disease (RD) are rare in degus. Non-infectious causes include dental disease, hyperthermia, traumatic head injury, metabolic acidosis, renal failure, poor ventilation, and dusty substrate. *The most common cause of upper respiratory signs in degus is dental disease resulting in invasion of the nasal sinus by elongation of maxillary cheek teeth apices, pseudo odontoma, or elondontoma*. Secondary bacterial rhinitis may result.

Degus have a much higher number of nasal cartilages than other rodents, with narrow lumina (they use these to condense and absorb water from their expired air), which is why *even minor nasal cavity obstruction can cause severe dyspnoea and open mouth breathing*. The increased water loss associated with open mouth breathing and the difficulty of drinking when forced to breath in this manner can quickly lead to severe dehydration.

Any degu presenting with respiratory signs should be admitted for skull and thoracic radiography. Blood biochemistry results are often nonspecific and associated with anorexia resulting in abnormal liver metabolism.

Intranasal tumours

Intranasal tumours can occur in degus, which are not associated with apices of teeth, but will give similar signs of elondontoma (sneezing, dyspnoea, nasal discharge, reduced air flow, intermittent open mouth breathing, anorexia, and dehydration) and due to the high prevalence of dental disease in this species, may occur concurrently to dental disease.

Nakata *et al*. (2022) looked at a case series of 20 intranasal tumours, 15 were visible on radiographs, the other 5 required CT scanning to be visualised. Eleven of the 20 had concurrent dental disease and all 20 developed severe dyspnoea with gastric tympany due to aerophagia. Most of the degus were over 2 years old with a median age of 3.6 years. All 20 were induced with iso and an excisional biopsy via lateral or dorsal rhinotomy on the side affected. The tumours were firm, and most were non-adherent to the surrounding tissue and as such were easily removed. The surgical wound should be left open to heal by secondary intention and repeated scab removal was needed. Six of the 20 died perioperatively, of the remaining degus all had improved air flow. On histopathology 16 were fibromas and 4 were osteomas, so it is fair to say that *intranasal tumours in degus are mostly benign mesenchymal tumours with various degrees of bone formation and this is unique to degus*. Although these tumours are benign, they should be considered locally invasive and prone to recurrence (7/20 recurrence confirmed 2–6 months post-operatively). Survival post-operatively varied from 60 days to 635 days.

Treatment depends on underlying cause and severity. In a retrospective study, 10/16 cases of rhinitis were associated with dental disease, and 4/16 were associated with otitis media.

Cardiology

The heart is found between intercostal spaces 3–5. There has been no vertebral heart score published for degus and there is only one published case report of congestive heart failure (due to a ventricular septal defect) in a degu which also had severe dental disease. In this case, a grade 4/6 murmur was heart and the radiographs revealed cardiomegaly, with a dorsally raised trachea and a diffuse interstitial pulmonary pattern consistent with pulmonary oedema. Other than furosemide, further cardiac medication was not given in this case and the degu died on recovery from a general anaesthesia given for a dental procedure.

Cardiac disease is rare in degus, but treatment options should be considered similar to other rodents until further information is available.

Ophthalmology in degus

The approach to ophthalmology in degus is similar to our approach in dogs and cats, they are just eyes. The most common ophthalmic conditions seen in degus are cataracts, corneal disease, and uveitis. Cataracts account for 80% of ophthalmic conditions in degus.

Degus have a vertical slit shaped pupil which is semi-constricted in ambient lighting, which becomes circular when pharmacologically dilated. All degus have a pupillary light response and have excellent vision.

Cataracts

Cataracts occur due to age-related changes or secondary to diabetes mellitus. Age-related cataracts are common in wild degus, but diabetes mellitus related cataracts are more common in the pet degu (Montiani-Ferreira *et al.*, 2022). Diabetic cataracts can develop in as short as 4 weeks after developing diabetes.

Any degu presenting with cataracts should have blood glucose and serum fructosamine checked. They are usually bilateral but may present initially unilaterally and at different stages.

Due to many cases of diabetes being type 2, *if the owner has changed the diet, the degu may present with cataracts and have normal blood glucose*. If the degu is obese and/or has a diet too high in sugar, a full dietary review should be performed as many cases of diabetes can be managed nutritionally. HEV'N manufactures a diabetic herb mix which may help with reducing blood glucose (see Diabetes section).

Corneal ulcers

Corneal ulceration is uncommon in pet degus but should be investigated in a similar manner to more familiar species using ophthalmic exam and fluorescein should be applied with any lesions documented (Figure 8.13).

Treatment

Analgesia such as meloxicam 0.5–1.5 mg/kg q12hrs, should be provided until the cornea heals.



Figure 8.13 Corneal ulceration.

Topical lubrication is also advisable q6–12hrs depending on the owner's ability to handle the degu. As always *it is important to balance the potential stress caused by medicating with the benefit provided*.

Topical antibiotics using a broad-spectrum eye drop such as a triple antibiotic drop containing polymyxin b/neomycin/gramicidin, or as guided by culture and sensitivity. A Gram stain can be performed and if Gram negative rods or cocci are identified, ciprofloxacin or ofloxacin may be preferred and can be adjusted if necessary once culture results are obtained.

Exophthalmos

The *most common cause of exophthalmos in degus is retrobulbar abscessation* secondary to dental disease. Other less common causes include orbital neoplasia, sinus infection, foreign body, trauma, and brain tumours extending into orbital.

The globe is normal in size, is often visual, and progression of the exophthalmos may be slow. Retropulsion of the globe is not possible.

Diagnosis

Diagnosis is via clinical signs. Radiographs are indicated to check for bone involvement and fully assess for dental disease. Careful oral examination under general anaesthetic should be performed.

Treatment

Treatment will vary slightly depending on aetiology. Consideration should be given to euthanasia in cases with severe dental disease and/or osteomyelitis. See Dental disease section for assessment and treatment options.

Enucleation should be performed transconjunctivally if there is no evidence of an abscess or neoplasia. Degus have an orbital venous sinus and a large Harderian gland.

Abscessation or neoplasia will require exenteration to remove all the structures within the bony orbit.

Exenteration

It is important to *advise owners that once healed, the skin is likely to sink into the socket*. If owners have had previous experience of enucleation performed transconjunctivally, they will not be expecting the less cosmetic appearance of a sunken, empty eye socket.

Perform a lateral canthotomy and remove eyelid margins. Excise all the tissue within the orbit, working close to the bone to remove the whole sinus together with the globe. Ligate or use a clip to control haemorrhage. Collaclot (CambCol) can be used to control any haemorrhage if needed. The skin is then sutured closed.

Orthopaedics: fractures

Fracture repair principles used for dogs and cats are easily adapted to small mammals (it is just mammalian bone), and degus have similar skeletal anatomy which allows texts aimed at repair of selected fractures in dogs and cats to be consulted for surgical approach.

Most fractures in degus are due to trauma, either being dropped by the owner, trapping a leg in the cage or from a bite. Owing to their legs being so close to their body (which makes lameness harder to detect), their amazing ability to hide pain, and today's busy lifestyle of many owners, it is not uncommon for a considerable delay to be present between the trauma occurring and presentation.

Pathological fractures can occur due to osteopenia (lack of exercise, lack of UVB, diet too high in phosphorus/too low in calcium), osteomyelitis, and neoplasia.

Various options exist for fractures in degus and many owners are keen to explore options. *Pain causes anorexia and gut stasis so prompt analgesia and supportive care is required*.

The main differences in degu orthopaedics which we need to appreciate are:

- Degus rest and move with their limbs very close to their body. This makes external coaptation more challenging to effectively apply – but not impossible.
- Dogs and cats find it easy to lift a painful limb while ambulating normally on the remaining three limbs – degus find this very difficult. Degus also rely on their front legs for eating and thorough grooming, and hind legs for balance when grooming perianal region or consuming caecotrophs.

Degus spend a lot of time resting predominantly on their hind legs. The proximal ends of the tibia and fibula are fused.

Degu bones are thin and fragile which can make surgical repair challenging.

Supportive care (check for signs of shock and treat appropriately) should be given and owners should be advised that depending on the presentation, stabilisation may take several days, with the exception of spinal fractures, shock is more life threatening than a fracture is.

General anaesthesia is required to ensure diagnostic quality radiographs. Two orthogonal views should be taken, and I advise full body radiographs to assess for other skeletal injuries. Careful handling under general anaesthesia should be used if suspicious of a vertebral fracture as the epaxial muscles will not be supporting the vertebrae. If an open fracture is present, then a swab should be taken of the fracture to allow bacterial culture and sensitivity to be performed.

Regardless of fracture repair technique used, it is vitally important to adequately analgise (they will find this just as painful as you would), minimise stress, and restrict activity levels until healed.

Conservative management

Depending on fracture type, *strict cage rest in a single level enclosure and analgesia* may actually achieve good clinical resolution of fractures.

Surgical management

See intramedullary pinning, ESF, and amputation in Chapter 3. Hypodermic or spinal needles may be used as pins. Complications associated with surgical fixation include pin loosening and infection, non-union, necrosis of distal limb and auto mutilation. Degus usually adapt well to hind limb amputation.

Dermatology

Alopecia

The most common cause of alopecia is barbering, which can be a primary behavioural disorder associated with anxiety or stereotypy (common in degus less than 2 years old), or secondary to dental disease pain (seen mainly in older degus). Known triggers for barbering as a primary behavioural disorder are insufficient cage size, lack of exercise, lack of burrowing substrate, being kept as a solitary animal, inappropriate light dark cycle, or a high calorie low fibre diet.

Barbering may be self-directed or the most dominant degu may overgroom cage mates. Self-barbering usually is seen on the: distal forelimbs, feet, and the medial aspect of the thighs. There is usually no erythema unless a secondary bacterial infection has developed.

A full husbandry review should be performed, and owners need to understand that the alopecia will never resolve without

correction of the husbandry. A Pet Remedy plug-in may help reduce anxiety but this is in addition to, not instead of husbandry improvement.

Dermatophytosis

Dermatophytosis is rarely seen in pet degus. If diagnosed, consideration should be given to the potential of immunosuppression and co-morbidities or stressors should be investigated. Owners should be advised that dermatophytosis is zoonotic.

Trichophyton mentagrophytes (which does not fluoresce under a Wood's Lamp) and *Arthroderma benhamiae* have been reported in degus.

Clinical signs can include patchy, scaling alopecia, hyperkeratosis, affecting primarily the face (although the lesions can be anywhere). There are variable levels of erythema.

Diagnosis is via skin scrapings and *hair plucks from the edge of lesions* for fungal culture and/or microscopic examination.

Treatment is systemic therapy with either itraconazole 5–10 mg/kg q24hrs or terbinafine 10–20 mg/kg q24hrs, and continued until two negative fungal cultures are obtained 1 month apart. Not all owners will be able or willing to pay for repeated fungal cultures. Treatment should be for a minimum of three weeks and if owners cannot or will not repeat the cultures this should be recorded in the notes and owners should be made aware that without the tests, we cannot guarantee full resolution just because the obvious clinical signs have resolved. Terbinafine is preferred as it has been shown to be more effective in guinea pigs.

Disinfection of the environment with a suitable agent such as sodium hypochlorite or F10 is an important part of management. Wooden enclosures or shelves may be difficult to eradicate the dermatophytes from.

Ectoparasites

Ectoparasites are rare in degus. One case report details infestation with *Ornithonyssus bacoti* (tropical rat mite) which is zoonotic. The mite is seen both in the substrate and on the animal, and is visible to the naked eye. One other case report details *Demodex* spp., which was found on a deep skin scrape of a pruritic degu although the cage mates had no clinical signs. Selamectin 30 mg/kg q14days for two treatments is usually effective.

Fleas

In houses with dogs and cats, they may contract fleas. Heavy infestations of fleas may result in a dull coat, intense pruritus, and patchy alopecia. Diagnosis is achieved via careful clinical examinations for fleas and flea dirt. Treatment is topical selamectin 20–30 mg/kg, environmental control, and treatment of the dog or cat.

Neoplasia

Neoplastic disorders are considered rare in degus, which is surprising given their long lifespan. As of 2022, the retrospective studies that have been published indicated a prevalence of neoplasia as 6.2% in degus. Some of the most common neoplasms reported in descending order are cutaneous fibrosarcoma (see below), leiomyosarcomas of the reproductive tracts, chordoma, and biliary cystadenoma. Other neoplasms have been described as single case studies including: thymoma of cervical portion (should be included in differential list for ventral neck swellings), osteosarcoma, and lipoma.

Elondontomas are pseudo neoplastic masses and are detailed under dental disease. Other intranasal tumours can occur in degus, which are not associated with apices of teeth, but will give similar signs of elondontoma and due to the high prevalence of dental disease in this species, may occur concurrently to dental disease. These will be covered in the respiratory section.

Chordoma of the tail

Present in older animals as a slow growing, firm, round, non-painful, well defined, mass of the tail. Usually seen near the tip of the tail but has been recorded at the base also.

Radiographs will often reveal lysis of the caudal vertebrae and tail amputation to remove the mass is advisable. On histopathology may be classified as highly malignant but there has been no evidence of metastasis and further studies are needed to predict the clinical behaviour of this mass in degus. Given the current information available, resection could be curative.

Neoplastic skin disease

Otrocka-Domagała *et al*. (2022) found during their retrospective study (2014–2021) that although neoplasms were rare in degus, *malignant tumours represented the predominant group of neoplasms of cutaneous tumours* in degus (as well as in rats, African pygmy hedgehogs, and chinchillas.) The mean age was 4.2 years old (range: 1.5–6 years) and of the eight cases they had, seven were soft tissue sarcomas, and one was epitheliotropic lymphoma. *Any skin mass in degus should be investigated* either by performing a fine needle aspirate (FNA) (which would allow surgical planning) or by excision and histopathology.

Cutaneous fibrosarcoma were more common in female degus, the lesions were mainly found on the hind limbs in older animals and no metastasis were found.

Ear diseases

Degus are less prone to ear diseases than other rodents. Spontaneous otitis externa is undocumented in degus.

Otitis media/internal has been documented often in conjunction with secondary bacterial rhinitis caused by dental disease. Clinical signs are as seen with more familiar

species, otitis media can result in facial nerve paralysis, enophthalmos and debris protruding from the eustachian tube. Otitis interna may cause strabismus and vestibular signs (head tilt, ataxia, torticollis).

Diagnosis is based on clinical signs, skull radiography may show increased radiolucency within the tympanic bulla as well as alterations to the bullae structure. CT imaging would provide a higher level of detail and sensitivity but may be cost prohibitive for many owners.

Treatment of otitis media/interna includes analgesia, antibiosis, and personally I have had good success adding in propentofylline (5 mg/kg q12hrs) in rats.

Renal disease

Renal disease is uncommon in degus but nephrosis, pyelonephritis, glomerulonephritis, polycystic kidney disease, and one neoplasm have all been reported. Renal disease may occur secondary to diabetes mellitus.

Clinical signs may be nonspecific such as anorexia, weight loss, hunched posture or similar to more familiar species: polydipsia and polyuria.

Diagnosis is via biochemistry demonstrating azotaemia, there may or may not be a raised phosphorus, urinalysis, and ultrasound. There are no published urinalysis reference ranges for degus, but where urinalysis results have been published in associated with renal disease in degus the urine showed: haematuria, pyuria, occasional squamous and transitional epithelial cells, and a urine specific gravity (USG) of 1.016 (Cadillac *et al.*, 2003; Lester *et al.*, 2005).

Treatment is mainly supportive with fluid therapy and assisted feeding. If bacteriuria is demonstrated, antibiotic choice should be guided by culture and sensitivity, trimethoprim sulfamethoxazole should be avoided in degus with renal disease. Cases of diabetes mellitus should be treated.

Urolithiasis has not been reported in degus.

Diabetes mellitus

Hystricomorph rodents have a different insulin structure with lower physiologic activity (only 1–10% active) compared to other mammals. They compensate to control blood glucose levels by having a higher insulin concentration, a slower insulin degradation, and a higher number of insulin receptors compared to other mammals. *In other words, degus cannot regulate blood glucose concentrations as tightly as other mammals*.

Therefore, if they are fed easily digestible carbohydrates, they initially develop hyperinsulinemia, type 2 diabetes, cataracts, then failure of the beta cells of the pancreas leads to hypoinsulinemia and type 1 diabetes can develop.

Type 2 is reportedly more common, especially in obese animals.

Clinical signs are very similar to more familiar species: cataracts, polydipsia, polyuria, highly elevated blood glucose, glycosuria, ketonuria, low urinary pH, alongside more nonspecific clinical signs such as anorexia, weight loss, diarrhoea, dehydration, and lethargy.

> Hepatic lipidosis and ketonuria occur readily in degus if they have a reduced appetite for any reason, stress-induced hyperglycaemia being very common, and that if the hyperglycaemia crosses the renal threshold, glycosuria will occur in stressed degus. Therefore, diabetes mellitus should never be diagnosed on a single day's test results.

Diagnosis

A full clinical examination for co-morbidities should be performed.

Full biochemistry and haematology are recommended (see Venipuncture) as well as serum fructosamine measurement. Blood glucose levels should be checked on several occasions and persistent hyperglycaemia of over 11 mmol/l (200 mg/dl), hypercholesterolemia, and glycosuria is considered diagnostic.

Urinalysis should also be performed. If blood is present, then sediment examination for evidence of a secondary bacterial urinary tract infection is advisable.

Treatment

Perform a full dietary review. Obese degus are more likely to have type 2 diabetes, which will most likely respond to dietary management and weight loss. Stop all sugary treats, e.g. raisins or dried fruit, stop high fat treats, e.g. sunflower seeds, and start a high fibre diet.

The urine can be monitored at home with twice daily dip sticks to monitor glycosuria and ketone production. Should the glycosuria or ketonuria fail to resolve, glipizide starting at 0.6 mg/kg q12hrs and titrate upwards can be trialled, although there is scant data on the recommended dose.

Alternatively, insulin can be started, and owners should be advised on clinical signs associated with hypoglycaemia. Starting dose is recommended as 0.1–1 IU/kg q8–24hrs with incremental increases of 0.1 IU/kg. Owners may find volumes easier if it is worked out in millilitres rather than international units ease of pulling up and injecting. Owners should monitor at home using urinary laboratory sticks. Glucose curves can be performed in the clinic; however, the stress of hospitalisation may make them difficult to interpret due to stress-induced hyperglycaemia.

Reproductive disorders in degus

Female degu

Contrary to several authors, the degu does indeed have a regular cyclical oestrus occurring every 17–21 days and

they are spontaneous ovulators. Young degus have shorter cycles of 17.5 days compared to 21.2 days to older degus. Degus show increased levels of activity (including wheel running) when in oestrus (Mahoney *et al.*, 2011).

They are sexually mature from 4 months of age (but breeding is not advised until adult body weight is achieved, which is at 6 months old) and gestation is 87–93 days.

The vagina is situated between the urethral papillae and the anus and is normally obscured by a membrane. Absence of the vaginal membrane occurs during oestrus, parturition, or uterine disease.

During oestrus the vaginal membrane is absent, the perineum swells and becomes redder, and a white vaginal discharge develops. These signs vary greatly in severity between individuals.

Parturition usually occurs early in the morning, typically before 8 am, and is usually complete within 4 h. The litter size is between one and four precocial kits and the female will consume the placentas. Owners may become concerned if they see blood on the nose or paws of the degu but should be reassured that this is normal to see immediately post-consumption of the placenta.

Dystocia

Dystocia is very uncommon in pet degus and when occurs is usually in primiparous females with a small number of large pups although it can also occur due to stress during parturition, uterine inertia, dam obesity, dietary changes, or overfeeding carbohydrates.

Clinical signs include restlessness, frequent vocalisation, constant attention to the genital region and a widened vaginal opening. There may be apathy, anorexia, tachypnoea, and a blood tinged vulvar discharge. Unproductive straining may be obvious. *If there is non-productive straining for over 20 minutes, consider intervention*.

Radiographs are indicated to check number, size, presentation, and for malformation of the pelvis. Ultrasound will confirm if the pups are alive.

TREATMENT

Provide warmth, reduce stress, give fluids (oral if swallowing), and give analgesia.

Treatment of uncomplicated dystocia, i.e. uterine inertia, is the same as more familiar species: oxytocin 0.1–1 IU/kg SC and calcium gluconate 25–50 mg/kg, diluted with saline and given SC.

Caesarean section is indicated if medical management fails or in cases of dead, oversized or malpresented pups. Isoflurane or sevoflurane induction is performed without pre-medication to prevent the pups being adversely affected. Caesarean section is performed in a similar manner as in more familiar species and neutering at the same time is recommended (due to the increased risk of hematometra or pyometra).

Hematometra and pyometra

Endometritis and pyometra are rare and all reported cases occurred after prolonged parturition.

Clinical signs include acute onset depression and anorexia, or mild lethargy, or behavioural changes, open vulva and a vaginal discharge is seen in most cases (but closed pyometras can occur). Vaginal discharge varies in appearance from mucoid, to mucopurulent, to haemorrhagic, and can cause fur staining.

Uterine enlargement may be palpable during clinical exam and palpation of the uterus may elicit more vaginal discharge.

Diagnosis is achieved via clinical signs and ultrasound examination of the uterus. Ideally perform urine dipstick analysis to check for ketosis. Hepatic lipidosis, ketosis, and sepsis may be present and complicate the presentation.

Treatment of choice includes stabilisation, systemic antibiotics, analgesia, and ovariohysterectomy (OVH). If the degu is not stable enough for surgery or the owner does not want surgical intervention then medical management (as per guinea pigs) can be attempted with three SC injection of aglepristone (Alizin 30 mg/ml, Virbac Limited) 10 mg/kg given on days 1, 2, and 8, and stabilisation, systemic antibiotics, and analgesia.

Ovarian disease

Cystic ovarian disease and ovarian neoplasia is rare in degus.

Vaginal or uterine neoplasia

Uterine neoplasia is increasingly reported with leiomyosarcoma, leiomyoma, fibroma, and haemangioma all being cited. Uterine masses are often palpable and there may be a haemorrhagic vaginal discharge, anorexia, tenesmus, stranguria, ascites, and weight loss depending on location, size, and duration. OVH is the treatment of choice and prognosis is often favourable.

Mammary glands

There are four pairs of mammary glands: three pairs are lateral thoracic and one pair are inguinal. *Mammary neoplasia has not been reported in degus*.

Pregnancy toxaemia

Pregnancy toxaemia is a condition seen at the end of pregnancy or a few days post-parturition. Obesity, stress, and a large number of pups are all predisposing factors.

Sudden changes to diet and water can result in anorexia and dehydration. Anorexia or reduced food intake (e.g. owing to large uterus acting as space occupying mass) will lead to a negative energy balance and the development of ketosis.

Incidence of pregnancy toxaemia is higher during hot weather in guinea pigs so this should also be considered as a risk factor in degus and care should be taken to keep ambient temperature of between 20°C (68°F) and 22°C (71.6°F).

Clinical signs include anorexia, depression, hunched posture, salivation, dyspnoea, incoordination which may progress to seizures and death. Ketones may be smelt on the breath and urinalysis will show aciduria (should be alkaline in herbivores) and ketones. Biochemistry will show hypoglycaemia, hyperlipidaemia, and hyperkalaemia. Liver enzymes may also be elevated due to hepatic lipidosis.

Prognosis is poor even with aggressive treatment and death 24–48 h after developing clinical signs is common. IV fluids with added glucose should be given. Oral glucose, calcium gluconate and assisted feeding (q2–3hrs) should be started as soon as the degu is able to swallow. A short acting steroid (e.g. Dexadreson 2 mg/ml, MSD Animal Health UK Limited) should be administered IM at a dose of 0.2 mg/kg. Owners should be advised that steroid administration late in gestation can induce parturition or abortion. Owing to steroid administration, a non-steroidal anti-inflammatory, i.e. meloxicam, should not be given.

Prevention is preferable, ensuring that stress is minimised, that temperatures do not exceed 22°C (71.6°F), adequate space is given for exercise and that familiar food is readily available at all times.

Ovariohysterectomy

OVH is not commonly carried out in pet degus routinely, and is generally only indicated if there is pathology of the female reproductive tract. The procedure is similar to guinea pigs as degus also have short ovarian ligaments. Care should be taken upon entry to the abdomen because of the risk of damage to the large gastrointestinal tract.

Male degu

Male degus are sexually mature from 4 months of age. The testicles may be intraabdominal. *Penile disorders are very common therefore the prepuce and the glans penis should be carefully examined in every male degu*. Fur rings and paraphimosis are common.

Fur rings

Fur rings consist of a ring of constricting fur, wrapped around the penis, under the prepuce. This can lead to paraphimosis, stranguria, excessive grooming, or self-trauma. Carefully extrude penis to diagnose (Figure 8.14).

Treatment requires removal of the fur ring either by lubrication and cleaning, or by cutting the ring. In many cases this can be performed conscious but in fractious animals a sedation or general anaesthetic is more appropriate.



Figure 8.14 A fur ring had led to self-mutilation.

Paraphimosis

Fur rings and debris accumulation may cause (see above) or trauma. Over excitement or exhaustion may also result in paraphimosis which self corrects with time. Recurrent paraphimosis or paraphimosis which causes vascular compromise to the penis require surgical intervention. Recurrent paraphimosis has been successfully treated by surgically narrowing the preputial orifice as left untreated may progress onto penile ulceration, balanoposthitis, or penile necrosis. If the penis has been irreparably damaged, amputation of the penis should be considered as necrosis of the penis can cause urethral blockage.

Castration

Castration is not routinely performed in pet degus unless for population control. The technique is similar to the guinea pig, with bilateral incisions made at the cranial border of the palpable testicle, and a closed technique is used to prevent visceral herniation.

Gastrointestinal disease

What are normal faecal pellets like?

Normal faeces in degus are much smaller than rat faeces and often slightly curved. They are dark brown–black in colour, and dry (Figure 8.15).

Diarrhoea/soft faeces

Conditions leading to soft faeces or diarrhoea are much less common in degus compared to guinea pigs, however, *any systemic disease, stressful, or painful condition can result in secondary gastrointestinal problems*. These can have nonspecific clinical signs such as decreased food intake or anorexia, decreased faecal output, and lethargy.

In pet degus, sudden dietary changes, excessive amounts of fresh foods, or inappropriate oral antibiotic therapy are the most frequent cause of clinical signs.



Figure 8.15 Normal faeces from a degu.

Diarrhoea due to endoparasites has not been documented in degus.

A full history will establish if there have been any changes to the diet or oral antibiotics.

Gastrointestinal dysbiosis, hypomotility, and tympany

Hypomotility or stasis is commonly secondary to conditions such as: dietary change, low fibre diet, pain (anywhere), stress, obesity, or systemic disease.

Hypomotility of the gastrointestinal tract, regardless of cause, results in a dysbiosis, altered fermentation resulting in gas formation which causes tympany. Tympany is extremely painful – how painful do you find trapped gas?

Any change to the content, pH, or motility of the caecum can cause an overgrowth of Gram negative and some Gram positive bacteria, especially *Clostridium*, which can result enteritis and diarrhoea or in an often fatal enterotoxaemia and haemorrhagic typhlitis.

Treatment

Treatment involves identifying and treating the primary cause and aggressive supportive care. Fluid therapy, warmth, assisted feeding (reverses negative energy balance, helps reverse ketosis as well as establishing GI motility), famotidine, and analgesia. *If clostridial involvement is suspected, metronidazole 30 mg/kg q12hrs for 1 week should be started*.

Diarrhoea due to diet

Stop the new food or fresh foods. Give a hay-based diet and fluids if needed.

Antibiotic induced dysbiosis and enterotoxaemia

Narrow spectrum antibiotics (especially ones with predominantly activity against Gram positive bacteria) such as amoxicillin, ampicillin, cephalosporins, clindamycin, erythromycin, lincomycin, and penicillins should never be given orally to degus. Oral administration causes an overgrowth of Gram negative bacteria and Gram positive, especially *Clostridium*, which results in an often fatal enterotoxaemia and haemorrhagic typhlitis. If in doubt about the oral safety of an antibiotic always check the formulary prior to administration. That being said, even supposedly 'safe' antibiotics can cause issues with the gut microbiome in some individuals.

Treatment

Stop the oral antibiotics immediately. Assess hydration status and give IV fluids if necessary. Provide high-quality hay-based diet, and assisted feeding should be given in cases of anorexia. Collect faeces from the cage mates and add them to a syringe feed to help repopulate the gut. *If clostridial involvement is suspected, metronidazole 30 mg/kg q12hrs for 1 week should be started*.

Prognosis is likely to be poor.

Gas in stomach

Dental disease resulting in secondary rhinitis will frequently result in aerophagia causing distention of the stomach. This is often painful on palpation. Full investigation and treatment of the underlying dental disease is required and in some cases, euthanasia may be the best option.

Pancreatitis

Severe pancreatitis is reported in degus and all cases were associated with feeding a high fat diet. Symptomatic treatment of pancreatitis is the same across species, analgesia, assisted feedings, fluids, gastric protection, and dietary correction.

Euthanasia

End of life conversations can be a difficult area to broach with some clients. Occasionally owners will wait for us to broach the topic as they are 'worried' if they do so we will think they are not dedicated enough. Reassuring clients that sometimes we need to love our pets enough to let them go when the time is right can be invaluable.

Many owners will want to stay with their degu during this procedure and the process should be fully explained including possible events such as sudden jumping, the need to change veins or gasping. I administer a pre-med (e.g. medetomidine/butorphanol or midazolam/ketamine (maintains venous pressure) injected SC or IM), once the degu has fully relaxed, I will use the lateral saphenous vein by choice, to inject the pentobarbitone, to allow the owners to stroke the animals head.

If the vein is not accessible, the pentobarbitone may be injected into the liver or kidney as the animal is already under a deep sedation. If the owner chooses not

to stay with their pet, isoflurane may be used to induce anaesthesia and the pentobarbitone may be given by intracardiac injection.

I advise allowing the bonded cage mate to see its companion after euthanasia. I advise clients that I believe it allows the cage mate to accept that its companion will not return. Over the years, many owners have reported to me they felt the cage mate was looking for its companion constantly after losing them and certainly anecdotally this is reduced when they get the opportunity to 'say goodbye'. Whether this is purely anthropomorphic or not is up for debate, however it demonstrates to your owners that you care, and you listen to their concerns.

Many owners will opt for private cremation with return of their pets' ashes and all options that your practice offers for dogs and cats should also be made available to your degu clients. I offer a lock of fur, footprints (coloured ink pad from craft shop and a card). I send sympathy cards if I know the patient and owner well.

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9 African pygmy hedgehog

African pygmy hedgehogs (*Atelerix albiventris*), also known as the Central African hedgehog, white bellied hedgehog, or the four toed hedgehog (only has four toes on hind feet), first became popular pets in the 1980s. Unless they are purchased from an experienced breeder who invests a lot of time handling the young, these animals often are unsociable. Even well handled, friendly hedgehogs may react defensively when handled by individuals other than their owners. They are not rodents, they belong to the Erinaceidae family, and are omnivores. They are not a domesticated species, although most are now captive bred, and they have a strong instinct to hide illness and pain.

The dorsum of the hedgehog is called the mantle, *and is covered in thousands of sharp unbarbed spines, with the exception of a 0.5 cm × 2 cm strip along the middle of the crown of the head which is often bald*. The spines are strong, lightweight, and *do not easily epilate* due to a bulb at the base of the spine that holds the spine firmly in the follicle. The spines have a muscle attached in the dermis that allows the hedgehog to raise and flatten them. Spines last for around 18 months are replaced individually in a process known as quilling. The face, legs, and ventrum of the hedgehog are covered in pale fur. Owing to captive breeding, multiple colour varieties now exist with 92 varieties currently recognised.

As a defence mechanism hedgehogs roll into a ball, the panniculus muscle pulls the loose skin of the dorsum over the body and as the hedgehog tucks its head, feet, and tail under itself, the orbicularis muscle (which is in the junction between the spines and furred skin) tightens to close like a purse string suture protecting the vulnerable ventrum. The spines are raised and sit criss-crossed to prevent predators gaining access to the skin, to buffer falls, and help thermoregulation (Figure 9.1). Unfortunately for us, this makes *even a basic clinical exam challenging without anaesthesia*.

In 1991 the United States banned import of African pygmy hedgehogs from Africa and in some states a permit is required to keep one as a pet. This is because *African pygmy hedgehogs can carry Foot and Mouth disease*, both symptomatically and asymptomatically with the potential to spread the disease to cattle. A permit is required from the US Department of Agriculture for anyone who breeds, transports, sells, exhibits, or uses African pygmy hedgehogs for research or teaching purposes.

They are *solitary creatures* in the wild, only interactive with conspecifics for mating. Their gestation period is 34–37 days and they birth altricial young. Their average lifespan is 3–5 years but a maximum age of 10 years old has been recorded. Their adult body weight is 250–600 g and sources are contradictory as to whether males or females are bigger, in my experience the females tend to be bigger. They have an excellent sense of smell, excellent hearing (into ultrasonic range), but a poor sense of vision.

African pygmy hedgehogs are endemic to Central Africa stretching east to west and evolved to inhabit a diverse range of habitats including desert regions, grassland, open forests, scrub, and suburban yards. *They are nocturnal and spend daylight hours hidden* in burrows or in cavities, such as logs or crevices. They are *extremely active when awake*, often travelling long distances while foraging for food (home ranges have a 200–300 m radius from their burrow), they are good swimmers and good climbers. Their temperature preference



Figure 9.1 The spines are raised and sit criss-crossed to prevent predators gaining access to the skin, to buffer falls, and help thermoregulation.

is 24–30°C (75.2–86°F). *Contrary to several texts, African pygmy hedgehogs do hibernate in the wild during cold spells, but this is not recommended in captivity due to the accompanying immunosuppression and susceptibility to infection.*

Wild diet

African pygmy hedgehogs are omnivores, evolved to eat a wide variety of food such as invertebrates, small vertebrates (for example, frogs, snakes, lizards, young birds, and mice), eggs, carrion, and plants. They spend the majority of their awake time foraging for food.

Companionship

African pygmy hedgehogs are *strictly solitary* in the wild. In captivity they are likely to fight once reaching sexual maturity, although some breeders have kept groups together, it is advisable to keep them as single pets.

Enclosure

African pygmy hedgehogs are *extremely active and good at climbing therefore they need a large escape proof enclosure*. A minimum size of 0.6 m × 0.9 m (2 ft × 3 ft) is recommended but ideally should be much bigger than this to allow expression of normal behaviour, which is a legal requirement.

Hedgehogs must be given at least 1 h daily of supervised exercise outside of the cage. To maximise the benefit of the exercise time, *the ideal time to allow out of the cage is evening to mimic natural peak in activity*. Some hedgehogs really enjoy swimming and owners can offer a swimming tub in the playpen provided a ramp is included to allow a safe way for the hedgehog to leave the water.

The sides should be smooth to prevent climbing or have bars close (less than 2 cm) together to prevent limb entrapment. Hedgehogs are messy creatures who frequently walk through their faeces, spreading them about the enclosure so the floor should be made of plastic as wood can be difficult to keep clean.

Wild hedgehogs sleep in burrows, under logs, or in crevices. Cardboard boxes or wooden boxes can be used to mimic these sites, allowing a hidden, safe place to rest. Alternatively, a large, open terracotta plant pot placed on its side can be used as one of the resting hides as can hollow logs or tubes. The resting sites can be lined with shredded paper.

Recommended bedding includes dust extracted shavings and shredded newspaper, which should be at least 7.6–10.1 cm (3–4 inches) deep to allow digging. The substrate will require frequent changing due to their messy nature. *Food items such as meal worms can be hidden in substrate to encourage foraging behaviour.*

Many owners like to use blankets to line their enclosures but this should be discouraged due to the high risk of fibres becoming wrapped around toes and feet causing constriction injuries (Figures 9.2a & b).



Figure 9.2a Constriction injuries due to fibres being wrapped around the foot.



Figure 9.2b Removal of the fibres, in this case, saved the foot.

Solid wheels can be provided for running. *To prevent painful spinal conditions, it is important to provide a large enough wheel that they do not have to bend their back while running ideally at least 30 cm (12 inches) in diameter.*

They should be able to run with a straight back and without having to lift their head. Hedgehogs will often run several kilometres per night on a wheel!

Supplementary heating via an electric heat pad attached to a thermostat or an overhead ceramic non-light emitting bulb attached to a thermostat is recommended. Ambient temperature should be maintained above 18°C (64.4°F), with their preferred temperature being around 24–28°C (75.2–82.4°F). If the temperature dips below 7°C (44.6°F) the hedgehog will enter a state of torpor (similar to hibernation), which will result in immunosuppression and leave the pet susceptible to infections. If the temperature rises above 30°C (86°F) the hedgehog will also enter a state of torpor called aestivation. The humidity should be maintained at less than 40%.

A day–night cycle of 10:14h should be provided, with low intensity light advised during the day.

Hedgehogs have a relatively small cerebrum and their learning capabilities appear to be less than rodents or carnivores. They do recognise their owners and with patient training they may learn simple commands. For example, some will learn to use a litter tray and placing all the daily droppings into the litter may help encourage this behaviour. Wood pellet litter is best for this, sand or clay clumping litter should be avoided as it sticks to the animal and may become stuck in the prepuce.

Diet

African pygmy hedgehogs evolved to consume a wide variety of food items which vary seasonally. Unfortunately, the exact nutritional requirements of African pygmy hedgehogs are still unknown. *As hedgehogs are nocturnal, they should be fed at night*.

European hedgehogs possess chitinase in their gastric mucosa and their pancreas suggesting that they are able to digest the chitinous exoskeletons of insects. A study in African pygmy hedgehogs found that 64–68% of dietary fibre added as chitin was digested, compared to only 38% of dietary fibre was digested when added as cellulose. This is an important finding *as obesity is a huge issue in African pygmy hedgehogs, and we can utilise this knowledge by advising the addition of more plant material to the diet to dilute the calories as cellulose material is poorly digestible*.

As they are omnivores, studies have suggested their dietary requirement is more similar to canids than felids. They require a moderately high protein (30–50% dry matter basis), with a moderate to low fat content (10–20%).

The mainstay of pet hedgehogs' diet is either low calorie dry dog or cat food, tinned dog or cat food, commercial hedgehog food, or dry or semi moist insectivore diet. These should be supplemented with chopped vegetables and fruit. Kitten food or ferret food is too high in fat and should be avoided. *Dry food is better for dental health as moist or wet foods are linked to dental disease*.

Live, gut loaded insects can be fed to encourage hunting behaviour and environmental enrichment. Insects should be dusted with a calcium supplement prior to feeding to the hedgehog. Wax worms should be avoided completely due to their high fat content and other larval forms of insects (e.g. mealworms, morios, calci worms) should be sparingly fed again due to their higher fat content compared to beetles, locusts, or crickets.

Advise owners against digging up and feeding earthworms or slugs and snails to their pet due to the high risk of wild invertebrates being intermediate hosts for endoparasites (see Respiratory section). Invertebrates should not be the only form of feeding as hedgehogs are omnivores not insectivores. Feeding an insect-only diet has led to cases of metabolic bone disease due to inappropriate calcium phosphorus ratios.

Treats can include cooked meat (dusted with calcium supplement), mealworms, boiled or scrambled eggs, pinkie mice, or vegetable or meat jarred baby food.

Dairy should not be given as this species is *lactose intolerant* and severe diarrhoea may result. Hard food items such as nuts, grains, or chunks of carrot (grated carrot is fine) should be avoided as they tend to become lodged between the upper dental arcades.

Obesity is a huge problem in captive hedgehogs. Excessive food intake, inappropriate food intake, lack of exercise, and insufficient foraging opportunities (food mainly from a bowl), are all contributing factors. The current recommendation is to feed 3.5–7.5% of the body weight (translates to roughly 2–4 teaspoons of food) each night and if food is left by the morning, the hedgehogs has been fed too much and less should be given the following night. Owners *should be advised to weigh their hedgehogs weekly and adjust feeding levels accordingly*.

Hedgehogs may be slow to accept new foods therefore any dietary changes should be gradual.

Water

Water should always be available either in a shallow, heavy bottomed bowl or by a sipper bottle. Some hedgehogs take more time than others learning how to drink from a water bottle, therefore we should advise always offer water from a bowl until the owner is completely sure the hedgehog is drinking well from the bottle.

Sexing

Male

Male hedgehogs have a longer genital–anal distance (Figure 9.3). The prepuce is circular in shape found on the mid ventral abdomen. The testicles are often abdominal but gentle pressure on the abdomen will move the testicles to the para anal recesses, there is no scrotal sac. The penis can be manually extruded and has a prominent glans with bilateral hornlike protuberances. Male hedgehogs have nipples.



Figure 9.3 Male hedgehog. Note the wide genital–anal distance, the prepuce is circular and midway along the ventrum. Photo: Jenna and Matt Perlick.

Female

Female hedgehogs have a shorter genital–anal distance (Figures 9.4a & b). The urogenital opening is small and slit, a few millimetres cranial to the anus, although some older females may have a more circular appearing opening. There are three pairs of mammary glands.

Preventative health care

Regular weighing by owners is recommended, both to monitor for obesity and weight loss, which may be suggestive of an underlying condition. Hedgehogs' nocturnal nature and ball rolling tendency means *many conditions may be advanced prior to presentation*.

Hedgehogs have round, tightly curled nails, and regular nail clipping (especially if not given opportunities to dig) may be required to prevent ingrown nails and infection. A short anaesthetic will be required in virtually all hedgehogs to perform this.

Six monthly check-ups are advisable, especially due to the high prevalence of neoplasia in this species.

Neutering

Owing to their solitary nature, routine elective neutering is rarely carried out.

Hospitalisation

Like all prey species, hedgehogs find hospitalisation stressful. To minimise stress the hedgehog should be kept away from the sight, smell, and ideally sound of predators. *Ensuring dimmed lighting will reduce stress, especially as they are nocturnal*.



Figure 9.4a Female hedgehog. There is a short genital–anal distance. Most females have a slit like vulva.



Figure 9.4b Female hedgehog. There is a short genital–anal distance. Older females may have a more circular shape to the vulva.

How the kennel/cage is set up is also important. Slippery surfaces are stressful and can result in injuries, so line the floor with newspaper. Provide a box for a hide with shredded paper or a paper-based bedding, situated to allow direct visualisation of your patient.

The ward or hospitalisation cage should be kept at 27–29°C (80.6–84.2°F). Requesting that owners bring in familiar foods and live insects can encourage hedgehogs to eat, many owners love preparing a 'packed lunch' for their pet. However, it should be remembered that most hedgehogs eat mainly at night.

Hospitalisation sheets should be used, and the hedgehog should be weighed twice daily.

Assisted feeding

EmerAid Omnivore can be used for assisted feeding although jarred human baby foods, or blended low-fat dog food can be used if EmerAid is not available. EmerAid provides feeding guidelines but generally it is initially advisable

to feed 1% of the body weight, increasing the volume as the animal's appetite improves.

Tame hedgehogs may lap food from a spoon. Holding the hedgehog in dorsal recumbency in a towel will frequently cause the animal to start to unfurl, especially if the light has been dimmed, allowing the introduction of a 1 ml syringe and syringe feeding.

Live mealworms or crickets will often stimulate feeding in hedgehogs who are reluctant to eat familiar foods or refuse all attempts at assist feedings.

Intractable patients may require an oesophagostomy tube placement, which is performed similar to cats. This procedure is challenging in obese animals and care should be taken to ensure enough tubing is available to allow access to the feeding port even when rolled into a ball.

Venipuncture sites

Any venipuncture must be *performed under sedation or anaesthesia* due to the hedgehog's ability to curl into a ball! (See Anaesthesia section).

Many exotics texts describe performing a cranial vena cava stick to draw blood. This carries the risk of death due to uncontrollable haemorrhage, which is reported in the literature. There is also an increased risk of inadvertent cardiac puncture due to the heart being only 1cm away from the cranial vena cava in this species.

A maximum volume of 1% of bodyweight can be taken during venipuncture in healthy individuals, reducing to 0.5% in ill animals.

Veins suitable for venipuncture include the lateral or medial saphenous vein, cephalic vein, jugular vein, or femoral vein (Figures 9.5a–d). A 25–27g hypodermic needle and a 1–3 ml syringe will be suitable for most animals, although it is also acceptable to drip the blood from the needle directly into the collection tube. As for rodents, application of EMLA 5% cream (2.5% lidocaine, 2.5%



Figure 9.5a The cephalic vein.



Figure 9.5b The cephalic vein highlighted in yellow.



Figure 9.5c The lateral saphenous vein.

Table 9.1 African pygmy hedgehog biochemistry.

BIOCHEMISTRY (Heatley & Russell, 2020)	REFERENCE RANGE (UK units)	REFERENCE RANGE (US units)
ALKP	30–72 IU/l	30–72 U/l
ALT	29–77 IU/l	29–77 U/l
AST	16–56 IU/l	16–56 U/l
AMYL	340–680 IU/l	340–680 U/l
GGT	3–5 IU/l	3–5 U/l
LDH	183–699 IU/l	183–699 U/l
CK	450–1276 IU/l	450–1276 U/l
LIPASE	3.058 IU/l	3.058 U/l
GLU	3.27–6.61 mmol/l	59–119 mg/dl
CHOL	2.74–4.04 mmol/l	106–156 mg/dl
TRIGYLCERIDES	0.18–0.68 mmol/l	16–60 mg/dl
TBIL	0–10.2 µmol/l	0–0.6 mg/dl
UREA	3–6 mmol/l	18–36 mg/dl
CREA	17.7–53.1 µmol/l	0.2–0.6 mg/dl
TP	51–65 g/l	5.1–6.5 g/dl
ALB	25–33 g/l	2.5–3.3 g/dl
GLOB	22–32 g/l	2.2–3.2 g/dl
CA	1.85–2.55 mmol/l	7.4–10.2 mg/dl
PHOS	1.1–2.3 mmol/l	3.4–7.2 mg/dl
K	3.9–5.9 mmol/l	3.9–5.9 mEq/l
Na	132–150 mmol/l	132–150 mEq/l

Table 9.2 African pygmy hedgehog haematology.

(Heatley & Russell, 2020)		REFERENCE RANGE
Red blood cells	10^{12}/l	
Packed cell volume	%	29–43
Haemoglobin	mmol/l (g/l)	63.92–85 (103–137)
Mean corpuscular volume		44.6–55.4
Mean corpuscular haemoglobin concentration		287–343
Mean corpuscular haemoglobin		13.8–17.6
Platelets	10^9/l	
White blood cells	10^9/l	5–17
Differential blood count (absolute values)		
Monocytes	10^9/l	0–0.6
Lymphocytes	10^9/l	1.8–6.2
Neutrophils	10^9/l	3.3–10.2
Eosinophils	10^9/l	0.3–2.1
Basophils	10^9/l	0.1–0.7

prilocaine, Aspen) on sites of peripheral veins may help facilitate venipuncture.

Biochemistry and haematology profiles can be run 'in-house' using our usual mammalian profiles or sent to an external laboratory. Both options have pros and cons. The main pro for running in-house is that you obtain your results quickly whereas the main con is that you have to interpret whereas many labs will provide interpretation upon request. That being said, *biochemistry results are similar to dogs but hedgehogs normally have a higher urea and lower sodium than dogs*, see Table 9.1 for biochemistry and Table 9.2 for haematology reference ranges.

Jugular vein blood sampling in hedgehogs is slightly different compared to other species, and Doss (2020) described his technique for sampling the jugular vein at the thoracic inlet, where the internal and external jugular veins split (Figure 9.5d).

Procedure to perform a jugular vein blood sample.

- Use a 25–27g hypodermic needle and a 3 ml syringe.
- Place the hedgehog in dorsal recumbency with either the head facing towards or away from the



Figure 9.5d A jugular vein sample being taken as described by Doss (2020).

person performing the venipuncture. Palpate manubrium to locate the clavicle and use the tip of a gloved finger to locate the indentation between the base of the neck and the attachment of clavicle to the manubrium (moving the forelimb cranially and caudally can help locate the area by mobilising the clavicle). Swab the area with diluted chlorhexidine.

- Insert the needle at a 90-degree angle to the skin, into the indentation, as close to the cranial edge of the clavicle as possible. Start to gently aspirate as soon as the skin is punctured but in most hedgehogs the vein will be approximately 1 cm deep to the skin. Remove the needle and use a cotton bud to apply pressure to the site for 30 seconds to prevent haematoma formation.

Dehydration

Hydration status in hedgehogs can be challenging to ascertain. Skin tent, eye position, tacky mucous membranes, altered mentation, and weight loss can all be utilised.

Although any vein can be used for intravenous (IV) catheterisation, the defensive strategy of rolling into a ball, makes the use of IV catheters unfeasible in any but the most debilitated hedgehog.

Placement of a tibial intraosseous (IO) catheter should be considered in cases where fluid therapy is required. Placement is similar to other species, performed under an anaesthetic, and a 22–25g hypodermic needle with a standard injection catheter cap attached can be used. Tape the needle in place firmly with zinc-oxide tape and take two orthogonal views radiographs to ensure proper placement. *The advantage of placing the IO line in the tibia is that it is still accessible even when the hedgehog rolls into a ball* (Lennox, 2007).

Intraperitoneal (IP) fluids may be given but carry the risk of inadvertent organ puncture. A bolus of 5–10 ml may be given into the lower right quadrant of the abdomen. Holding the anaesthetised hedgehog in dorsal recumbency, with its head pointing towards the floor, will utilise gravity to shift the abdominal organs cranially, reducing the risk of accidental organ puncture.

The exact maintenance fluid rate is unknown for hedgehogs but is assumed to be 50–100 ml/kg/day. Fluids should be warmed to 35.4–37°C (95.7–98.6°F) prior to administration.

Drugs contraindicated in African pygmy hedgehogs

Owing to their simple digestion, non-reliance on hind gut fermentation, and omnivorous nature, most drugs that can be used in dogs and cats may be used in hedgehogs. Compounding companies may be able to create palatable versions of medications, many hedgehogs seem to like fruit flavoured drugs.

Species-specific anatomy/behaviour

Hedgehogs hide illness and pain.

Hedgehogs have a *lower core body temperature than most mammals*, 35.4–37°C (95.7–98.6°F).

African pygmy hedgehogs are omnivores belonging to the Erinaceidae family and have brachydont dentition (brachydont: close rooted, not continuously growing). Their dental formula is: 2 × (I 3/2; C 1/1; P 3/2; M 3/3) = 36. There is a large gap between the first sets of upper and lower incisors with the lower set fitting into the upper gap at rest.

Hedgehogs (wild and captive) perform a 'self-anointing' behaviour, often when exposed to a strong new smell or object. Thick foamy saliva is produced that is then frantically groomed over the quills. Why this behaviour is performed is unknown.

They have a simple digestive tract, no caecum and *can vomit*.

There are only four toes on the hind feet, and five toes on the front feet.

Their tibia and fibula are fused distally, and the radius and ulna are fused.

The defensive mechanism is to roll into a ball by contracting the panniculus muscle which pulls the loose skin of the dorsum (mantle) over the body, while they tuck their head and legs under, and the orbicularis muscle tightens like a purse string suture to close the aperture. The mantle is protected by thousands of unbarbed spines.

If the panniculus or orbicularis muscle is damaged by trauma or surgery, it must be repaired in a separate layer to the skin. *Failure to do so will result in wound breakdowns when the hedgehog curls into a ball*.

Sick hedgehogs may lose the ability to regulate their body temperature and become torpid.
Never assume a hedgehog is in shock or near death without warming the animal first as it may be torpid.

Hedgehogs enter torpor when exposed to low or extremely high (>30°C) ambient temperatures and which may mimic being in shock.

Any hedgehog presenting in torpor should be warmed before drug or fluid administration. This is because the metabolism drops so low the drugs and fluids will not be absorbed or metabolised properly.

Males have large seminal vesicles which may account for up to 10% of their body weight when reproductively active and may be palpable in the caudal abdomen. Male hedgehogs have open inguinal canals.

Pain in African pygmy hedgehogs

In Chapter 1 we discussed that all vertebrates have the same anatomical and physiological pain pathways. One of our jobs as veterinary professionals is to *actively look for pain,* however, *hedgehogs are particularly difficult to assess for pain as clinical exams are rarely able to be performed in a conscious animal. In addition, their nocturnal nature may mean that owners do not observe subtle abnormal behaviours, therefore the pathology may be severe before the owner presents the animal*. Any change in a hedgehog's behaviour, weight loss (unless planned), or demeanour, no matter how subtle, should be viewed as significant. Additionally, owners may not be aware that hedgehogs are classed as geriatric by 3 years old, so these changes could also indicate serious age-related health issues.

There is very little in the literature on specific studies performed in hedgehogs investigating pain and analgesic efficacy. Doses can be found in James Carpenter's *Exotic Animal Formulary* now on its sixth edition (Carpenter & Marion, 2022). They are extrapolated from other omnivores, carnivores, or based on experience. Regular reassessment to ensure adequate control of pain is being achieved is vital.

One way to assess pain is to administer an analgesic (as a therapeutic trial) and if the hedgehog resumes a more normal behaviour, the pretreatment signs were most likely down to pain. I find therapeutic trials useful and if I direct owners to three certain behaviours to monitor, it can really help the owners to understand and see the improvement in their pet.

Osteoarthritis

Any species with a skeleton can develop osteoarthritis, in species that actively hide pain we have to actively look for skeletal pain in these species. Hedgehogs develop osteoarthritis and obesity will exacerbate it. *Limping or changes in gait may indicate osteoarthritis but may also indicate other conditions such as: trauma, constriction injury of a foot, intervertebral disc disease, or neurological disease. Full clinical examination and diagnostics such as imaging and blood work early in the case will provide the best chance of a definitive diagnosis in these secretive patients, allowing a more accurate treatment plan and prognosis*.

Analgesia

There is very little in the literature on specific studies performed in hedgehogs investigating pain and analgesic efficacy. One study on buprenorphine found that a single dose of 0.05 mg/kg SC provided analgesia for 48 h (Doss & Mans, 2020). Doses can be found in Carpenter's *Exotic Animal Formulary* and are extrapolated from other omnivores, carnivores, or based on experience.

Consideration of the type of pain we are looking to control will lead to logical choices of the type of analgesics we select. *Multimodal analgesia is recommended*. Regular reassessment to ensure adequate control of pain is being achieved is vital.

Clinical exam

The majority of hedgehogs are reluctant to be handled by unfamiliar people and will use the defensive mechanism of curling into a ball, often also hissing and spitting at the same time. The quills are very sharp and while they are unlikely themselves to do much damage to human skin, the fact that *they regularly walk through and roll about in their own faeces means any wounds produced by quill pricks have the potential to become septic*. Thick towels or thin leather gloves may be worn to handle conscious hedgehogs. There are several methods suggested to uncurl hedgehogs for clinical exams but in my experience none of them are reliable and a full clinical exam cannot be performed.

A hedgehog who is normally defensive but becomes suddenly easy to handle is likely to be severely ill.

If the hedgehog presents in a state of torpor, a clinical exam may be possible without anaesthesia, but *fluids or medication should not be administered until the patient has been warmed to normal body temperature*. Venipuncture is likely

Table 9.3 African pygmy hedgehog analgesics.

Analgesic	Dose
Meloxicam	0.2–0.5 mg/kg PO, SC q12–24hrs
Buprenorphine	0.05 mg/kg SC q48hrs
Gabapentin	5–20 mg/kg PO q8–12hrs
Maropitant	2 mg/kg SC
Methadone	0.3–0.7 mg/kg SC q4–6hrs dependant on pain scoring
Tramadol	5–15 mg/kg PO q6–12hrs
Lidocaine	2 mg/kg (maximum 4 mg/kg)
Bupivacaine	1 mg/kg (maximum 2 mg/kg)
Acupuncture	

Table 9.4 Biological data.

Parameter	Reference range
Average longevity	3–5 years
Respiratory rate	25–50 bpm
Heart rate	180–280 bpm
Body temperature	35.4–37°C (95.7–98.6°F)
Maintenance fluid therapy	50–100 ml/kg/day
Sexual maturity	6–8 months (as early as 2 months of age in some individuals)
Gestation	34–37 days

Table 9.5 Commonly used antibiotics in African pygmy hedgehogs.

Drug	Dose
Amoxicillin/clavulanic acid	12.5 mg/kg PO q12hrs
Ciprofloxacin	10–20 mg/kg PO q12hrs
Chloramphenicol	30–50 mg/kg SC, IM, IV, PO q12hrs
Clindamycin	5.5–10 mg/kg PO q12hrs
Doxycycline	5–10 mg/kg PO (no dairy) q12hrs
Enrofloxacin	10 mg/kg PO SC q12hrs (IM painful and may result in sterile abscess/muscle necrosis)
Erythromycin	10 mg/kg PO, IM q12hrs
Marbofloxacin	5 mg/kg PO q12–24hrs
Metronidazole	20 mg/kg PO q12–24hrs
Oxytetracycline	50 mg/ kg PO q12hrs
penicillin G	40,000 IU/kg SC q3–5days
Silver sulfadiazine cream	Topical q12–24hrs
Trimethoprim sulfamethoxazole	30 mg/kg PO q12hrs
Tylosin	10 mg/kg PO, SC q12hrs

to be more difficult and should not be performed until the animal has been warmed up.

In virtually all other hedgehogs, an anaesthetic will be required for a thorough clinical examination. Clinical exams should be performed in a similar manner as more familiar species – we are assessing body systems. Particular attention should be paid to the oral cavity, ears (should be free of debris), skin, and abdominal palpation.

Permission should be gained to perform venipuncture and diagnostic imaging if required rather than have to anaesthetise the animal again.

General anaesthesia

Reducing risks associated with general anaesthesia starts before admission. *As hedgehogs can vomit, it is recommended to fast for at least 80 minutes prior to induction.*

Advise owners to bring a 'packed lunch' of favourite and familiar foods as hedgehogs are less likely to consume unfamiliar food.

There are a number of anecdotal injectable anaesthetic protocols for hedgehogs, but doses vary widely necessitating caution with use. Injectable protocols are also reportedly unreliable (most likely due to the erratic absorption caused by subcutaneous (SC) fat layer), with rough prolonged recovery periods, and decreased food intake for days afterwards. This is why gaseous anaesthetics are favoured, despite no analgesia being provided by isoflurane or sevoflurane. Hawkins *et al.* (2020) reported that *the speed and quality of isoflurane recovery was considered superior when compared to published injectable sedation protocols*.

Pre-anaesthetic blood tests

In hedgehogs, it is impossible to perform venipuncture without anaesthesia. Blood sampling is recommended in all sick hedgehogs. See Tables 9.1 and 9.2 for reference ranges.

Pre-emptive analgesia/pre-medication

Once admitted (and if not already on medications) administer meloxicam 0.2–0.5 mg/kg and/or another analgesic if appropriate depending on the surgical procedure. These can either be given orally in the case of meloxicam or by SC injection, *ideally using the ventral furred skin* to avoid the large amount of fat present subcutaneously to the mantle which can interfere with absorption.

Whether I administer a pre-medication is dependent on the stress levels of the patient and the surgical procedure being performed. It is important to consider the stress of being handled and being injected. *What should also be considered in hedgehogs is the fact that medications injected into the mantle can have an unpredictable absorption due to the large amount of fat present subcutaneously.* In the conscious hedgehog it is very difficult to give IM injections or ensure that SC injections miss the fat layer.

In my experience induction using isoflurane while swaddled in a towel results in a fast, seemingly minimal stress induction. The drugs used for premedication are the same drugs we use in more familiar mammals. The doses can be found in Table 9.6.

Induction

My preferred inhalational gaseous anaesthetic is isoflurane as it results in a fast, minimal stress induction, and a smooth recovery. The hedgehog can be placed inside a large dog mask as an induction chamber, or alternatively,

Table 9.6 Pre-medicant dose.

Drug	Dose and route	Reversal
Midazolam	0.5–1 mg/kg IM SC	Flumazenil 0.1 mg/kg SC
Midazolam + butorphanol	0.2–0.5 mg/kg + 0.2–0.5 mg/kg IM	Flumazenil 0.1 mg/kg SC
Midazolam + buprenorphine	0.2–0.5 mg/kg + 0.03 mg/kg IM	
Medetomidine + butorphanol	0.05–0.2 mg/kg IM + 2 mg/kg IM	Atipamezole 0.5–1.5 mg/kg IM SC



Figure 9.6 The end of the Ayres T Piece can be held snugly into the small opening on the ventrum.



Figure 9.7a Wiggling the tube nozzle, the nose of the hedgehog uncurls.

with the hedgehog held on its back in a towel, the end of the Ayres T Piece can be held snugly into the small opening on the ventrum of the hedgehog (Figure 9.6). Allow four breaths of 100% oxygen (this is all that is required to fully saturate the blood with oxygen), then turn the vaporiser straight to 5% isoflurane. *The nose of the hedgehog is right below this opening and as the animal will quickly become induced (most are less than 40 seconds), I find that if I gently wiggle the tube nozzle, the nose of the hedgehog uncurls and the nozzle can fit directly over the nose* (Figure 9.7a & b).

Turn the vaporiser to 2.5% and assess anaesthetic depth (using the same reflexes we are used to in more familiar mammals, a moistened cotton bud can be used to check the blink reflex), adjusting the setting on the vaporiser as necessary.

The cornea should be protected by applying an ocular lubricant.

Intubation

I rarely intubate hedgehogs, but if I am performing surgery on the head, intubation with a 16g IV cannula (with the stylet removed) or a 1–2 mm diameter ET tube is possible using a variation of the direct visualisation method in rabbits, although passing the cannula to the side of the otoscope speculum rather than down it.



Figure 9.7b Then the nozzle can fit directly over the nose.

Maintenance

I use an Ayres T Piece circuit, with an oxygen flow of 1.5 l/min. Isoflurane 2–4% as required.

Monitoring

Monitoring the anaesthetic is the same as for more familiar species. *Hedgehogs have a lower core body temperature than most mammals, 35.4–37 °C (95.7–98.6 °F), which is important to remember when monitoring for hypothermia*.

Theatre

Hedgehogs have a high surface area to volume ratio which means they lose heat rapidly. The resulting hypothermia can cause fatalities. Keeping the ambient temperature in theatre high, wrapping the extremities in bubble wrap, placing a towel on the operating table, and using a plastic drape (also allows visualisation of your patient) are all easy ways to help mitigate heat loss. Minimise the number of times the door is opened as each time it happens the ambient temperature in the room will drop. Hypothermia leads to prolonged recovery and increased risk of death.

Positioning

Hedgehogs have a small thoracic cavity in comparison to their large abdominal cavity which can make breathing more difficult due to the weight of the abdominal organs sitting against the diaphragm. Placing a folded drape or face cloth under the thorax elevates the thorax and allows the abdominal organs to move away from the diaphragm. If the hedgehog is not intubated, ensure the head and neck are straightened to allow for easier breathing.

Surgical Preparation

To conserve body temperature, minimise the surgical clip. Quills may be plucked like feathers (quite difficult to do) or a size 10 clipper blade may be used to clip them. Only one agent is used for surgical scrub, either chlorhexidine or an iodine-based skin disinfectant depending on the surgeon's preference. We do not use a surgical spirit-based spray as during evaporation this causes loss of heat. Using warm water for the surgical scrub will help reduce heat loss.

Transparent, plastic surgical drapes are ideal as they allow visualisation of the patient while conserving body heat. The required surgical window can be cut into the drape and a surgical spray adhesive is used in lieu of towel clips.

EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) can be applied to the wound postoperatively if no other local anaesthetic infiltration has been performed in the skin. This helps with the immediate post-operative skin pain.

Recovery

Maintain the hedgehog on oxygen, and if intubated, extubate when the jaw tone increases. Shivering thermogenesis is a sign of recovery from anaesthesia. Continue to monitor while in an incubator or a warm room (ideally around 27°C (80.6°F)) until up and moving then the temperature of the room can be reduced.

Syringe feed (EmerAid Omnivore) once the hedgehog is fully conscious.

Advise the owner to monitor food intake and weigh daily to ensure there is adequate food/fluid intake. In the vast majority of cases with good analgesia this will not be an issue (Table 9.3).

Obesity

Over feeding and lack of exercise are extremely common in African pygmy hedgehogs. *Some owners equate love with food*, taking great joy in feeding their pets their favourite foods which are often high in fat. *Obesity can cause: an inability to fully roll up, osteoarthritis, hepatic lipidosis, and potentially cardiac disease*.

Education about required exercise and the reductive effect obesity can have on lifespan are key to get the owners onside as I find shame-based approaches rarely work. *Gradual reduction in food, increasing the plant component of the diet, increasing exercise, and providing more foraging opportunities* will help decrease body condition and increase fitness. Owners should weigh their pet weekly.

Dental disease

Hedgehogs have brachydont dentition (Brachydont: close rooted, not continuously growing). The dental formula is: $2 \times (I\ 3/2;\ C\ 1/1;\ P\ 3/2;\ M\ 3/3) = 36$. The teeth are white with a large gap between the first sets of upper and lower incisors with the lower set fitting into the upper gap at rest, which is suited to spearing invertebrates. The canines are small, and the premolars and molars are flat and broad.

Dental disease is extremely common in hedgehogs and has been associated with feeding a mainly moist or soft diet. Plaque build-up, periodontal disease, fractured teeth, and tooth root abscesses are frequently seen. Clinical signs are as expected: halitosis, pawing at the mouth, drooling, anorexia, and weight loss.

Radiographs may help accurately determine the abscessed tooth (see Figure 9.8). Analgesia, systemic antibiotics (my antibiotic of choice for periodontal infection is clindamycin), and extraction of the affected tooth as per more familiar species is required (Figure 9.9). Cat dental instruments can be used to elevate and extract the tooth. Scale and polish as per more familiar species.

As squamous cell carcinoma is very common and may mimic periodontal or gingival disease, treatment failure should raise the index of suspicion for neoplasia and a biopsy should be performed (Figure 9.10).

Prevention

Feeding a mainly dry kibble diet, raw vegetables, and live invertebrates with exoskeletons such as beetles or

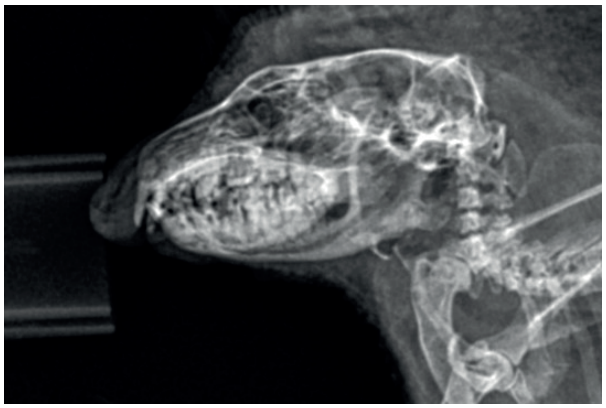


Figure 9.8 Radiograph showing tooth root infection of a maxillary molar leading to loss of bone.



Figure 9.9 Extraction of the molar radiographed in Figure 9.8.

crickets may help improve oral health by reducing plaque build-up.

Palatable enzymatic dog or cat oral pastes may be used to help reduce risk of gingivitis or tartar build-up, by placing the paste on a piece of favourite kibble. Excessive wear of teeth may occur in aged animals necessitating a softer diet being fed (Figure 9.11).

Oral masses

Oral masses are usually diagnosed in hedgehogs over 2 years of age.

Gingival hyperplasia

Gingival hyperplasia is seen initially as a focal area of gingival thickening, slowly growing into a protruding, non-ulcerated mass and is commonly associated with dental disease. There may be a reduced appetite and gingivitis. Gingival hyperplasia may be seen in variable locations and surgical resection is curative.



Figure 9.10 Squamous cell carcinoma may mimic periodontal or gingival disease. The hedgehog in this Figure presented with a facial swelling which may have been a tooth root abscess but turned out to be an infected squamous cell carcinoma.



Figure 9.11 There is excessive wear present on the molars.

Oral squamous cell carcinomas

Squamous cell carcinomas (SCC) are extremely common and in the early stages may mimic periodontal disease and gingival hyperplasia (see above). They are highly locally invasive (soft tissue and bone), causing deformity, swelling, loss of teeth, halitosis due to secondary infection and tissue necrosis, and reduced appetite. Ulceration may be present, and the tumour *can either protrude from the site or invaginate*. Metastasis to the lungs and regional lymph nodes have been recorded.

Del Aguila *et al.* (2019) reported that over 70% of the SCC they diagnosed occurred caudal to the right maxillary

second premolar, 6% occurred caudal to the left maxillary second premolar, 23.5% occurred caudal to the mandibular second premolar (2 left, 2 right). *They concluded that clinical signs, growth pattern, and anatomical location could be used to suspect a diagnosis of SCC, however histopathology is still required for a definitive diagnosis* (Figures 9.12a–d).

Surgical resection is difficult due to the invasive nature of the tumour and recurrence is common.

A spindle cell tumour and a hemangiosarcoma have also been reported as occurring in the oral cavity of hedgehogs.

Respiratory disease

The respiratory system in hedgehogs is similar to that of other small mammals, and they normally have a moist nose.

Predisposing factors for respiratory disease (RD) include suboptimal environmental temperature, dusty or



Figure 9.12a Squamous cell carcinoma which has led to the loss of teeth and is encroaching on the hard palate.



Figure 9.12b An intra-oral view of the hedgehog pictured in 9.10. which at first examination may be mistaken for dental disease.

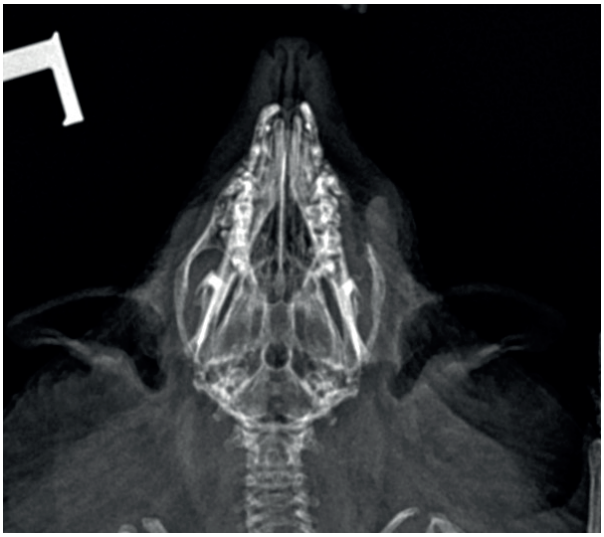


Figure 9.12c A radiograph of the hedgehog in Figures 9.10 and 9.12 showing the invasive nature of a squamous cell carcinoma and the level of bone destruction.



Figure 9.12d An intra-oral view of the hedgehog pictured in 9.10 and 9.12b once the discharge has been cleaned out of the sockets. Histopathology tissue from the right upper arcade confirmed a squamous cell carcinoma. Note the very different appearance from the squamous cell carcinoma pictured in 9.12a.

unsanitary bedding, malnutrition, concurrent disease, and any cause of immunosuppression. Pneumonia is generally more common in hedgehogs that are old, debilitated, and/or have concurrent disease but can occur at any age. The normal RR is 25–50 breaths per minute.

Lungworm nematodes *Capillaria aerophila* and *Crenosoma striatum* are common in Eurasian Hedgehogs, can affect African pygmy hedgehogs, but are considered rare in captive bred animals. However, as *Capillaria aerophila* is found in earthworms as a carrier host, and *Crenosoma striatum* is found in land snails or slugs as intermediate hosts, it is *important to question the owner as to whether they forage for earthworms, slugs, and snails to feed to their pet hedgehog* as if the answer is yes, we need to remember to include these worms in our differential list for RD. Additional clinical signs associated with lung worm involvement include weakness, emaciation, anaemia, and death.

Clinical signs

Clinical signs are similar to those seen in other species and include coughing, sneezing, nasal discharge, exercise intolerance, abdominal breathing, and wheezing. *Hedgehogs make a variety of grunts, snuffles, and what can sound like wheezes as part of their normal vocal range, so it is important to check in with the owner what noises they are worried about and what is normal for their pet.*

Placing the hedgehog in a transparent box will allow visualisation from below to observe the colour of the lips and feet. The rest of the clinical exam generally requires an anaesthetic in the vast majority of patients.

Investigation

If the hedgehog is fed on wild sourced prey items such as earthworms, slugs or snails, *samples of saliva, any nasal discharge, and faeces should be examined for the bipolar eggs of* Capillaria *and/or the larvae of* Crenosoma *as they frequently occur together.* Treatment options for lungworm include: ivermectin, levamisole 10 mg/kg SC repeat after 48 h then in 14 days, tetramisole, or fenbendazole 10–30 mg/kg q24hrs for 5 days.

Their nasal passage is very small, making deep nasal swabs impossible to perform. Tracheal swabs or tracheal washes are challenging to perform but not impossible. Corynebacterium has been reported to cause bronchopneumonia and pulmonary abscesses. Other bacteria cultured from the respiratory tract include *Pasteurella*, *Bordetella bronchiseptica*, *Haemophilus*, *Salmonella*, *Staphylococcus*, and haemolytic *Streptococcus*.

Radiography

Thoracic radiography is useful both to assess the lungs, and to check for neoplasia, or cardiac disease as they may both present as respiratory signs. When positioning for lateral radiography, *it is important to pull the mantle dorsally* (using surgical tape if needed). This *will remove the quills from the radiograph and enable easier interpretation of images*. The DV view is still valuable but has the added complication of the quills (Figures 9.13a–c).

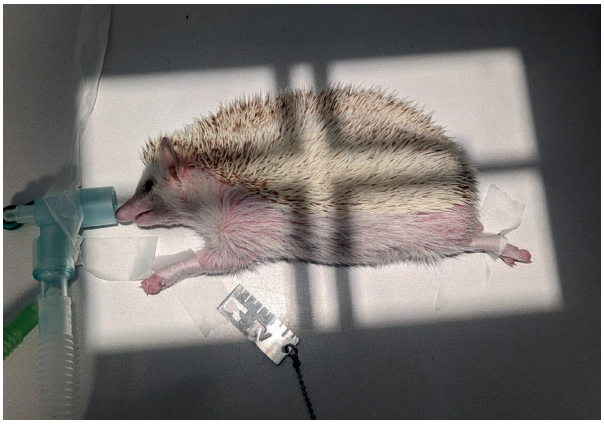


Figure 9.13a Positioning for lateral radiograph without pulling the mantle dorsally. The spines are radiopaque and may either mask or mimic pathology.



Figure 9.13b Positioning for lateral radiograph pulling the mantle dorsally.

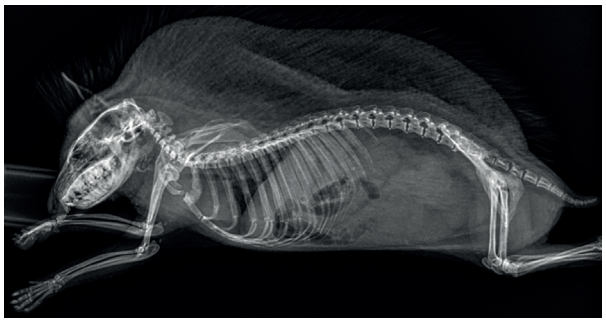


Figure 9.13c A lateral radiograph with the mantle pulled dorsally.

Treatment

Antibiotic options should be ideally guided by culture and sensitivity. Antibiotic choices for dogs or cats for respiratory system disease can be used in hedgehogs (see Table 9.5 for commonly used antibiotics in African pygmy hedgehog). An analgesic should be given and unless steroids have been used because of a severe lungworm infection, I usually use meloxicam. Some cases may benefit from bromhexine to help clear mucous, although owners should be advised the cough may initially worsen.

Nebulisation therapy

Oral medication can be challenging to give to hedgehogs unless they are very palatable (and the patient is still eating), or the animal is very tame, allowing syringe administration. Nebulisation can be a useful method of medicating as per pet rats. See Table 9.7 for suggested doses.

Cardiac disease

A high incidence of cardiomyopathy is reported in African pygmy hedgehogs, approaching 40% in one study (Raymond & Garner, 2000). Cardiac disease is diagnosed predominantly in male hedgehogs over 3 years old but has been documented in a 1 year old and reported in females. Cardiac insufficiency as a result of myocardial mineralisation has been recorded in association with chronic renal failure. The normal heart rate is 180–280 bpm.

Clinical signs

Respiratory signs are commonly associated with cardiac disease in many species and hedgehogs are no different. There is a risk of sudden death associated with cardiac disease. Dyspnoea, tachypnoea, pallor, or cyanosis of the lips, ears, feet, and tail, or extremities that feel cool to the touch are commonly seen with heart disease. A heart murmur, irregular heart rhythm, ascites, oedema, or weak femoral pulses should raise the index of suspicion for heart disease as it would in more familiar species. *Acute renal tubular necrosis due to poor renal perfusion is commonly associated with cardiomyopathy and biochemistry assessment is advisable*.

Table 9.7 Nebulisation drugs suggested in African pygmy hedgehogs.

Drug	Dose (most nebulisers will hold 10 ml)
Saline	10 ml good for hydration of respiratory tract
Acetylcysteine (mucolytic)	10–20 mg per 1 ml saline (higher doses may be associated with bronchospasm)
Bromhexine (mucolytic)	0.15 mg per 1 ml saline
F10	1:250 or ready to use (already prediluted)
Amikacin	5 mg per 1 ml saline
Doxycycline	13 mg per 1 ml saline
Enrofloxacin	10 mg per 1 ml saline
Gentamicin	5 mg per 1 ml saline

Assessing cardiac size radiographically

Normal cardiac size

The normal position of the heart is lying between the fifth and seventh ribs, and there should be no tracheal elevation.

Vertebral heart score

Black *et al*. (2011) established a vertebral heart score (VHS) for African pygmy hedgehogs, which can be used as a tool to aid in the diagnosis and monitoring of cardiomegaly. The method is very similar to what we perform in canine VHS using a right lateral radiograph. A line is drawn from the carina to the cardiac apex to form the long axis (LA), the short axis (SA) line is drawn perpendicular to the LA along the widest point of the heart. Both the LA and the SA are measured independently using the vertebrae, starting at the fourth thoracic vertebrae (T4), to the nearest 0.25 of a vertebra. Both measurements are added together to form the VHS.

> The normal VHS for hedgehogs was established to be: 8.16 +/– 0.48 (7.25–8.75).

Black *et al*. (2011) also reported two hedgehogs with significant valvular insufficiency had VHS much higher than normal *which suggests that VHS may also be useful for diagnosing asymptomatic valvular insufficiency*.

Treatment of cardiac disease

Treatment options include: enalapril 0.5 mg/kg q24hrs, furosemide 2.5–5 mg/kg q8hrs, pimobendan 0.3 mg/kg q12hrs, and digoxin 0.01 mg/kg q12–24hrs. Treatment may initially alleviate clinical signs, but prognosis is generally poor. *This may be due to the fact that heart disease is often advanced before clinical signs become apparent*. Earlier diagnosis may improve prognosis.

Dermatology

Skin disease is the most common reason for owners presenting their hedgehog to the practice according to several reports. This is likely due to the fact that owners can easily notice skin disease.

Mites are the most common cause of skin disease in this species, with secondary dermatophytosis or pyoderma being common. Epitheliotropic T-cell lymphoma may present as a non-healing, crusting, ulcerated lesion, so *if a lesion is*

not healing as expected, a skin biopsy is recommended. Normal quills do not easily epilate, so it is abnormal to lose more than a couple of spines during an examination.

The mantle of the hedgehog (dorsal surface head, neck, and body) is covered in thousands of spines, apart from a 2 cm linear tract midline on the crown of the head which is bald. Significant fat stores are found subcutaneously, and brown fat is found dorsally. *Subcutaneous drug absorption may be erratic, which does not appear to be clinically significant for most drugs, but it is one of the reasons that injectable anaesthesia is not commonly advocated in hedgehogs*. A mild pruritus is normal during quilling (change of spines).

The face, legs, and ventrum of the hedgehog are spine free and covered with pale fur. There are sweat and sebaceous glands ventrally and on the soles of the feet.

Ectoparasites

Most hedgehogs carry a few *Caparinia tripilis* mites and some authors regard them as commensal, however, stressful situations will cause the host parasite relationship to become imbalanced and parasite levels may increase. *Secondary dermatophytosis is common with severe mite infestations, therefore failure to fully respond to mite treatments should raise suspicion of a secondary fungal infection*. All hedgehogs presented to the clinic should be examined for mites and mixed species mite infestations have been recorded. *Severe cases of infestation should be fully worked up to find the underlying reason for immunosuppression*.

Caparinia tripilis are non-burrowing mites most commonly found on hedgehogs and are just visible to the naked eye as mobile, white/yellow powdery spots. Hedgehogs as young as 4 months old can be affected. *Chronic cases may present in acute crisis as the hedgehog may have hidden clinical signs until severely debilitated*. Mites are easily recognisable on microscopic examination as they have three long setae on the third pair of legs (remember 3 on 3).

Clinical signs can vary greatly from subclinical to severe. The hedgehog may present for pruritus and quill loss. White or brown crust (mite droppings) and hyperkeratosis may be seen at the base of quills, around the eyes, and behind the ears. Powdery white deposits may be seen around the eyes and nose. Generalised erythema, areas of lichenification, and self-inflicted excoriations may be seen. Quill loss can be severe and if the mites enter the ear canals (common in severe infestations) and secondary otitis externa may result. Lethargy, decreased appetite, weight loss, dehydration and death can occur if left untreated.

Otodectes cynotis and *Notoedres cati* have both been reported as a cause of ear disease in hedgehogs, causing pruritus, crusting around the ears, and waxy debris in the ear canal. Examination of the debris and crusts under microscope will achieve diagnosis. *It is recommended to fully clean the ear canals while the hedgehog is under anaesthesia as owners will not be able to apply ear drops or clean the ears at home*. The external ear canal is very small, and the tympanic membrane is unlikely to be visualised without an endoscope.

Demodex canis was reported in a single case study as causing a generalised erythema, with scaling around the ears, thorax, pelvis, and the central portion of the abdomen. The quills were easily epilated. The mite was diagnosed on skin scrape with speciation occurring via DNA. This case had failed to respond to ivermectin and although selamectin or the combination of imidacloprid 10% + moxidectin 1% has been shown to work in cases of *Demodex*, a single dose of the combination of 2.5 mg/kg afoxolaner + milbemycin oxime resolved all clinical signs with no apparent side effects.

Sarcoptes scabiei is a zoonotic, which has been recorded in hedgehogs.

Ornithonyssus bacoti, also known as the tropical rat mite, feeds on, but does not live on the animal and will be seen as black/dark brown dots in the substrate. Clinical signs include faking skin and quill loss. Owners should be counselled that it is zoonotic (although usually will not preferentially bite humans) and that due to most of the life cycle occurring off the host, complete eradication can be difficult. The mite may carry diseases (e.g. murine typhus, Q fever, both of which are rare in the UK) transferable through the bite to humans.

Diagnosis

Diagnosis can be achieved by clinical exam, using cellophane tape to gather material from areas of alopecia to examine under the microscope. Skin cytology can also be performed the same way to check for bacterial or fungal involvement. Quills may be plucked from the edge of lesions to be examined or a sterile toothbrush can be used to take a brush sample of mites from spines even in conscious rolled hedgehogs. Skin scrapes should be performed with extreme care as hedgehogs have a thin epidermal layer over their mantle.

Treatment

Mites are easily treated with topical selamectin 20–30 mg/kg, single application is likely to be curative in most cases, although another application can be applied 30 days later. All hedgehogs in the house should be treated at the same time.

Other treatments options include, as a single dose, imidacloprid 10% + moxidectin 1% 0.1 ml/kg (Advocate for cats) topically, fluralaner 15 mg/kg PO (Bravecto), and sarolaner 2 mg/kg PO. Some infestations did not respond to ivermectin, which is why I no longer use ivermectin in this species.

All bedding should be destroyed, and the enclosure lined with paper until treatment has eliminated the mites. Cage furniture should be cleaned and disinfected.

Dermatophytosis

Dermatophytosis is extremely common in hedgehogs. *Trichophyton erinacei* is the most common isolate. *Trichophyton mentagrophytes* or *Microsporum canis* are also reported and it should be remembered that hedgehogs (like other species) can be asymptomatic carriers of this zoonotic disease. *There are many reported cases of dermatophytosis in people in association with owning hedgehogs and veterinary staff should wear gloves if handling a hedgehog with skin disease*.

Clinical signs and diagnosis

Clinical signs of dermatophytosis and mite infestation are very similar, and we should remember that dermatophytosis often occurs secondarily to mite infestations. Most cases are non-pruritic unless there is a concurrent mite infestation or a secondary bacterial pyoderma. Facial alopecia occurs with crusts forming on the edges of the pinnae, crusting around the base of the quills and quill loss on handling. It is most frequently seen on the head with slow spread to the rest of the body.

Diagnosis is via microscopic examination of the hair shaft and fungal culture. *Trichophyton mentagrophytes* does not fluoresce under a Wood's Lamp.

Treatment

Treatment is always recommended due to the zoonotic risk for the owners. *Topical treatment is not recommended due to hedgehogs finding dips and baths extremely stressful* – this can lead to immunosuppression, and treatment failure may occur due to the ventrum of the hedgehog not being treated.

Systemic therapy with either itraconazole 10 mg/kg q24hrs or terbinafine 100 mg/kg q24hrs should be started and continued until two negative fungal cultures are obtained a month apart. *Not all owners will be able or willing to pay for repeated fungal cultures*. Treatment should be for a minimum of 4 weeks and if owners cannot or will not repeat the cultures this should be recorded in the notes and owners should be made aware that without the tests, we cannot guarantee full resolution just because the obvious clinical signs have resolved. *Studies in Eurasian Hedgehogs report terbinafine is much more effective than itraconazole*.

Disinfection of the environment with a suitable agent such as sodium hypochlorite or F10 is an important part of management. Wooden enclosures may be difficult to eradicate the dermatophytes from.

Pinnal dermatitis

Pinnal dermatitis is common and the animal presents with bilateral ragged, hyperkeratosis of the ear margins. Differentials include: dermatophytosis, mites, nutritional deficiencies, and poor humidity. A full husbandry review, examination of any aural debris and skin cytology should gain a diagnosis with treatment depending on the underlying cause.

Pododermatitis

Hedgehogs are messy creatures and have a habit of walking through their faeces, spreading them throughout their enclosure. If the substrate is not changed frequently enough, the build-up of urine and faeces can cause pododermatitis. More frequent cleaning should be instigated. *Bacterial infections will require systemic therapy as topical therapy will not be possible. Analgesia should be given*.

Hair or fibres may become trapped around toes of the feet causing constriction injuries, avascular necrosis, and secondary bacterial infections. The hedgehog is often badly lame, but the condition may be advanced before presentation. The material constricting the foot should be cut away and the area assessed for viability. Amputation may be required if the foot is unsavable. Ideally a swab should be taken from any wounds for culture and sensitivity and antibiotic choice guided by the results. Clavulanated amoxicillin 12.5 mg/kg q12hrs may be given while waiting on the results in cases of bacterial infection.

Allergies

Allergic skin disease is reported and may be diagnosed on skin biopsy, or suspected on response to treatment after ruling out mites, etc. Clinical signs are similar to more familiar species, progressive alopecia, pruritus, and facial swelling. Treatment options include antihistamines, steroids, using dog and cat doses, or dietary change.

Skin neoplasia

Nodular or ulcerative lesions (*especially if failing to respond as expected to treatment*) may be neoplasia and investigation via a FNA or biopsy is advisable.

Lymphoma may present as a non-healing, crusting, ulcerated lesion. Papilloma, SCC, mast cell tumour, sebaceous gland carcinoma, cutaneous hemangiosarcoma, various stromal tumours, and mammary tumours (see neoplasia) are recorded. Radiotherapy and chemotherapy have been attempted but generally reported to be unsuccessful. Future studies may provide other treatment options but for the moment, *surgical resection is the treatment of choice*.

Papilloma are sporadically seen and may range from single nodules through to widely disseminated masses seen over most of the integument.

Cutaneous mast cell tumours are generally benign although an invasive form is reported. They are most frequently located on the head, neck, or axillae as single or multiple masses.

Hemangiosarcoma are sporadically seen as dark red to black, ulcerated cutaneous masses. Metastasis may occur to the lung, liver, or kidney. Recurrence is reported in one case 7 months post-resection.

Sebaceous gland carcinoma are often seen on the ventral chest as firm, non-movable, subcutaneous masses. Pulmonary metastasis is recorded and recurrence as soon

as 3 months post-resection has been recorded. The cut surface of the mass will ooze a purulent exudate from a necrotic centre.

Cutaneous stromal tumours are locally invasive but slow to metastasise. Lipoma, fibroma, fibrosarcoma, soft tissue sarcoma, myxoma, and myxosarcoma are all reported. Peripheral nerve sheath tumours are reported with some appearing as a red, raised skin mass.

Neoplasia

African pygmy hedgehogs are *very prone to neoplasia* and neoplasia is reported as occurring in all body systems. *Up to 85% of neoplasia in this species is malignant* and generally carries a poor prognosis. Earlier diagnosis and intervention may improve prognosis. Unfortunately, *many cases are diagnosed in chronically debilitated animals presented for urgent care due to their ability to hide illness, their nocturnal nature, and their reduced tolerance to regular handling by the owner*.

The three most commonly seen neoplasia are: mammary adenocarcinoma, alimentary lymphosarcoma, and oral SCC (see Dental disease section). It is not uncommon for hedgehogs to have more than one type of neoplasia concurrently.

The average age *neoplasia is reported is 3.5 years of age but has been reported in as young as 1 month old. Radiotherapy and chemotherapy have been attempted but generally reported to be unsuccessful*. Future studies may provide other treatment options but for the moment, early detection and surgical resection is the treatment of choice.

Mammary neoplasia

Mammary tumours are common in female hedgehogs, usually being diagnosed in animals over 3 years old. They can present as single masses or multiple nodules along the ventrum (Figure 9.14). Many of these are malignant (adenocarcinoma) and can metastasise so thoracic radiography is recommended. Surgical excision is the treatment of choice but recurrence rate is reportedly high. Primary inflammatory diseases of the mammary glands are rare in hedgehogs.

Lymphosarcoma

Lymphosarcoma is common in hedgehogs over 3 years old, may be seen in any body system and is often multicentric. Peripheral lymph nodes may be palpably enlarged (*normal lymph nodes are not palpable*).

Osteosarcoma

Several cases of osteosarcoma have been reported in hedgehogs between 2 and 5 years of age. Reported sites include the ribs, vertebral column, skull, mandible, limbs, and extraskeletal sites. Radiographic characteristics are the same as seen in other mammalian osteosarcomas, and diagnosis is via FNA or biopsy. *Unlike in dogs, metastasis appears to be rare in hedgehogs (Reyes-Matute* et al.*, 2017). Amputation in cases of appendicular osteosarcoma may be curative*.



Figure 9.14 A large mammary mass presenting as a solitary mass.

Endocrine neoplasia

Several cases of adrenocortical carcinoma and thyroid carcinoma have been reported but none of the cases displayed endocrine clinical signs.

See dermatology section for skin neoplasia.

Reproductive medicine

Sexual maturity is reached at an earlier age in captivity compared to the wild, likely due to the ready availability of nutritious foods. Both males and females are often quoted as reaching sexual maturity by 6–8 months of age, but it may be as early as 2 months of age in some individuals. It is advised not to breed until at least 6 months of age.

Female

Clinical signs of reproductive disease are dependent on the organ involved. Uterine disease will often present with vaginal bleeding or another discharge whereas ovarian disease is more likely to present with abdominal distention or palpable abdominal masses. It is common for both organs to have concurrent disease. *Vaginal bleeding may be confused for haematuria*.

Mammary neoplasia is common and is covered in the neoplasia section.

Ovarian disease

Cystic ovaries are uncommon in hedgehogs, but ovarian neoplasia is common.

Granulosa cell tumours are the most frequently diagnosed neoplasia. The median age diagnosed with granulosa cell tumour is 3.6 years (range 2.2–4.5), hedgehog presenting either with haematuria or abdominal distention due to ascites. Wu *et al.* (2021) suggested that ascites on presentation may be a prognostic indicator as their retrospective study showed that hedgehogs presenting with haematuria had a longer survival time post-surgery (>250 days) compared to hedgehogs that presented with ascites (approximately 130 days). It was also *advised to minimise handling of the diseased ovary during surgery to limit the risk of dissemination of neoplastic cells*, especially in cases with ascites. As endometrial hyperplasia and mixed endometrial tumours were identified in 87.5% of the excised uteri, they also postulated that in hedgehogs with granulosa cell tumours, overproduction of sex steroids might induce uterine diseases (similar to seen in dogs and humans). No distant metastasis was noted in this, albeit small, study. Tumour regrowth was only seen in hedgehogs with ascites during this study.

Malignant ovarian teratoma is also reported and may incorporate both ovaries and part of the uterine horn.

Ovariohysterectomy is the treatment of choice. I have had the experience of submitting a uterus for histopathology due to uterine mass but with a grossly normal ovary, and the histopathologist found a granulosa cell tumour in the ovary! It is recommended to submit even grossly normal ovaries for histopathology in hedgehogs that are neutered due to vaginal bleeding or uterine tumours.

Uterine disease

CYSTIC ENDOMETRIAL HYPERPLASIA

Clinical signs are similar to neoplastic uterine disease, i.e. vaginal bleeding.

Endometritis/pyometra

Pyometra and metritis are recorded with the expected clinical signs. Diagnosis and treatment options are the same as for more familiar species.

Uterine neoplasia

Uterine neoplasia has been recorded in females as young as 1 year of age. The most common clinical sign is vaginal bleeding. Metastasis are not uncommon and thoracic radiographs are advised (*ensure the mantle is pulled dorsally* for the lateral radiograph). Cervicouterine SCC are recorded. Ovariohysterectomy is the treatment of choice.

Ovariohysterectomy (OVH)

There is no uterine body and the horns start straight from the single cervix. The horns are tightly curved compared to other species. The ovaries are fully encased in a fat filled bursa and are found within 1cm of the end of the uterine horns. The surgery is performed as per more familiar species, and although intradermal sutures are recommended, personally I have not had any issues with dissolvable skin sutures (Figure 9.15). *Hedgehogs are more likely to interfere with their wounds if the analgesia is inadequate.*

Pregnancy diagnosis

Rather than anaesthetise the female to palpate, *any female that has been housed with a male and gains 50 g in weight within 2–3 weeks is assumed to be pregnant*. The male should be removed as he is likely to kill any young that is born, and feeding should be increased to cope with the additional energy demands of pregnancy and lactation. Provide an additional hide within the enclosure to provide her an opportunity to rest away from the young, which may help reduce the risk of infanticide. Young can be moved to separate cages at 8 weeks old.

Gestation is usually 34–37 days but delayed implantation can occur which would increase gestation to 40 days. Parturition occurs late at night or early in the morning. Female hedgehogs are very prone to stress resulting in killing of the young and owners should be advised to take care not to disturb the nest for at least five days postpartum.



Figure 9.15 Skin sutures after an ovariohysterectomy was performed due to a uterine mass and a granulosa cell ovarian tumour. With adequate analgesia hedgehogs tend to leave their wounds alone.

Dystocia

Dystocia is reported with clinical and treatment options similar to more familiar species.

Males

Prepuce

Inflammation and secondary trauma of the prepuce is associated with substrate becoming trapped in the prepuce. Removal of the substrate, gentle cleaning of the area, application of F10 barrier ointment may be useful although the owners will not be able to continue this, and analgesia. Substrate type should be reviewed to prevent recurrence.

There is one recorded case of a Sertoli cell tumour in an undescended testicle, otherwise testicular neoplasia appears to be rare.

Castration technique

Castration procedure is similar to that in guinea pigs. *Closed castration is recommended as per guinea pigs as hedgehogs also have an open inguinal canal.*

Neurology

Neurological disease is common in African pygmy hedgehogs but it should be *remembered that hedgehogs are not uncommonly presented in torpor or with severe weakness secondary to another illness which can mimic neurological disease*, so each case should be assessed, and warmth provided if needed.

Wobbly hedgehog syndrome (WHS)

Wobbly hedgehog syndrome (WHS) causes diffuse vacuolation of central nervous system (CNS) white matter, usually resulting in an ascending ataxia, progressing to paralysis and dysphagia. Most hedgehogs affected *are less than 2 years of age*, there is no known cause, although there is a strong suggestion of an inherited component, and despite many attempts, *no treatment has worked*. Survival times from onset of clinical signs to death or euthanasia are generally 15–25 months.

Clinical signs

Clinical signs may be subtle in the beginning and often wax and wane which can give false hope a treatment is working.

The first sign noted by the owner may be an inability of the hedgehog to roll into a ball. A lack of coordination, stumbling or tripping progresses into falling to one side, tremors, unilateral exophthalmos, scoliosis, neurogenic muscle atrophy, self-mutilation, severe weight loss despite a good appetite, and an ascending paralysis from the hind legs (occurs in 70% of cases) which will progress to dysphagia.

Diagnosis is presumptive after ruling out other conditions causing falling to one side (e.g. otitis interna) or progressive ataxia (intervertebral disc disease, brain tumour, hepatic encephalopathy), as definitive diagnosis is only achieved on post-mortem. *Radiography is advised to rule out intervertebral disc disease and biochemistry to look for liver involvement.*

Brain neoplasia

In one study, 11% of cases diagnosed as WHS antemortem were definitively diagnosed as brain tumours on post-mortem. *Clinical signs can be very similar, but age and rate of progression may help differentiate, although both diagnoses have a grave prognosis.*

Most cases of brain neoplasia *occur in hedgehogs over 3 years old (unlike WHS cases, which tend to be less than two), rate of progression is much more rapid leading to death or euthanasia within 1–9 months of first onset*. Some hedgehogs may display urinary bladder retention and anuria which is not seen in cases of WHS. Seizures have been reported with brain neoplasia. Several types of brain neoplasms have been recorded with astrocytoma, then ganglioma's predominating. It is highly likely brain neoplasms are underdiagnosed, being misdiagnosed as WHS without follow up necropsy.

Other conditions

Other conditions that may present with neurological signs include trauma, intervertebral disc disease, vertebral osteosarcoma, hypocalcaemia, otitis interna/media, and *Baylisascaris* species migration.

Ophthalmology

Although the approach to the ocular exam is the same as with more familiar species, the challenge is keeping the hedgehog unrolled to examine the eyes!

Some hedgehogs will allow this if they are held by their owners and the lights are dimmed (we turn off the lights but turn on an old radiograph light box to partially illuminate the room). Alternatively, placing the hedgehog upside down on a roll of sticky tape to balance the animal, will often result in them uncurling to right themselves enough to allow a quick look at their eyes or application of fluorescein. Many hedgehogs will need to be anaesthetised to look at their eyes

All hedgehogs lack a menace response and pupillary light response is challenging to assess given how tiny their pupils are.

Corneal ulcers

Corneal ulceration is very common in hedgehogs. Owners may initially notice the hedgehog partially shutting their eyes due to the pain, or a cloudy appearance to the cornea due to oedema (Figure 9.16). Some owners will not notice it at all and be surprised when it is diagnosed.

Diagnosis is the same as for other species, the application of fluorescein will highlight areas of corneal erosion.



Figure 9.16 Corneal ulceration highlighted by fluorescein application.

The challenge is the ability of the owner to apply eye drops (as per dogs) regularly. This is purely down to the nature of the hedgehogs, some owners will be able to apply the medication, especially when they know that loss of the eye is a risk of not treating if the ulcer does not heal. *Bribing the hedgehog with treats to enable the owner to put the drops in, or cradling the hedgehog in its back will allow some owners to medicate. Analgesia* should be given as we know that corneal ulceration is painful in any species.

Other hedgehogs will not tolerate application of the eye drops at all, and in these cases the best we may be able to do is provide analgesia, advise the owners the risks of non-treatment, record the conversation in the notes, and monitor the animal for signs of healing or perforation.

Cataracts

Cataracts are uncommon in this species but have been diagnosed. Hedgehogs have poor, monochromatic vision and rely mainly on their hearing and sense of smell, so seem unbothered by cataracts.

Exophthalmos

Hedgehogs have a shallow orbit, which may predispose them to exophthalmos and ocular injury. Differentials for exophthalmos include retrobulbar abscess (tooth root abscess), retrobulbar inflammation, retrobulbar tumour, or if in conjunction with neurological signs, wobbly hedgehog syndrome should be considered (occurs in 28% of WHS cases). Most of these will present unilaterally.

Enucleation is generally required and is performed as per rabbits as *hedgehogs also have a periorbital sinus. Hedgehogs cope well with one eye, the loss does not seem to bother them, possibly because they have poor eyesight anyway, relying more on hearing and sense of smell.*

Renal disease

Renal disease is common with some studies reporting an incidence of up to 50%. Tubulointerstitial nephritis is the most common histological diagnosis. Polycystic kidney disease is reported sporadically. Degenerative renal disease is most commonly seen in hedgehogs over 3 years old but renal disease has been reported in hedgehogs as young as 7 months of age.

Clinical signs are as per more familiar species: polyuria, polydipsia, lethargy, and weight loss. On post mortem the kidneys may be small with a roughed pitted surface.

Diagnosis is achieved on biochemistry, haematology, and urinalysis. Diagnostic imaging is advisable, which may show renomegaly, shrunken kidneys, neoplasia, or urolithiasis. *Myocardial mineralisation has been recorded in association with chronic renal failure.*

Treatment of renal disease

The mainstay of treatment is fluid therapy, aluminium hydroxide 100 mg/kg per feed or products such as Ipakitine (Vetoquinol) to reduce hyperphosphatemia if present, famotidine 1 mg/kg q24hrs or cimetidine 10 mg/kg q8hrs to reduce gastritis.

Advise owners to ensure the bedding is absorbent and frequently changed to wick urine away from the body, avoiding scald or pododermatitis.

Renal neoplasia is reported, including adenocarcinoma, haemangioma, and hemangiosarcoma.

Urolithiasis

All the uroliths found in African pygmy hedgehogs that were analysed by the University of Minnesota Urolith Centre up to 2020 were calcium phosphate. Unfortunately, the urine pH was not recorded in these cases as in dogs and cats, calcium phosphate stones are uncommon, and have been associated with alkaline urinary pH, hypercalciuria, and primary parathyroidism.

Clinical signs are of cystitis as seen in other animals. Diagnosis is via urinalysis (crystalluria, blood) and diagnostic imaging. Treatment is surgical removal via cystotomy as per more familiar species. There is some suggestion of a link between a cat food diet and calculi formation so changing to an insectivore diet instead of cat food, along with the plants to reduce rate of recurrence.

Urinalysis

Urinalysis normals have not been published for this species but given the diet, an acidic pH would be expected, and due to country of origin they should be able to concentrate their urine. Crystals should be considered abnormal on a sediment exam.

Gastrointestinal disease

Normal faeces are dark brown and vary in consistency from pellet-like through to soft but formed, depending on diet. Primary gastrointestinal (GI) disease (usually diarrhoea) can be caused by incorrect diet, infectious (bacterial, yeast), neoplasia, foreign bodies (hair, carpet fibre, rubber) or endoparasites (uncommon).

As hedgehogs can vomit, clinical signs associated with gastrointestinal disease are similar to dogs and cats, although often may be nonspecific. *Severe gaseous dilatation of the gastrointestinal tract can be a nonspecific finding in sick hedgehogs, which can mimic acute obstruction*. Stressed hedgehogs may develop bright green diarrhoea.

Diarrhoea

Dietary indiscretion

Inappropriate foods (e.g. milk), new treats, fatty meat scraps, or some commercial diets can result in diarrhoea. Exposure to foods like the above will become apparent during the history taking (exactly the same as when we take the history for dogs with diarrhoea). The hedgehog will likely still be eating.

Stop the inappropriate food or change the brand of the commercial diet and supportive care should resolve the diarrhoea within a couple of days.

Endoparasites

Endoparasites are rare in pet hedgehogs. There is one case reported of a fatal cryptosporidiosis in a juvenile hedgehog. A faecal flotation and a wet mount microscopic examination of diarrhoea should be performed especially if the animal is a new addition.

Salmonellosis

Hedgehogs may be asymptomatic carriers of *Salmonella* and *should not be handled by immunosuppressed people* due to the zoonotic risk, and as carriers of *Salmonella* are intermittent shedders, *faecal culture cannot be relied upon to rule out carrier status* of an animal. Multiple cases of human salmonellosis have been recorded and 90% of the recent outbreaks in the US were directly in association with owning a pet African pygmy hedgehog. Hedgehogs have soft droppings and their tendency to walk through their faeces, may potentially lead to dissemination of *Salmonella* through the house if the animal is allowed to roam. Subclinical carriers of Salmonella *should not be treated as it will not eliminate the bacteria*.

Reported strains isolated are *S. typhimurium*, *S. tilene*, and *S. enterica* serovar Stanley. Predisposing factors include stressors such as poor ventilation, overcrowding, concurrent diseases, poor nutrition, or recent transport.

Clinical signs include anorexia, lethargy, mucoid green or bloody diarrhoea, dehydration, and weight loss.

Severe cases may present with septicaemia, multisystemic involvement, neurological signs, tachypnoea, and collapse. Intussusception may occur.

Salmonella commonly displays multidrug resistance to antimicrobials, both in human medicine and veterinary, so culture and sensitivity is strongly advised rather than empirical therapy.

Other bacteria can also cause diarrhoea, gastroenteritis, hepatitis, pancreatitis, and peritonitis, resulting in vomiting, anorexia, diarrhoea, abdominal discomfort, and potentially jaundice.

Diagnosis

Faecal culture and sensitivity (use fresh samples) is recommended in cases of diarrhoea. As *Salmonella* can be shed intermittently, a negative result does not exclude *Salmonella* as a causative bacterium. Although endoparasites are rare, faecal flotation and wet mounts may be examined microscopically.

Treatment

Mild cases may be self-limiting and probiotics alone may be all that is required. Supportive care through warmth, fluid therapy, analgesia (buprenorphine 0.05 mg/kg SC q48hrs) and assisted feeding can be given as needed.

Systemic antibiotics are indicated in potentially life-threatening cases, but *owners should be informed that it is well documented that treating with antibiotics results in prolonged faecal shedding of* Salmonella *and may induce carrier status, to the extent, some authors do not advocate treating with antibiotics at all*. If the decision to use antibiotics is made, treating for up to 3 weeks is recommended. Antibiotic choice should be guided by culture and sensitivity. Antibiotics which may be useful in confirmed cases include ampicillin, amoxicillin, fluoroquinolones, tetracyclines, erythromycin, clindamycin, streptomycin, and trimethoprim sulfamethoxazole (take care if used in dehydrated animals). Any therapy is likely to be required parentally until the gastrointestinal tract is functioning more normally.

Severely affected cases rarely will make a full recovery and serious consideration should be given to euthanasia on welfare grounds.

Owners and staff should be reminded of the potentially serious zoonotic risk. *Strict hygiene measures and barrier nursing any hedgehog with diarrhoea will minimise the risk*.

Alimentary *Candida albicans* was reported in a pet hedgehog causing weight loss, depression, and blood in the faeces.

Foreign body

Hedgehogs are inquisitive animals who may consume items that can cause an obstruction. *Items recorded to cause GI obstruction in hedgehogs include human hair, carpet fibres, and rubber items*.

Clinical signs are often nonspecific such as lethargy, anorexia, vomiting, and abdominal pain. Radiographs may

show the classical gas pattern associated with foreign body obstruction of the bowel, however, *severe gaseous dilatation of the gastrointestinal tract can be a nonspecific finding in sick hedgehogs, which can mimic acute obstruction. A foreign body may be found on palpation or an abdominal ultrasound may be used to confirm the obstruction*. Fluid therapy and warmth should be provided, and surgical intervention performed similar to the approach used in dogs or cats.

Neoplasia

GI neoplasia may present as chronic weight loss and anaemia. Alimentary lymphosarcoma is the most common neoplastic process in the gastrointestinal tract, with gastric carcinoma, small intestinal carcinoma, and colonic carcinomas being single case reports.

Seminal vesicle benign cystic hyperplasia in male hedgehogs may result in full or partial GI obstruction. Palpable as a caudal abdominal mass and may present with nonspecific signs such as reduced appetite, green mucoid faeces, and lethargy. Diagnosed is via an exploratory laparotomy and excision of the seminal vesicles should be curative.

Hepatic disorders

Hepatic lipidosis is a common finding, especially in sick hedgehogs, but may be subclinical in obese individuals. Clinical signs include lethargy, anorexia or reduced appetite, and jaundice (most easily visualised in the inguinal region). As hepatic lipidosis is usually secondary to another condition, unless jaundice is present the clinical signs of hepatic involvement may be masked by the primary condition, therefore consideration of hepatic lipidosis involvement should be given to any sick hedgehog. Cirrhosis of the liver is commonly reported in association with hepatic lipidosis.

Liver failure has been associated with neoplasia (common) and exposure to human herpes simplex virus 1 (cold sore virus), therefore owners with active cold sores should be discouraged from handling and kissing their pet until the lesion heals.

Skeletal conditions

Lameness

Hedgehogs should walk with their ventrum pulled up off the ground, but weak or wary hedgehogs crouch close to the ground while walking.

Common causes of lameness are ingrown nails, osteoarthritis, constriction injuries to foot, neoplasia, pododermatitis, neurological disease, intervertebral disc disease, or long bone fractures.

Osteoarthritis

Any species with a skeleton can develop osteoarthritis, species that actively hide pain mean that we have to actively look for skeletal pain in these species. Hedgehogs develop osteoarthritis and obesity will exacerbate it.

Limping or changes in gait may indicate osteoarthritis but may also indicate other conditions such as trauma, constriction injury of a foot, intervertebral disc disease, or neurological disease. Full clinical examination and diagnostics such as imaging and blood work early in the case will provide the best chance of a definitive diagnosis in these secretive patients, allowing a more accurate treatment plan and prognosis.

Intervertebral disc disease

Intervertebral disc disease (IVDD) is a progressive debilitating condition that is not uncommon in hedgehogs and clinical signs are similar to more familiar species, including lameness, proprioceptive deficits, progressive ataxia, and bladder atony with urine retention. *As clinical signs may resemble WHS it is vital that diagnostic imaging is performed*.

Most cases of IVDD are diagnosed in hedgehogs over 4 years of age, whereas most cases of WHS are diagnosed in hedgehogs less than 2 years old.

Radiography may show spondylosis, narrowed disc spaces, disc mineralisation, with frequently multiple discs affected. Most commonly its cervical or lumbar discs involved.

Raymond *et al*. (2009) reported that in the cases in the hedgehogs they looked at histologically there are features found in chondrodystrophic breed-associated disc disease in dogs.

Medical treatment options are similar to dogs with a combination of meloxicam 0.2–0.5 mg/kg q12–24hrs, gabapentin 5–20 mg/kg q8–12hrs, diazepam (as per dogs), and tramadol 5–15 mg/kg q6–12hrs. Prognosis is likely to be poor as the condition is often advanced at time of diagnosis and oral medication may be challenging in all but the tamest of patients.

Orthopaedics

Fractures

Long bone fractures are usually due trauma, either trapping a leg in a wire cage, or a spoked running wheel. *Splinting or surgical fixation is possible but must withstand the rolling up of the hedgehog*.

Amputation

Amputations are well tolerated by hedgehogs. The mantle should be pulled and restrained dorsally for surgery. Forequarter amputation should be performed when a forelimb is involved, and a high femoral amputation if a hindlimb is involved.

Euthanasia

End of life conversations can be a difficult area to broach with some clients. Occasionally owners will wait for us to

broach the topic as they are 'worried' if they do so we will think they are not dedicated enough. Reassuring clients that sometimes we need to love our pets enough to let them go when the time is right can be invaluable.

Many owners will want to stay with their pet during this procedure and the process should be fully explained including possible events such as sudden jumping, the need to change veins or gasping. I administer a pre-med (e.g. medetomidine/butorphanol or midazolam/ketamine (maintains venous pressure) injected SC or IM), once the hedgehog has fully relaxed, I will use the lateral saphenous vein by choice, to inject the pentobarbitone, to allow the owners to stroke the animals head. If the vein is not accessible, the pentobarbitone may be injected into the liver or kidney as the animal is already under a deep sedation. If the owner chooses not to stay with their pet, isoflurane may be used to induce anaesthesia and the pentobarbitone may be given by intracardiac injection.

Many owners will opt for private cremation with return of their pets' ashes and all options that your practice offers for dogs and cats should also be made available to your hedgehog clients. I offer footprints (coloured ink pad from craft shop and a card) and I will send sympathy cards if I know the patient and owner well.

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10 Ferrets

Ferrets (*Mustela putorius furo*) have been kept as pets since the 1980s in the US and Europe, and are increasing in popularity worldwide. They have been domesticated for approximately 3,000 years, being first described in classic literature as being kept in North Africa more than 1,000 years BCE (Bradley *et al*., 2006). I have been keeping pet ferrets in the UK for over 20 years.

Traditionally kept as a working animal (used for hunting rabbits, flushing prey for birds of prey, or killing rats) there are now more pet ferrets than working ferrets in the UK.

Ferrets are the most highly regulated of all the commonly kept exotic small mammals partially due to the risk of them becoming an invasive species and their potential as a vector for rabies. They are illegal to own in the US states of New York and California (despite being the third most common mammalian pet in the USA). Hunting with ferrets is illegal in some countries and states so readers are advised to check the local regulations.

Other uses of ferrets include as laboratory animals researching human influenza, endocrinology, and pharmacology to name a few, and for dragging cables for oil, electricity, and phone companies. A variety of coat colours have led to showing of ferrets and ferret racing can still be popular.

Contrary to popular belief, ferrets are inquisitive, playful, affectionate, highly intelligent (thought to have the same cognitive ability as dogs), and endearing pets. The average lifespan is 6–8 years but double figures have been recorded. Pet ferrets are relatively easy to keep once their husbandry requirements are understood. They are either asleep or in constant motion, which includes high levels of play that continues throughout life.

It should be noted that most drugs mentioned are unlicensed in ferrets and an off-licence consent form should be signed by the client prior to use in the UK. Readers outwith the UK are advised to check the regulations where they are based.

To establish the ideal housing and environmental enrichment for the domestic ferret I would usually examine the natural ecology of the species, however little is known of ferret behaviour outside of captivity. As a domesticated species our understanding of their natural behaviour mainly comes from studies of small feral ferret populations and from studying the wild ancestor, the European polecat (*Mustela putorius*).

Behaviour

When excited and playing they produce a vocalisation known as dooking. This is often referred to as a giggle by owners. They also perform a 'weasel war dance' throwing themselves randomly side to side and appear to be trying to 'catch invisible bad guys'.

Poole (1978) described the ferret play facial expression as an open mouth with all teeth covered by the upper lips (contrasted to aggression with the lips pulled back to bare the teeth). To someone unfamiliar with the species an animal randomly bouncing in all directions coming towards you with its mouth open and a sharp pointed face it is understandable why some interpret this behaviour as aggression (including my husband the first time he encountered it).

A 'bottle brush' tail indicates arousal, which can be fear or excitement. If they are frightened, they will hiss and express their anal glands. Ferrets cannot spray their anal gland secretion the way that skunks can. They will scream with pain or extreme fear.

Occasionally they will chatter as a greeting and some will wag their tails. Grooming of the owner is a common occurrence.

Companionship

They should be kept in minimum groups of two as Reijgwart *et al*. (2016) showed that conspecifics provided environmental enrichment, indeed, ferrets are renowned for continuing to participate in play behaviour with conspecifics into and throughout adulthood. *An absence of play behaviours in a normally playful species may indicate reduced welfare and may be due to illness, pain, stress, or unfulfilled needs*.

The *personalities of the ferrets should be observed to ensure compatible individuals are housed together*. Incompatible individuals housed together lead to chronic stress resulting in stress-induced gastric ulceration often complicated by *Helicobacter mustelae* infection, which is ubiquitous in ferrets. It should be remembered that the European polecat is a solitary species so although generally ferrets should be

kept with conspecifics, it is not surprising that *there will be individual ferrets who prefer solitary living*!

Enclosure

The home range size for European polecats is a massive 181 hectares. When feral ferrets, home ranges were studied in New Zealand there was a difference noted between the sexes with female ferrets having an average home range of 12.4 hectares (124,000 m^2) compared to the 31.4 hectares (314,000 m^2) for males.

It is therefore hardly surprising that the current recommended minimum cage size for 1–2 ferrets of 1.5–2 m^2 will result in behavioural problems unless environmental enrichment and regular exploration time outside of the enclosure are provided. Two of the commercially available ferret cages in the UK, a single level providing only 0.4 m^2 and a multilevel enclosure providing 1.21 m^2, do not even meet the recommended minimum enclosure sizes which is misleading for ferret owners and is a massive welfare concern. Owners should provide as large an enclosure as possible. My own ferrets are housed in a converted chicken coop that provides 5.02 m^2 floor space on the lowest level, multiple levels (which provide additional floor space), and climbing opportunities. *Ferrets are very sensitive to zinc toxicity so avoid using galvanised caging*.

Cold temperatures are tolerated well, especially if there are multiple ferrets and warm bedding material provided such as fleece blankets. Straw bedding provides warmth, but sharp pieces of straw may cause corneal damage. The optimal temperature range for ferrets is quite wide being 4–18°C (39–64°F). Ferrets will suffer from heat stroke if exposed to temperatures higher than 30°C (86°F). Provision of trays of water to allow water play and cooling, and plastic bottles of frozen water during hot weather are easy ways to prevent heat stroke.

Some owners allow their pets to free roam in the house. A litter tray should be provided for each room the ferret is allowed access to due to their fast gut transit time. However, it should be advised *that free roaming is fraught with danger* as 'ferret proofing' a house is exceedingly difficult. Unfortunately, cases such as ferret versus recliner or heatstroke after climbing inside a washing pile destined for the tumble drier are all too common.

Ferrets are relatively easy to litter train as they tend to have two favoured latrine sites, therefore the easiest method of litter training is to place the litter trays in their favoured spots. Large corner trays with higher sides on two edges are recommended due to the ferret's tendency to back into a corner while passing faeces. The recommended litter is wood based pelleted litter as it provides good odour control without being irritating to the ferret's sensitive respiratory tract. Frustratingly around 27% of ferrets will defecate outside of the litter tray at least once daily, which may be territory marking rather than a failure to litter train.

Ferrets live up to their Latin name *Mustela putorius furo*, which translates to weasel (or mouse eating) smelly thief. They will steal objects and hide them, occasionally chewing and swallowing parts of their hoard. They seem to specifically like chewing on rubber; be it a hairbrush handle or a toy which will sometimes result in a foreign body, so rubber or latex toy types should not be given. They also tend to hide and hoard food, so any cache sites should be regularly checked for decomposing food.

Hammocks should be provided for resting areas as they are highly valued by ferrets. Ideally it is recommended to provide as many hammocks as the number of ferrets kept in the enclosure, although in my experience ferrets all tend to pile into the same hammock and sleep on top of each other.

Environmental enrichment

It is known that low numbers of enrichment types alongside limited opportunity to explore outside of their enclosure coupled with restricted housing size can result in ferrets exhibiting abnormal behaviours and increased biting of conspecifics and owners. Freedom to exhibit normal behaviour is just as important in ferrets as any other species and *environmental enrichment should be considered critical to their wellbeing*. Cruden (2011) demonstrated that ferrets in barren cages showed stereotypical behaviour and became lethargic.

Any wellness examination for companion animals is not complete without discussion as to whether that animal's psychological needs are being met. Providing various enrichment items will easily elicit play behaviour. Environmental enrichment can easily be incorporated into any housing setting.

The five types of environmental enrichment reportedly most enjoyed by ferrets are tunnels, digging substrates, tactile interaction with owners, exploration of new areas, and boxes.

Tunnels including parts of drainpipes as well as bendable expanding pipes likely are enjoyed because they mimic burrows. Dig boxes filled with soil can be created out of storage boxes with a hole cut into the lid to limit the mess created (especially if the enclosure is indoors!). Ferrets are a burrowing species therefore provision of dig boxes should be strongly recommended.

Cat toys which they can carry and 'hunt' are appreciated.

Many ferrets easily accept harnesses for walking in. Owing to their necks being wider than their head, collars are difficult to keep on.

Diet

Ferrets are *obligate carnivores* with a short digestive tract and no caecum. Their intestinal length is only five times their body length (compared to the cat's intestinal length of approximately 8 to 10 times their body length); this means that *their digestion is relatively inefficient and relies on small, frequent meals of high protein and fat content*.

They have a meat protein requirement of 30–40% and a fat requirement of 15–20%, however lactating females

do require a higher fat content ideally at least 25%. Diets providing less than 30% animal protein will result in poor coat quality.

It is presumed that they require a dietary source of taurine as both dilated cardiomyopathy and retinal degeneration have been diagnosed in ferrets but it has not yet been definitively linked to taurine deficiency. As both these conditions have been linked to taurine deficiency in other obligate carnivores, it is reasonable to assume ferrets have a requirement until proven otherwise.

Ferrets rely heavily on their sense of smell when it comes to food choices. They develop an olfactory preference (or imprint) to food types within the first few months of age which means it *can be frustrating to change an adult ferret's food as some individuals will literally starve themselves if their preferred food type is unavailable*. It is for this reason that breeders are recommended to feed a variety of foodstuffs to ensure variable diet acceptance in future, e.g. if there is a shortage of the usual kibble.

With the development of ferret specific formulated diets this has increased the ease of feeding. However, it should be noted that the *quality varies between manufacturers* including the level of carbohydrates. Owners may need to try a few different types of kibble to see what works for their own ferret. Diets that are too high in plant proteins may predispose to the development of bladder stones so it is important to ensure the diet has a high animal protein source.

Ferrets generally will eat to calorific requirements and ad lib feeding is recommended.

Dietary environmental enrichment

Consider feeding kibble in a cat treat ball to provide environmental enrichment. This helps provide normal hunting behaviour, which ferrets are highly motivated to spend large portions of their active time doing.

Intermittent feeding of high-quality tinned or pouched kitten food along with a variety of meat-based snacks keeps dietary intake varied and interesting. Treats can be hidden in boxes or paper bags to encourage foraging behaviour. A boiled, cooled in shell egg can provide amusement while they break into the egg to consume. *Frequent raw eggs are not recommended as it can induce a thiamine deficiency*.

Change where in enclosure the food is offered or even offer a whole (dead) mouse occasionally. *Offering whole prey items is not without risk as they may be sources of harmful pathogens* such as *E. coli*, *Salmonella* spp., *Mycobacteria* spp. (if wild caught), or *Campylobacter*.

Nutritional secondary hyperparathyroidism (aka swimmers)

The calcium phosphorus ratio should be at least 1:1 and commercial ferret food generally provides between 1.2:1–1.7:1.

Unfortunately, inappropriate raw feeding has become increasingly popular among ferret owners, which can lead to nutritional secondary hyperparathyroidism and exposure to pathogens. *Many owners are completely unaware of the high levels of bacteria found in raw meat and the absolute requirement for an appropriate calcium phosphorus ratio*. Nutritional secondary hyperparathyroidism occurs when an all meat diet is fed with either no or insufficient calcium supplementation.

Owners may present kits and tell you their kit has developed 'swimmers', which is a term breeders use for 5–6 week old kits that crawl on their stomachs (they cannot support their own weight) while their legs splay outwards giving the appearance of swimming. The kits may be lethargic. *Kits are normally extremely active and should be well up on their feet running by 5–6 weeks of age*. The muscles of the ferret may be stronger than its bones causing bending or deformity of the spine, legs, or rib cage. Folding fractures are common and the kits will be painful. The jill should be examined also.

Diagnosis is easily achieved by radiography demonstrating poor mineralisation of the skeleton, in many cases the teeth are more radio dense than any other part of the skeleton. Folding fractures of the spine and long bones are commonly seen. Milder cases may show bending of the long bones instead of folding fractures (Figure 10.1).

Treatment is dependent on the severity of the condition. Many of these kits will require euthanasia. In milder cases analgesia and appropriate feeding may be all that is required although owners should be counselled that kits with long bone deformities are likely to have altered gaits which may lead to degenerative joint disease formation at a younger age.

This is a *completely avoidable problem and has been caused by inadequate diet and my opinion is this is unnecessary suffering*. This condition will not occur if commercial ferret food is fed.

Water

Fresh clean water should always be available, their water intake is 60 ml/kg/day on average. Ferrets love playing in their water so multiple sources of fresh water should be made available.

Sexing

Ferrets are sexually dimorphic and can be readily sexed.

Female

Female ferrets are called jills or bitches. The vulva is situated directly below the anus. Female ferrets are generally much smaller than males.

Male

Male ferrets are called hobs or dogs. The scrotal sac containing the testicles is situated directly below the anus, the

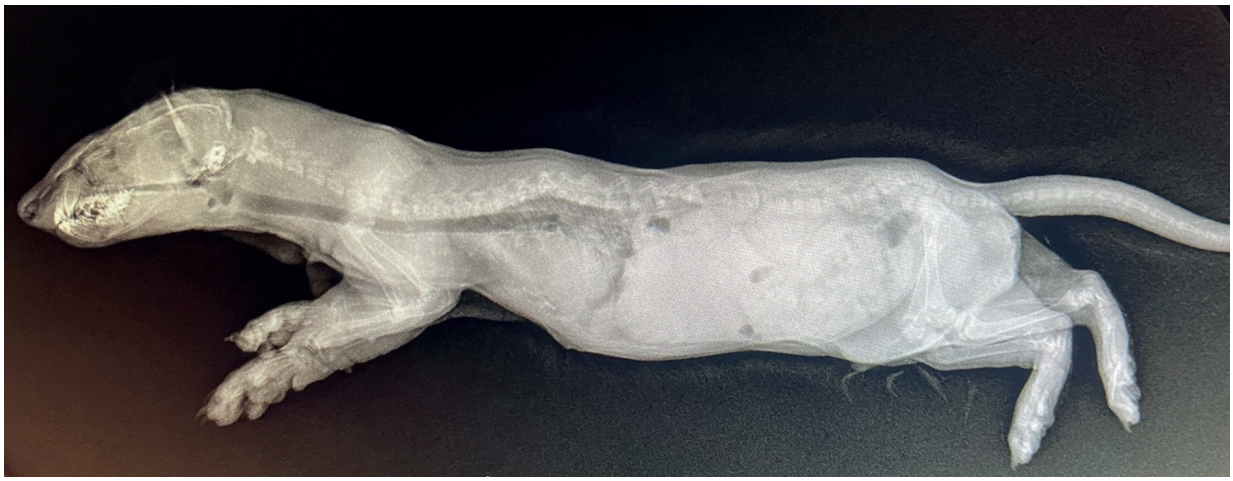


Figure 10.1 Radiograph of ferret kit with 'swimmers'. There is a folding fracture in the thoracic spine.

prepuce is found approximately a third of the way along the ventral abdomen (much like a dog).

Both sexes have nipples.

Preventative health care

Vaccination

Vaccine reactions

The quoted risk of vaccine reaction from a retrospective study of 3,587 ferrets who received rabies or distemper vaccinations by Moore *et al*. (2005) is 0.51% for rabies, 1% for distemper, and 0.85% if both vaccinations are given on the same day. They reached the conclusion that the risk of vaccine reaction increased with the number of distemper vaccinations that were administered. *It is recommended to give distemper and rabies vaccinations separately with 2–4 weeks between vaccines*.

The most common adverse events that are seen were hypersensitivity reactions manifested as vomiting and diarrhoea. An early sign of an anaphylactic vaccine reaction is significant piloerection of the hair of the body and tail along with generalised hyperaemia of the skin. Other type 1 hypersensitivity reactions seen include drooling, restlessness, respiratory distress, bleeding, collapse, and, rarely, death.

As type 1 hypersensitivity reactions tend to occur within 5–25 minutes of administering the vaccine, *it is recommended that owners should wait in the practice for 30 minutes post-vaccination*.

Treatment should be instigated at the first sign of a reaction, exact treatment required depends on the severity of the reaction. Diphenhydramine 0.5–2 mg/kg SC, IM, or PO should be given, and owners should be advised that drowsiness may be seen with diphenhydramine. Vomiting may be controlled with metoclopramide 0.5–1 mg/kg SC, IM, or PO. Epinephrine 0.02 mg/kg SC, IM, or IV should be given in cases of severe reaction. Intravenous (IV) fluids are administered at 10 ml/kg/hour to counteract shock. Some cases may require oxygen and thermal support.

Premedication with diphenhydramine PO 30 minutes prior to vaccination may decrease the risk of reaction. Despite regularly vaccinating ferrets against distemper in the UK, thankfully I have never seen a vaccine reaction in ferrets.

Distemper vaccination

Ferrets are extremely susceptible to canine distemper virus which causes virtually a 100% mortality. There is no licensed vaccination in the UK. In the USA Purevax Ferret Distemper vaccine, Merial is the approved vaccine. When Purevax Ferret was unavailable, Nobivac DPv was used with no serious side effects noted (Wade, 2018). *Any working ferret, ferret that attends shows, or a ferret that is walked should be vaccinated against distemper*.

I currently use Virbac Canigen DHPPi, a half dose given twice, 2–4 weeks apart from 10 weeks of age. I have also used Nobivac DHPPi, MSD Animal Health. Ferrets are so susceptible to distemper that administration of the wrong canine distemper vaccine may cause clinical distemper in the ferret, so before another brand is given the manufacturer should be consulted.

In the US, the American Ferret Association recommends that vaccines should be given at 8 weeks old then repeated at 11 weeks and again at 14 weeks of age followed by annual boosters. If the ferret is presented older than 14 weeks, then two doses should be given 3 weeks apart followed by annual boosters.

Although yearly boosters have frequently been recommended, Wagner (2012) demonstrated that *protective titre levels were maintained for over three years post-vaccination in at least 95% of ferrets and for over four years in 90% of ferrets*. Titre levels can be measured to assess whether repeat vaccination is required with titre levels over 1:50 are considered protective in ferrets.

Therefore, booster vaccinations should be given every 1–3 years depending on risk assessment and titre levels. Currently I vaccinate my own ferrets every 3 years.

Rabies vaccination

Rabies is almost invariably fatal in ferrets and is zoonotic. There are licensed rabies vaccinations for ferrets in the UK and USA. The initial vaccine can be given from 12 weeks of age with boosters required annually. Ferrets are considered immunised 28 days post-primary vaccination.

In the UK rabies vaccination is only given if the ferret is travelling abroad.

In the USA the requirement for ferrets to be rabies vaccinated may vary between states but owners should be advised that should a ferret unvaccinated for rabies bite someone, the Centers for Disease Control and Prevention may require the ferret to be quarantined for observation or order mandatory euthanasia and testing for rabies. Government regulations may mean that even rabies vaccinated ferrets may be sacrificed and tested for rabies should they bite a human. *Readers are advised to check local regulations*.

Ectoparasitic control

Ferrets should be risk assessed for ectoparasite control. Working ferrets are more at risk of being exposed to fleas and ticks than ferrets who never leave home. Ferrets can develop flea allergic dermatitis and an argument can be made for year-round preventative treatment for individuals who are allergic to fleas. See Dermatology section for recommended treatments.

Heartworm prevention

Ferrets have a small heart in comparison to dogs and cats, which means that one or two adult worms may be enough to cause clinical disease (see Heartworm section). Ferrets that live in a heartworm endemic area should be placed on a preventative treatment protocol regardless of whether they are housed indoors or outdoors. Housing ferrets indoors during mosquito season may help limit exposure.

Preventative therapy should be started one month before the start of the expected heartworm season and continued on until 1 month after the end of the heartworm season. Options for prevention include: ivermectin 0.05–0.1 mg/kg q30days SC or PO, selamectin 18 mg/kg q30days topically, or milbemycin oxime 1.15–2.33 mg/kg q30days PO.

Neutering

Ovariohysterectomy

Ovariohysterectomy is only advised if there is a medical reason, such as ovarian tumour, uterine mass, or pyometra, due to the association between surgical neutering and the development of adrenal gland disease. Increasing day length is the trigger for sex hormone production in ferrets. Simply put, longer day length triggers GnRH release from the brain, which goes to both the adrenal glands and the gonads. When the levels of sex hormones are at the required level, the gonads provide negative feedback to the brain that then results in GnRH no longer being produced and cessation of sex hormone release. In surgically neutered ferrets the gonads have been removed therefore there is no negative feedback possible. Continuous stimulation of the adrenal gland over years results in the area of the adrenal gland responsible for production of sex hormones becoming bigger (I tell clients it is a bit like lifting weights, do it often enough your muscles get bigger) until eventually the levels of sex hormones produced are high enough to produce clinical signs. In implanted ferrets the signal for the production of sex hormones is prevented therefore the gonads shrink and the adrenal glands do not become stimulated.

Owners who have heard of entire ferrets developing adrenal gland disease will sometimes use that as a fact disproving the link between neutering and development of adrenal gland disease. My response to those clients is simply this: any organ can develop a tumour, sometimes those tumours are functional and functional means producing hormones that have a clinical effect. *The point is not that entire ferrets may rarely develop adrenal gland disease, but that by surgically neutering ferrets we are highly likely to induce adrenal gland disease*. Figures 10.2a–c show a diagrammatic explanation of the theory behind the link between neutering and adrenal gland disease (reproductive medicine section for more on adrenal gland disease).

The surgical technique is similar to in the bitch with one notable exception. The ovary is encapsulated in a fatty bursa which completely encloses the ovary in the ferret. During surgical neutering it is *important not to enter the bursa* to prevent leaving part of the ovary behind. Ovarian remnant syndrome is a common consequence of opening the ovarian bursa.

Castration

Surgical castration is only advised if there is a medical reason such as testicular tumours or cryptorchidism due to the association between surgical neutering and the development of adrenal gland disease (reproductive medicine section). The technique is the same as in dogs.

Vasectomy

See the reproductive medicine section for the technique.

Hospitalisation

Ferrets are predators and should be hospitalised away from prey species as even the smell of ferrets elicit fear in prey species. Personally, I hospitalise ferrets in my cat ward.

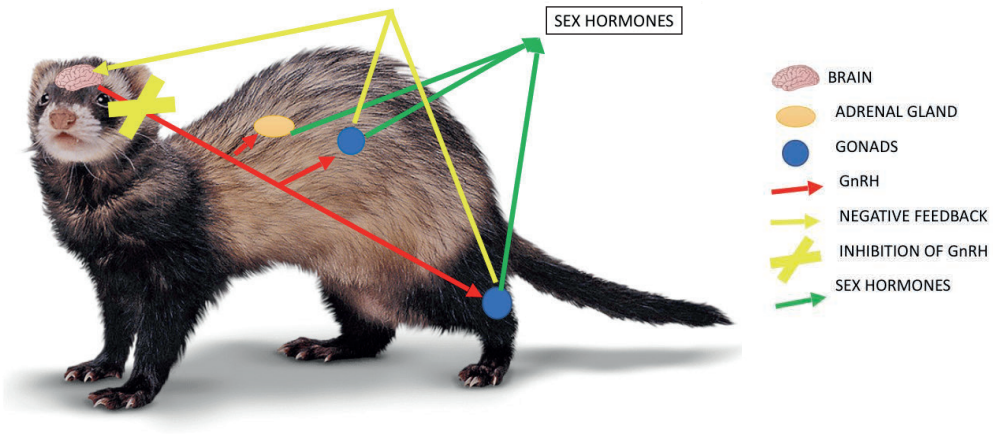


Figure 10.2a Longer day length triggers GnRH release from the brain which goes to both the adrenal glands and the gonads. When the levels of sex hormones are at the required level, the gonads provide negative feedback to the brain which then results in GnRH no longer being produced and cessation of sex hormone release.

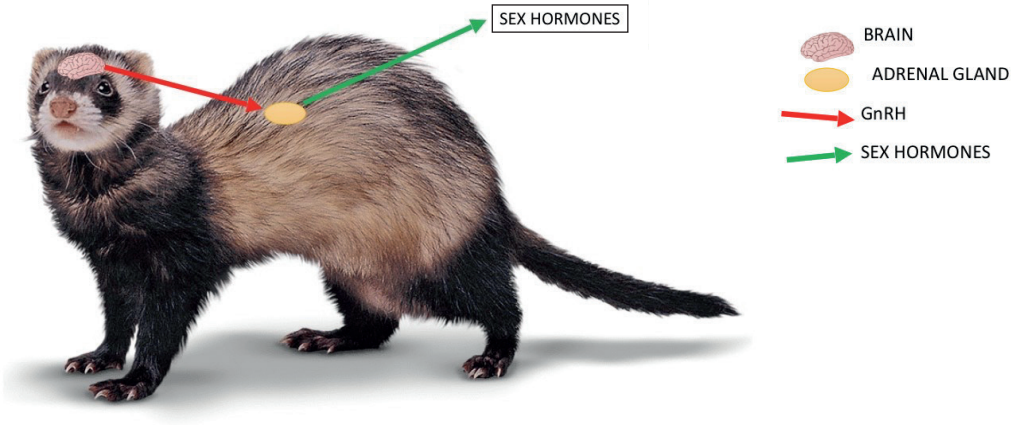


Figure 10.2b In surgically neutered ferrets the gonads have been removed therefore there is no negative feedback possible. Continuous stimulation of the adrenal gland over years results in the area of the adrenal gland responsible for production of sex hormones becoming bigger until eventually the levels of sex hormones produced are high enough to produce clinical signs.



Figure 10.2c In implanted ferrets the signal for the production of sex hormones is prevented therefore the gonads shrink and the adrenal glands do not become stimulated.

Ferrets are renowned escape artists and the small size of most jills means they may be able to squeeze between the bars of the hospital kennel. Cable tying Perspex to the front of the kennel may be required to keep determined jills in.

Litter trays with pelleted wood litter should be provided in a corner and a blanket or towel can be given as bedding. Requesting the owner brings in the ferret's favoured blanket and their bonded cage mate (if they have one) will help reduce the stress of hospitalisation. Ensure the ferret has short nails to prevent the nails becoming caught on the bedding. Many ferrets will choose to sleep in their litter tray when in the clinic.

Ferrets are used as respiratory models for human research. They are susceptible to influenza virus and COVID-19 therefore *any staff who have severe cold like symptoms should ideally not work with the ferret, or gown, glove, and mask up as there is a real risk they could infect the ferret.*

Venipuncture sites

Venipuncture can easily be performed in the conscious ferret using the cephalic, jugular, or lateral saphenous veins (Figures 10.3–10.5). The jugular should be used for large samples, but I regularly obtain 2 ml from the cephalic vein. If you are presented with a fractious ferret or are unused to sampling ferrets then *mask induction with isoflurane could be considered, which would also allow full body radiography to be*



Figure 10.4 Lateral saphenous vein.



Figure 10.3 Jugular vein blood sample.



Figure 10.5 Cephalic vein blood sample.

performed. This will decrease blood parameters such as packed cell volume, haemoglobin, white blood cell counts and plasma protein levels. Owing to these factors, a conscious blood sample should be attempted.

Maximum recommended safe volumes are estimated as 1% of the lean body mass in healthy individuals, reducing to 0.5% of the lean body mass in sick animals. So even in an ill 600 g jill that equates to 3 ml. Full biochemistry and haematology can be run on in-house analysers or can be sent to an external laboratory. Handheld glucometers should be on the dog setting and the toe paw pads can be used to gain a drop of blood via lancet needle.

Drugs contraindicated in ferrets

Benzocaine induced methemoglobinemia occurred in ferrets that had their larynx sprayed with benzocaine to facilitate endotracheal intubation. If the methemoglobinemia is of a high enough concentration, there will be decreased oxygen carrying capacity of the erythrocytes. This did not occur when lidocaine was used and so benzocaine should not be used. Paracetamol should not be given to ferrets. Ibuprofen should not be given to ferrets.

Species-specific anatomy/behaviour

- Most ferrets have a diminished or absent menace reflex.
- There is a well-developed retrobulbar venous sinus.
- Well-developed masticatory muscle which gives a strong bite.
- The entire oesophagus of the ferret is striated muscle.
- Ferrets are spontaneous secretors of hydrochloric acid.
- Ferrets paw frantically at their mouths when they feel nauseous.
- Unsophisticated gastrointestinal microbiome.
- High proteolytic activity but ferrets have lower numbers of other digestive enzymes compared to other species.
- The whole intestinal tract can secrete cholecystokinin, the hormone responsible for bile secretion.
- Jejunum and ileal segments of the small intestine are indistinguishable and may be referred to as the jejunoileum.
- Foreign bodies should be considered an emergency as the intestine is prone to rupture and the foreign body will rarely pass naturally.
- Ferrets gain up to 40% extra body weight during the winter and lose the weight the following spring. If the photoperiod does not vary seasonally (e.g. the ferret is housed indoors and exposed to artificial lighting) the natural physiologic change in weight may not occur.
- Ferret necks are wider than their heads so it is not possible to maintain an Elizabethan collar in place.
- Healthy ferrets often have a high BUN.
- Manipulation of the neck during intubation causes stimulation of the vagus nerve and an increase in intestinal motility (may increase risk of rupture in cases of intestinal foreign body).
- Jills are induced ovulators and will stay in oestrus until they either ovulate, are exposed to shorter day length, or die.
- The ovary is encapsulated in a fatty bursa which completely encloses the ovary.

When compounding medication for ferrets it is important to know that some ferrets will prefer sweet fruit flavours to meat flavoured medications.

Pain in ferrets

Despite being a predator and domesticated, ferrets are incredibly stoical and very good at hiding pain. *Their ancestors' innate solitary lifestyle has been suggested as a reason for not having obvious pain displays*.

Species-specific behaviours that can be used as indicators of nonspecific pain in ferrets include: general malaise, anorexia/decreased appetite, lethargy, depression, inactivity (or lack of exploration of new areas), staying rolled up in a ball or reluctance to curl into a ball (which is the normal sleeping position for ferrets), failure to groom normally, and increased depth and frequency of breaths.

Reijgwart *et al*. (2017) developed the ferret grimace scale (https://journals.plos.org/plosone/article/figure?id=10.1371/journal.pone.0187986.g003). *Orbital tightening (squinting of the eyes) was shown to have high sensitivity, specificity, and accuracy for detection of pain*. The other facial features had little to no extra value for pain determination.

Different types of pain may show in different ways, just as in more familiar species.

Dental pain may result in tooth grinding and a decrease in food and water intake. Ferrets with tooth pulp inflammation flick their tongue/have tongue protrusions aimed towards the affected side.

Otitis media/interna may cause pain radiating to the temporomandibular joint (TMJ) resulting in difficulty eating or reluctance opening their mouths.

Lameness or stiff gait may indicate skeletal pain, but a stiff stilted gait may also be seen with abdominal pain. Other indicators of abdominal pain include walking with a hunched, arched back, immobility, and teeth grinding when presented with food.

Osteoarthritis in ferrets

Johnson-Delaney (2020) states that ferrets become geriatric at 3 years of age and that is the age where conditions associated with older age become commonly diagnosed.

The gait of the ferret should be observed as the loss of the normal 'hump' in the back can indicate generalised weakness, pain, or hind limb ataxia.

A full skeletal pain examination and range of movement exams should be performed as per other species. Spinal, hip, and stifle osteoarthritis is commonly seen in ferrets. Radiography can be utilised to investigate osteoarthritis or spinal pain in exactly the same way as more familiar species.

Joint supplements such as chondroitin sulphate and glucosamine have been shown to attenuate pain associated with osteoarthritis and promote cartilage regeneration in other species but no information is yet available in ferrets. Joint supplements that are developed for cats could be offered to ferrets.

As well as multimodal analgesia, some husbandry changes can be considered. Lowering the side of litter trays to allow easier access by the ferret is advisable. Providing thicker bedding to help with warmth and joint support is often appreciated. If climbing becomes difficult for the ferret, then housing in a single level enclosure should be considered and will stop the risk of falls. The owner should be advised that the ferret will still require a large floor space and daily access to time outside of the enclosure. Ferrets play at all ages, but older ferrets tend to play for shorter periods.

Intervertebral disc disease

Intervertebral disc disease (IVDD) is occasionally seen in ferrets but not as often as you may think given their long backs. The wild ancestor of ferrets is the European polecat, who evolved to have this elongated spine, without human interference. Given that the shape of the ferret has obviously been an evolutionary advantage (most mustelids have a similar build) it is not surprising that IVDD is not as common in ferrets as it is in some breeds of dog.

Clinical signs

Several cases have been reported with ages varying from 7 months to 6 years of age. Clinical signs are dependent on the area of the spine affected and full spinal examination along with examinations of the reflexes should be performed in the same manner as more familiar species. Although no consistent cause has been identified, both trauma and discospondylitis have been seen.

Diagnosis

Radiographs may show narrowing of disc spaces, sclerosis, or osteomyelitis of vertebral end plates. Advanced imaging may be required for definitive diagnosis in some cases.

Table 10.1 Ferret analgesia.

Analgesic	Dose
Meloxicam	0.2 mg/kg q24hrs PO SC, females may require more frequent dosing
Carprofen	1–5 mg/kg q12–24hrs
Buprenorphine	0.04–0.06 mg/kg q4–6hrs OTM
Butorphanol	0.3 mg/kg q2–4 hrs SC
Gabapentin	3–10 mg/kg q8–12hrs PO
Methadone	0.3–0.6 mg/kg SC q4–6hrs dependant on pain scoring, repeated doses should be given at a lower dose
Tramadol	5–10mg/kg q12–24hrs
Lidocaine	1 mg/kg (maximum 2 mg/kg)
Bupivacaine	1 mg/kg (maximum 1.5 mg/kg) (may last up to 4 days)
Amantadine	3–5 mg/kg q24hrs PO
Acupuncture	

Treatment options are dependent on actual diagnosis but will be similar to more familiar species.

Analgesia in ferrets

Consideration of the type of pain we are looking to control will lead to logical choices of the type of analgesics we select.

Multimodal analgesia is recommended just as in more familiar species. I manage owners' expectations by explaining that 'pain winddown' can take 8–12 weeks in chronic pain cases but that I reassess after 2 weeks.

Oral or parenteral vitamin B has been shown to reduce inflammatory and neuropathic pain in many species and can be used as part of the multimodal analgesic approach at a dose of 1–2 mg/kg q24hrs.

My preferred non-steroidal anti-inflammatory (NSAID) is meloxicam. One study showed that meloxicam SC at 0.2 mg/kg q24hrs achieved blood levels that were considered to provide analgesia in other species, however this study did not assess analgesic efficacy in ferrets and noted differences between male and female ferrets. Female ferrets did not achieve blood levels of meloxicam as high as the male ferrets did and also had faster elimination of the drug than males. More work is required to assess efficacy and if female ferrets require a higher dose or more frequent dosing than males. Currently I use 0.2 mg/kg PO, SC q24hrs in males and females. Just as with more familiar species I utilise meloxicam in many types of pain.

Carprofen has been used in ferrets with clinical reports stating dose range of 1–5 mg/kg q12–24hrs. Some authors advise the concurrent use of H2 receptor antagonists such

as famotidine. There have been no studies performed on the use of carprofen in ferrets.

Some texts will list a dose for ibuprofen for use in ferrets. However, *as ferrets are especially sensitive to the toxic effects of ibuprofen and the narrow safety margin, myself along with other authors (Petritz & de Matos, 2023) do not recommend the use of ibuprofen in ferrets*.

Buprenorphine 0.04–0.06 mg/kg q4–6 hrs can be given by the oral transmucosal (OTM) route. Only the single use vials can be used in this manner as the multidose bottle contains preservative which is reportedly foul tasting. One study (Katzenbach *et al.*, 2018) showed that 0.04 mg/kg provided blood levels consistent with analgesia in other species for 4–6 h when given IM. SC administration can be used.

Katzenbach *et al.* (2018) also looked at butorphanol and recommended dosing at 0.3 mg/kg q2–4hrs SC but the actual dosing frequency required is still unknown.

In my experience tramadol works extremely well for various types of moderate pain. Liquid tramadol is often more cost effective when obtained from a compounding company and compounding companies may be able add flavouring to make it more palatable. In my experience ferrets take tablets more easily than liquids but owners may need to experiment to see what works for their ferret. Many compounding companies can make tramadol tablets at the required dose with palatable flavour. I use 5–10 mg/kg q12–24 hrs. *Ferrets are more susceptible to the sedative effects of tramadol*.

Morphine can cause severe sedation, respiratory depression, drooling, nausea, vomiting, and agitation in ferrets. Reportedly administering antiemetics prior to administration of morphine reduces vomiting but *I personally do not use morphine in ferrets*.

Methadone use has not been reported in ferrets but should be considered in cases of severe pain as it is associated with less gastrointestinal side effects compared to morphine in dogs. I will start at 0.3 mg/kg and increase up to a maximum of 0.6 mg/kg, q4–6 hrs with further dosing dependent on pain levels. Repeat doses may be given at lower doses due to accumulation, but pain scoring should be repeated to ensure any lower dose provides adequate analgesia. Ferrets are more reportedly more sensitive to the sedative and respiratory depressant effects of opiates and should be closely monitored. If concerned the effects can be reversed by administration of naloxone or butorphanol.

Gabapentin 3–10 mg/kg q8hrs works well for neurogenic pain and is my go-to analgesic if I am suspicious of disc disease. *With ferrets I will start at the lower dose as they are potentially more sensitive to the sedative effects and explain to the owners that we may not achieve full analgesia at the lower dose*. The dose may need adjusted up if the patient has been taking the medication for a long time. This further highlights the importance of regular pain scoring.



Figure 10.6 Acupuncture for osteoarthritis. Photo: Dr Krissy Green.

Amantadine is an antiviral which works on the NMDA receptors and provides analgesia as part of a multimodal approach. The current dose recommended is 3–5 mg/kg PO q24hrs.

Acupuncture has shown great success in alleviating chronic pain as part of a multi-modal approach (Figure 10.6).

Local anaesthetics may be used perioperatively as part of a multimodal analgesic approach. The toxic doses and effects of lidocaine and bupivacaine are similar to those seen in dogs and cats. Signs of toxicity include vomiting, arrhythmias, hypotension, depression, muscle tremors, ataxia, and nystagmus. Bupivacaine may provide some effect for up to 4 days (Johnson *et al.*, 2011). Recommended maximum doses are: bupivacaine 1.5 mg/kg; lidocaine 2 mg/kg, although 1 mg/kg will provide local anaesthesia.

Clinical exam

Ferrets have relatively poor eyesight and removing a startled ferret from a carrier could result in a nip. *Owing to their curious nature, it is advisable to open the carrier and allow the ferret to amble out*. The gait of the ferret should be observed as the loss of the normal 'hump' in the back can indicate generalised weakness, pain, or hind limb ataxia. The ferret can then be lifted by grasping around the shoulders for the clinical exam.

A full clinical exam should be performed, utilising the same skills as in more familiar species, such as dogs or cats.

Table 10.2 Ferret biological data.

Parameter	Reference range
Average longevity	6–8 years
Respiratory rate	33–36 bpm
Heart rate	200–250 bpm
Body temperature	37.8–40°C (100.4–104°F)
Maintenance fluid therapy	60–100 ml/kg/day
Sexual maturity	First spring after birth
Gestation	42 days

Table 10.3 Antibiotics commonly used in ferrets.

Drug	Dose
Amoxicillin	30 mg/kg PO q8hrs
Amoxicillin/clavulanic acid	25 mg/kg PO q8–12hrs
Azithromycin	5 mg/kg PO q24hrs
Cefovecin	8 mg/kg SC q2–3days
Cephalexin	15–30 mg/kg PO q8–12hrs
Ciprofloxacin	10–30 mg/kg PO q24hrs
Chloramphenicol	50 mg/kg SC, IM, IV, PO q12hrs
Clarithromycin	12.5 mg/kg PO q12hrs
Clindamycin	6–10mg/kg PO q12hrs
Doxycycline	10 mg/kg PO q12hrs (no dairy) q12hrs
Enrofloxacin	10 mg/kg PO SC q12hrs
Erythromycin	10 mg/kg PO q6hrs
Gentamicin	2 mg/kg PO q12hrs
Marbofloxacin	5 mg/kg PO q12–24hrs
Metronidazole	15–20 mg/kg PO q12hrs
Oxytetracycline	20 mg/kg PO q8hrs
Silver sulfadiazine cream	Topical q12–24hrs
Trimethoprim sulfamethoxazole	30 mg/kg PO q12hrs
Tylosin	5–10 mg/kg PO, SC q12hrs

Personally, I find it easier to perform a clinical examination with the ferret in my arms (Figure 10.7). Ferrets have delicate pinnae and their earwax is normally a dark brown colour. Ear mites (*Otodectes cynotis*) are frequently missed as even large infestations rarely cause any irritation.

The teeth can be examined by supporting the ferret along your forearm and using the other hand to lift the lips. Alternatively, scruffing the ferret will cause the vast majority of ferrets to automatically yawn (*this is a stress response so should be a last resort*) allowing a full albeit quick look in the mouth. Dental disease is common in pet ferrets and treatment is the same as in dogs and cats (see section on dental disease).



Figure 10.7 Examining a ferret while held.

When auscultating the thorax, it is important to be aware that *the ferret's heart is situated more caudally than in other species* and can be located at the 8th rib rather than medial to the elbow with dogs or cats (Figure 10.8). *Thoracic compliance should always be checked* by laterally compressing both midway along the thorax and the costal arch. Reduced thoracic compliance always requires investigation. The femoral pulse quality and rate is more easily palpated with the ferret resting on your forearm.

A full skeletal pain check should be performed. Ferrets are not as prone to spinal disease as might be expected given their long backs (*the difference being they evolved to be this shape, we did not artificially select for it*) but they can develop disc disease and spinal osteoarthritis.

The abdomen should be palpated, and hydration status can be assessed using the same parameters as dogs or cats. Normal ferret faeces are soft but formed (Figure 10.9). Ferrets with anorexia will often have scant green tinged faeces due to the fact that the whole intestinal tract can secrete cholecystokinin, the hormone responsible for bile secretion.



Figure 10.8 The heart is positioned much more caudally in ferrets (pinkie finger at elbow, pointer finger over heart).



Figure 10.9 Normal ferret faeces are soft but formed.

Dehydration

Mild dehydration can be corrected orally using a rehydration formula; however, many ferrets may not find these formulas palatable. Alternatively, a mix of highly palatable soft food such as Hill's Prescription Diet™ a/d Canine/Feline with warm water will often be readily consumed by ferrets.

Moderate or severe dehydration will require intravenous fluid therapy. Maintenance fluid requirement is considered to be 60–100 ml/kg/day. In hobs, a 22G IV catheter can easily be placed in the cephalic vein, while jills may require a smaller gauge. *The catheter gauge should be judged on vein size rather than the size of the animal*. Application of a topical eutectic mixture of local anaesthetics (EMLA 5% cream, 2.5% lidocaine, 2.5% prilocaine, Aspen) may make placement easier. As ferrets' necks are wider than their head it is not feasible to maintain a Buster collar in place, *but most ferrets will leave their bandages alone*.

Intraosseous catheters can also be utilised for fluid therapy and can be placed as for puppies and kittens. *It is my opinion that subcutaneous fluids should not be given to dehydrated ferrets due to their prolonged absorption times and pain associated with administration*.

Assisted feeding

The term assisted feeding is preferable to force feeding when discussing with owners. Supportive feeding should be calculated at a minimum of 400 kcal/kg body weight and split into four to six meals daily. Oxbow Carnivore Care or EmerAid Carnivore are two palatable options that many ferrets will readily take.

Gastric protectants

Ferrets are prone to stress-induced gastric ulcers. When planning elective procedures consideration should be given to a short course of famotidine 2.5 mg per ferret q24hrs preoperatively and continuing post-operatively.

Bismuth subsalicylate (Pepto-Bismol® 17.5 mg/kg q12hrs) has been shown to reduce the ability of *Helicobacter mustelae* to adhere to damaged mucosa, but recently it has been difficult to source in the UK.

Sucralfate (75–125 mg/kg q8–12hrs) must be administered 30 minutes before food or famotidine as sucralfate needs acidic pH to work. In the UK sucralfate can be easily sourced from compounding companies. I obtain sucralfate from Summit.

General anaesthesia

Ferrets can vomit so should be starved before surgery. Their gut transit time is approximately 2.5–3.5 h; as such, they should not be starved for as long as dogs and cats before surgery, as this holds a real risk of inducing hypoglycaemia, especially in older ferrets where subclinical insulinoma is common. *The recommended time for starving before surgery is a maximum of four hours*. I usually request owners remove food at 7am if I am aiming to operate at 10am.

Pre-anaesthetic blood tests

The need for these is debated among vets. Personally, I advise they are an option but acknowledge in the young healthy individual the vast majority will be within reference ranges. *The benefit is that it will give you a personalised reference range for this individual patient and as ferrets are good*

at hiding disease, occasionally we get unexpected results which may alter our plans.

Blood can be taken from the cephalic vein (my vein of choice for bloods or catheter placement) or the lateral saphenous vein. The jugular can also be used, with the ferret restrained like a cat with the front legs held extended down at the edge of a table and the head held dorsally. *The jugular is more lateral in a ferret compared to a cat* (Figure 10.2). There is nothing wrong with aseptically dripping the blood from a needle into the collection tube. Biochemistry can be run 'in-house' using the same pre-op profiles used for dogs and cats. (See Tables 10.4–10.6 for reference ranges.)

Ideally blood samples should be taken from a conscious ferret as anaesthesia will decrease blood parameters such as packed cell volume, haemoglobin, white blood cell counts and plasma protein levels (see Venipuncture section).

Pre-emptive analgesia

Just as in more familiar species, pre-emptive analgesia is important to prevent central sensitisation to pain, provide a more stable anaesthetic, ensure good welfare, and reduce incidence of wound interference.

Premedicant sedation is also an opportunity to provide pre-emptive analgesia depending on agents chosen.

Table 10.4 Ferret biochemistry.

BIOCHEMISTRY (Hein *et al.*, 2012)	REFERENCE RANGE (UK units)	REFERENCE RANGE (US units)
ALKP	13.3–141.6 IU/l	13.3–141.6 U/l
ALT	49.0–242.8 IU/l	49.0–242.8 U/l
AST	40.1–142.7 IU/l	40.1–142.7 U/l
AMYL	19.4–61.9 IU/l	19.4–61.9 U/l
GLDH	0.0–2.5 IU/l	0.0–2.5 U/l
GGT	0.2–14.0 IU/l	0.2–14.0 U/l
LDH	154.4–1780.6 IU/l	154.4–1780.6 U/l
CK	94.0–730.9 IU/l	94.0–730.9 U/l
LIPASE	73.2–351.1 IU/l	73.2–351.1 U/l
GLU*	3.5–8.5 mmol/l	63–153 mg/dl
CHOL	2.4–7.1 mmol/l	92.8–274.56 mg/dl
TRIGYLCERIDES	0.5–2.8 mmol/l	44.25–247.8 mg/dl
BA	0.0–28.9 µmol/l	0.0–28.9 µmol/l
TBIL	0.0–3.3 µmol/l	0–0.19 mg/dl
UREA	4.8–16.9 mmol/l	28.83–101.5 mg/dl
CREA	23.0–76.7 µmol/l	0.26–0.87 mg/dl
TP	54.7–77.9 g/l	5.47–7.79 g/dl
ALB	28.0–43.9 g/l	2.8–4.39 g/dl
CA	2.0–2.6 mmol/l	8–10.42 mg/dl
PHOS	1.0–3.1 mmol/l	3.1–9.6 mg/dl
K	3.9–5.9 mmol/l	3.9–5.9 mEq/l
Na	140.1–169.7 mmol/l	140.1–169.7 mEq/l
FRUCTOSAMINE	121.1–201.6 µmol/l	
CORTISOL	0.0–101.5 nmol/l	
THYROXINE	15.9–42.0 nmol/l	
PROGESTERONE	0.0–0.4 ng/ml	
OESTRADIOL	0.0–12.2 pg/ml	

* Hein *et al.* (2012) noted that the lower end of the reference range for glucose was lower than expected compared to laboratory ferrets and acknowledged that the animals were fasted and early insulinoma could not be ruled out in the pet ferrets used in this study.

Table 10.5 Ferret protein electrophoresis values.

PROTEIN ELECTROPHORESIS VALUES FOR FERRETS (Melillo, 2013)	UK Units	US Units
Albumin	56–72 g/l	5.6–7.2 g/dl
Alpha$_1$ globulins	3.3–5.6 g/l	0.33–0.56 g/dl
Alpha$_2$ globulins	3.6–6 g/l	0.36–0.60 g/dl
Beta globulins	8.3–1.2 g/l	0.83–1.2 g/dl
Gamma globulins	3–8 g/l	0.3–0.8 g/dl
A/G	1.3–2.1	1.3–2.1

Table 10.6 Ferret haematology.

HAEMATOLOGY (Hein *et al.*, 2012)	UNITS	REFERENCE RANGE
Red blood cells	10^{12}/l	7.4–13.0
Packed cell volume	l/l	0.4–0.7
Haemoglobin	mmol/l	8.6–13.6
Mean corpuscular volume	fl	49.6–60.6
Mean corpuscular haemoglobin concentration	mmol/l	17.8–20.9
Mean corpuscular haemoglobin	fmol/l	1.0–1.2
Platelets	10^9/l	171.7–1280.6
White blood cells	10^9/l	3.0–16.7
Differential blood count (absolute values)		
Monocytes	10^9/l	0.0–0.5
Lymphocytes	10^9/l	0.6–10.5
Band neutrophilic granulocytes	10^9/l	0.0–0.1
Segmented neutrophilic granulocytes	10^9/l	0.9–7.4
Eosinophilic granulocytes	10^9/l	0.0–0.7
Basophile granulocytes	10^9/l	0.0–0.2
Differential blood count (relative values)		
Monocytes	%	0.0–6.5
Lymphocytes	%	12.6–80.6
Band neutrophilic granulocytes	%	0.0–1.2
Segmented neutrophilic granulocytes	%	17.2–81.9
Eosinophilic granulocytes	%	0.0–5.7
Basophile granulocytes	%	0.0–1.4

Pre-medication

Ferrets, like many small mammals, resent injections. They have a small muscle mass which makes IM injections challenging and often painful. Phillips *et al.* (2015) evaluated two doses of transmucosal detomidine gel (Dormosedan Gel 7.6 mg/ml; Zoetis, New York, NY USA) and found that both doses (2 and 4 mg/m^2) resulted in a rapid, reversible, safe, and effective sedation that was suitable for physical examination and venipuncture in all but one ferret, which required masked with isoflurane to facilitate venipuncture with no reaction. As we know from other studies that body surface area in ferrets is virtually the same as cats, we can use this information to use transmucosal gel at 2 mg/m^2 as a stress free pre-medication for mask induction in ferrets. *It is important to remember that detomidine can cause a transient hyperglycaemia which if used to facilitate venipuncture* (especially if investigating insulinoma) could confuse or make the results more difficult to interpret. In my experience most ferrets do not require sedation for venipuncture.

Other options for pre-medication include:

- medetomidine 0.02 mg/kg and butorphanol 0.1 mg/kg in same syringe IM or SC
- medetomidine 0.02 mg/kg and ketamine 5 mg/kg in same syringe IM
- midazolam 0.2 mg/kg and butorphanol 0.2 mg/kg IM.

Detomidine and medetomidine can be reversed with an equal volume of atipamezole SC, IM, or IV. Midazolam may be reversed with flumazenil 0.05–0.1mg/kg SC, IM. See Table 10.7 for Premedication Doses).

Induction

My induction agent of choice in ferrets is isoflurane. I wrap the ferret in a towel (bunny burrito style) and pre-oxygenate for four breaths then induce with 5% isoflurane. I have performed induction with and without premedication. Ferrets find IM injections painful and stressful, so whether I administer pre-medication will depend on the animal's personality and the specific surgery I am performing. Alternatively, transmucosal detomidine gel (see above) may be used as a premedication without causing distress.

Intubation

Intubation is recommended in all but the shortest of procedures. It is important to remember that manipulation of the neck during intubation can cause stimulation of the vagus nerve and an increase in intestinal motility (may increase risk of rupture in cases of intestinal foreign body).

Intubation is performed similarly to cats, using 2.5–3.5 uncuffed endotracheal (ET) tubes, and spraying of the larynx with lidocaine (Intubeaze® 20 mg/ml

Table 10.7 Premedicant doses.

Drug	Dose and route	Reversal
Transmucosal detomidine gel	2 mg/m² OTM	Atipamezole equal volume IM SC
Buprenorphine	0.04–0.06 mg/kg OTM	
Midazolam	1–2 mg/kg IM SC	Flumazenil 0.1 mg/kg SC
Midazolam + butorphanol	0.2 mg/kg + 0.2 to 0.5 mg/kg IM	Flumazenil 0.1 mg/kg SC
Medetomidine + butorphanol	0.02 mg/kg + 0.1 mg/kg IM, SC	Atipamezole equal volume IM SC
Medetomidine + ketamine	0.02 mg/kg + 5 mg/kg IM	Atipamezole equal volume IM SC

Laryngopharyngeal Spray Solution for Cats Dechra) is recommended to prevent laryngospasm. Ferrets may retain some jaw tension despite being under a surgical plane of anaesthesia which can make intubation more challenging. The ET tube should be tied over the ferret's nose as the neck is wider than the head making it hard to keep the tubes tied behind the head.

Maintenance

I use a T-piece circuit, with an oxygen flow of 1.5 l/min. Isoflurane 2–4% as required.

Monitoring

Monitoring the anaesthetic is the same as for more familiar species (a moistened cotton bud can be used to check the blink reflex). Multiparameter monitoring is possible.

Theatre

Ferrets have a high surface area to volume ratio which means they *lose heat rapidly*. The resulting hypothermia can cause fatalities. Keeping the ambient temperature in theatre high, placing a towel on the operating table, and using a plastic drape (also allows visualisation of your patient!) are all easy ways to help mitigate heat loss. Minimise the number of times the door is opened as each time it happens the ambient temperature in the room will drop. *Hypothermia leads to prolonged recovery and increased risk of death*.

Positioning

Placing a folded drape or face cloth under the thorax elevates the thorax and allows the abdominal organs to move away from the diaphragm, which makes breathing easier. If the ferret is not intubated, ensure the head and neck are straightened to allow for easier breathing.

Surgical preparation

To conserve body temperature, minimise the surgical clip. Only one agent is used for surgical scrub, either chlorhexidine or an iodine-based skin disinfectant depending on the surgeon's preference. We do not use a surgical spirit-based spray as during evaporation this causes loss of heat. Using warm water for the surgical scrub will help reduce heat loss.

Transparent, plastic surgical drapes are ideal as they allow visualisation of the patient while conserving body heat. The required surgical window can be cut into the drape and a surgical spray adhesive is used in lieu of towel clips.

Application of a topical EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) can be applied postoperatively to the wound.

Recovery

Detomidine and medetomidine can be reversed with an equal volume of atipamezole SC, IM, or IV. Reversing the detomidine or medetomidine will also reverse the analgesia provided by them, but that is not reason enough for not reversing.

Maintain the ferret on oxygen, and if intubated, extubate when the jaw tone increases. Shivering thermogenesis is a sign of recovery from anaesthesia. Continue to monitor while in an incubator or a warm room (ideally around 26°C (79°F)) until up and moving then the temperature of the room can be reduced to 20°C (68°F).

Offer food as soon as the ferret is fully awake. If the ferret will not eat, consider syringe feeding with a carnivore care diet.

Owners should be advised that ferrets can bruise significantly postoperatively which can look alarming. (Figure 10.10). It is unknown why some ferrets do this.

Dental disease

The ferret's permanent dentition is fully developed at eighty days of age and their normal bite is scissor bite. Their adult dental formula is: 2 × (I 3/3, C 1/1, P 3/3, M1/2).

Nausea in ferrets presents as drooling and pawing at their mouths. Owners may interpret this as oral pain. The pawing may result in traumatic ulceration of the mouth (Figure 10.11). Trimming the front nails short will reduce any trauma, although the underlying cause should always be addressed.

A full examination of the oral cavity is essential as foreign bodies may be found lodged across the hard palate between the upper dental arcades. This is more commonly seen when ferrets are fed whole carcass prey. I see this more commonly in kits.

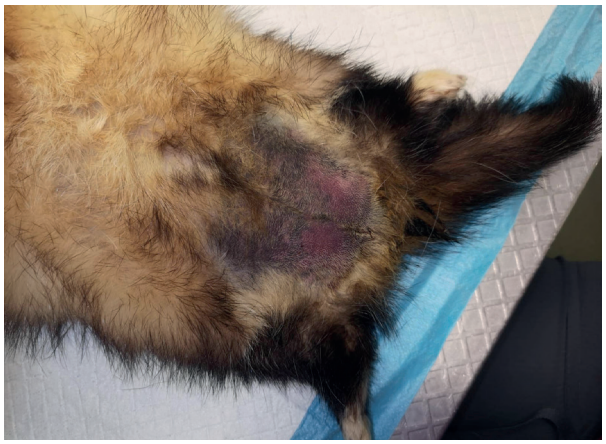


Figure 10.10 Some ferrets bruise significantly post-operatively.



Figure 10.11 Ferrets paw at their mouths when nauseous and may cause trauma.

Development of dental disease is extremely common in pet ferrets although in one study 83.7% were affected by gingivitis only (Nemec *et al.*, 2016). Caries are uncommon even in animals with significant calculus.

Fractures of the canines are frequently seen, but minimal intervention is required unless the pulp cavity has been exposed. The high frequency of dental disease may be in part genetic (Nemec *et al.*, 2016) as well as dietary. Soft diets may predispose to dental disease. Ferrets evolved carnivore dentition, not teeth to eat hard kibble therefore *increased wearing of the teeth may be seen in captivity*.

Periapical abscessation can occur and it is possible to present as a nasal discharge due to fistula formation.

Oral neoplasia is uncommon in ferrets (Hoefer, 2020), although squamous cell carcinomas (SCC) and various sarcomas have been described. Oral neoplasia carries a poor prognosis (Brown, 1997).

Dental radiographs

Dental radiography is recommended prior to any extractions as root numbers of teeth can vary between ferrets (Nemec *et al.*, 2016).

Dental surgery

Dental surgery in ferrets is a similar procedure to that performed in dogs and cats. Dental abscesses may be treated similarly to dogs and cats.

Post-dental care

Owners can be taught to brush their ferret's teeth with an enzymatic toothpaste made for cats.

Heat stroke

Ferrets cannot tolerate environmental temperatures over 30°C (86°F) and it can result in the onset of heat stroke in as short as 10 minutes. *As ferrets do not pant as a physiological mechanism to maintain body temperature, i.e. to lose heat, panting in ferrets should be seen as a sign of marked respiratory distress*.

Clinical signs are similar as in more familiar species, including open mouth breathing, panting, red mucous membranes, weakness, severe depression, muscle cramps, diarrhoea, and vomiting. Left untreated it will progress to seizures, shock, coma, and death from multiorgan failure can occur in as short as 30 minutes.

Ferrets found suffering from heat stroke should be cooled by wetting of the fur, fans can be used to increase evaporative heat loss from drying of the wet fur, and surgical spirit on the foot pads can be used to increase heat dispersal. IV fluids should be administered to counteract shock and support organ function. Severe cases will require broad-spectrum antibiotics due to bacterial translocation from the intestine. Gastric protectants should be given.

Respiratory disorders

Ferrets have a large pulmonary capacity for their size (three times higher than expected for an animal this size) and can breathe through their mouth and nose. The left lung is divided into the cranial and caudal lobes, the right lung is divided into cranial, middle, caudal, and accessory lobes. The normal respiratory rate is 33–36 bpm. Pulse oximetry correlates well with oxygen saturation as measured by blood gas analysis in ferrets.

Pneumonia is generally considered uncommon in this species. Respiratory disorders present similarly to more familiar species. Thoracic compliance should always be checked as reduction in thoracic compliance suggests presence of

pathology e.g. pleural effusion, pulmonary consolidation, mass, increased heart size, or abscess. *Panting in ferrets should be seen as a sign of marked respiratory distress*. Anxious ferrets with severe respiratory distress may benefit from a low dose sedation.

Investigations can include full clinical examination, biochemistry and haematology, thoracic radiography, ultrasonography, thoracocentesis, or bronchoalveolar lavage.

Radiographs

To reduce the risk of artefact from hypostatic congestion we always perform the dorsoventral thoracic radiograph first before progressing onto right and possibly left lateral radiographs. Assess the radiographs for the exact same patterns as in more familiar species, paying particular attention for: bronchopneumonia, lung lobe consolidation, pleural effusion, neoplasia, widened mediastinum, or tracheal elevation (Figures 10.12a & b).

Lymphoma/sarcoma may increase the size of the mediastinal lymph nodes causing dyspnoea, and should be included in the differential list for ferrets presenting with respiratory signs and reduced thoracic compliance.

Other diagnostics

Ultrasonography can be utilised to differentiate between soft tissue opacities and fluid. *Owners can find the strangest things distressing, so it is important to tell owners that their ferret will be shaved for ultrasound as many owners will not make the connection between ultrasound and the need for clipping*.

Drainage of a pleural effusion will immediately provide relief while hopefully allowing a cytological diagnosis.

Bronchoalveolar lavage (BAL), can be performed, with 1–1.5 ml/kg of warm sterile saline being the recommended volume. Intubate with a sterile ET tube, pass a suitably sized dog urinary catheter that has been prefilled with sterile saline and already has the syringe attached. This prevents the saline for the BAL being lost in the dead space of the catheter. Instil the saline and then aspirate. Scant saline may be recovered but keeping the syringe attached to the catheter as it is withdrawn, allows the saline that has been aspirated into the catheter to be collected into a sterile tube for submission for cytology and ideally culture.

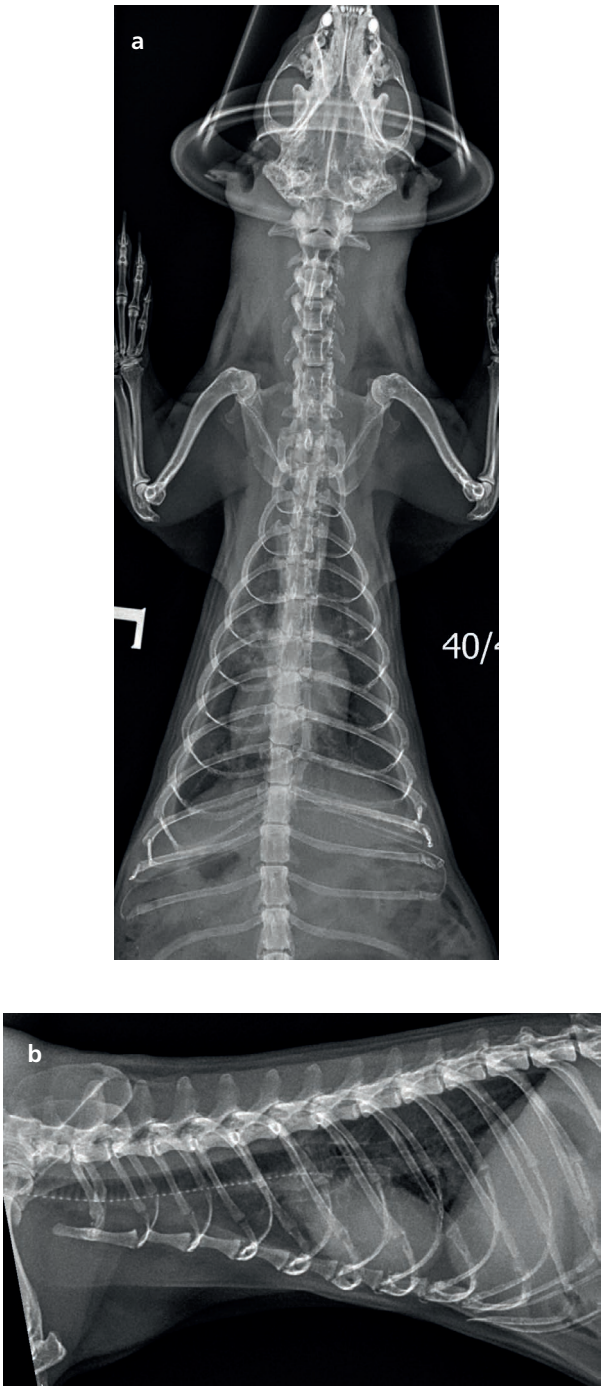


Figure 10.12 Radiographic appearance of a case of pneumonia.

Treatment

Ultimately the treatment required will be dictated by the actual diagnosis. Treatment options are very similar to options available in dogs and cats.

Nebulisation works well in ferrets. N-acetyl L-cysteine 10–20% at 22 mg/ml saline q8–12 hrs as needed works well as a mucolytic but owners should be warned that *initially coughing will increase as the mucous breaks down and is cleared*. Antimicrobials, bronchodilators (e.g. terbutaline 2.5–5 mg/kg q12–24hrs), and other drugs can be delivered via nebulisation at dosages extrapolated from dogs or other species. See Table 10.8 for suggested doses.

For neoplasia or cardiology see the relevant sections.

Allergies

Seasonal allergies may be seen in ferrets presenting as sneezing and rhinitis, which respond to antihistamine treatment. Reducing aerosol usage such as air fresheners, scented candles, and products such as Febreze spray, may also help.

Table 10.8 Nebulisation doses suggested in ferrets.

Drug	Dose (most nebulisers will hold 10 ml)
Saline	10 ml good for hydration of respiratory tract
Acetylcysteine (mucolytic)	10–20 mg per 1 ml saline (higher doses may be associated with bronchospasm)
Bromhexine (mucolytic)	0.15 mg per 1 ml saline
F10	1:250 or ready to use (already prediluted)
Aminophylline	3 mg per 1 ml saline
Amikacin	5 mg per 1 ml saline
Doxycycline	13 mg per 1 ml saline
Enrofloxacin	10 mg per 1 ml saline
Gentamicin	5 mg per 1 ml saline

Bacterial respiratory disease

Ferrets can be susceptible to bacterial respiratory disease (RD) such as rhinitis, tracheitis, and pneumonia. Mixed infections are common and, in many cases, bacterial involvement may be secondary to another pathogen/condition. Examples of isolated bacteria include *Bordetella bronchiseptica*, *Escherichia coli*, *Pseudomonas* spp., and *Mycobacterial* spp. *Nontuberculous mycobacteriosis* is well documented in ferrets and often present with eyelid lesions and conjunctivitis. *Nontuberculous mycobacteriosis* is potentially zoonotic although it is generally immunocompromised humans who are at risk.

Viral respiratory disease

Influenza

Ferrets are susceptible to human influenza; it should be remembered that this and other zoonotic conditions can go both ways. *Any veterinary staff who are displaying flu-like symptoms, even mild ones, should either avoid working with ferrets or wear gloves and a mask*. They are also susceptible to avian, swine, and equine influenza viruses.

Clinical signs include transient diarrhoea, fever, anorexic, lethargy, and upper respiratory signs such as coughing and sneezing. *In the early stages this could resemble distemper infection*. Exudative conjunctivitis with periocular crusting and photophobia may also be seen.

Treatment is symptomatic and antihistamines e.g. chlorpheniramine 1–2 mg/kg q8–12hrs PO may help. Meloxicam 0.2 mg/kg q24hrs can be given. Most uncomplicated cases resolve in 7–10 days. Although pneumonia appears to be uncommon, the ferret should be monitored for any signs of secondary bacterial involvement (e.g. *Streptococcus pneumoniae*) such as green/yellow sputum and any worsening of respiratory signs. Any ferret with signs of secondary bacterial infection should be treated with a broad-spectrum antibiotic such as clavulanate amoxicillin 25 mg/kg q8–12 hrs. As most ferrets respond well to symptomatic treatment and supportive care, antiviral medications are often not required.

Neonatal ferrets, juveniles, or ferrets debilitated from co-morbidities are at a higher risk of developing severe, acute bronchopneumonia and will display severe, persistent, dyspnoea which may result in death.

Distemper

Distemper virus is uncommonly seen, but as ferrets are so susceptible to the disease it should be considered in unvaccinated ferrets. Rescue centres, or ferrets living with dogs will have a higher risk of exposure and young ferrets are more susceptible. *Any ferrets presenting with respiratory, gastrointestinal, neurological, and dermatology signs should have distemper included on the list of differentials*. Incubation in ferrets is typically 4–10 days with onset of clinical signs seen 7–9 days.

CLINICAL SIGNS

Clinical signs include fever, diarrhoea (may have melena), nasal and ocular mucopurulent discharge (eyelids may stick together), corneal ulceration, anorexia, and several days after ocular signs a generalised orange-tinged dermatitis (if ferret lives long enough dermatological signs resolve 17–22 days post-infection). Respiratory signs may develop into a severe fulminating pneumonia due to secondary bacterial involvement, if they survive this phase, central nervous system (CNS) signs such as myoclonus, paresis, muscle tremors, hyperexcitation, convulsions, and coma may develop.

Hyperkeratosis of the foot pads or nose is not as commonly seen nor as pronounced as seen in dogs due to the rapid disease progression in ferrets. *Most ferrets will die within one week of onset of clinical signs* but depending on the strain of the virus, the course of the disease varies from 2–35 days.

DIAGNOSIS

Diagnosis may be challenging, and a combination of history, clinical signs, and diagnostic testing are used. Haematology often shows a severe leukopenia, thrombocytopenia, and a regenerative anaemia. Microscopic examination of conjunctival smears for inclusions are diagnostic although false negatives are possible if early in the course of the disease. Immunohistochemistry of upper respiratory tract or skin biopsies, and polymerase chain reaction (PCR) are other options.

TREATMENT

Treatment is not advisable as this condition is 100% fatal in ferrets.

Cardiology

Cardiac disease is relatively common in ferrets and is diagnosed in middle to aged animals. Dilated cardiomyopathy is the most commonly diagnosed heart condition. Aleutian disease and lymphosarcoma can also cause damage to the heart. *Thromboembolic disease secondary to cardiac disease in ferrets has not been reported.*

A ferret's heart is situated more caudally in the thorax compared to more familiar species, is best auscultated between ribs 6 and 8, and should be auscultated in the same manner as more familiar species. The quoted resting heart rate of normal ferrets varies widely depending on text read from 180–400 bpm. *However, Van Zealand and Schoemaker (2023) quote the resting heart rate as 200–250 bpm, which is consistent with my clinical observations. Van Zeeland and Schoemaker (2022) define tachycardia in the ferret as a heart rate over 250 bpm and severe tachycardia as over 300 bpm, which may lead to life-threatening conditions being missed if clinicians have read texts with the wide heart rate range.*

Thoracic compliance is best checked by laterally compressing the thorax midway, then again at the costal arch. Reduction in thoracic compliance will occur with conditions that take up space in the thorax, e.g. dilated cardiomyopathy, pleural effusion, and lymphoma to name a few.

Pulses

The femoral pulse quality and rate is more easily palpated with the ferret resting on the forearm of the examiner (Figure 10.13).

Clinical signs

Clinical signs associated with cardiac disease (coughing, tachypnoea, syncope, exercise intolerance, pleural effusion, respiratory distress, ascites, weak/irregular pulses, and prolonged capillary refill time) are similar to more familiar species but in ferrets may only be recognised in advanced cases.

Capillary refill time is most easily determined using the unpigmented foot pads. There will frequently be *a reduction in thoracic compliance*. Nonspecific signs such as lethargy, weight loss, anorexia, and weakness of the hind legs are common.



Figure 10.13 Taking femoral pulse.

Non-invasive blood pressure measurement

Reference ranges in ferrets were established using 0.2 mg/kg midazolam IM and butorphanol 0.2 mg/kg IM as a sedation. The recommended site for measurement is the tail, and the cuff size should be 40% of the circumference of the tail. If the cuff is too large it will result in an underestimation of the blood pressure.

Van Zeeland *et al.* (2017) wrote that the arterial pressures of healthy adult ferrets were established at 95–155 mmHg (systolic), 69–109 mmHg (mean), and 51–87 mmHg (diastolic).

Modified vertebral heart score

A modified vertebral heart score (mVHS) has been established in ferrets. The reference range for mVHS is 5.4–5.8 (Godart *et al.*, 2023; Stepien *et al.*, 1999).

Measurements are taken from right lateral thoracic radiographs. The LA line is drawn: Length of the heart from ventral border of bifurcation of the mainstem bronchi to apex, the SA line is drawn: maximal width of the heart measured at the middle third perpendicular to the LA measurement. Both lines are then measured individually against T5–T8 to the nearest 0.25 vertebrae.

Treatment of cardiac disease is similar to in dogs and cats, frequently the cat doses are used. Monitoring includes regular clinical examination, monitoring changes in body weight, renal values, heart rate, and rhythm. If diuretics and angiotensin-converting-enzyme inhibitors (ACEI) are used concurrently it may result in severe azotaemia. In cases of symptomatic pleural effusion, thoracocentesis may be used to alleviate the dyspnoea.

Dilated cardiomyopathy

The exact cause of dilated cardiomyopathy (DCM) is unknown in ferrets. Deficiencies in taurine or carnitine have been suggested as possible causes but are unproven. *DCM is the most commonly diagnosed cardiac disease in ferrets.*

Clinical signs

Clinical signs vary from asymptomatic through to lethargy, weakness, anorexia, weight loss, exercise intolerance, respiratory distress, weak pulses, pulse deficits, tachycardia (>250 bpm), heart murmur (systolic), gallop rhythm/arrhythmia, hypothermia, pale mucous membranes, cyanosis, increased respiratory effort, muffled heart, increased respiratory sounds (moist rales or crackles), prolonged capillary refill time (>2.5 s), hind end paresis, hepatomegaly and/or splenomegaly, or ascites.

Diagnosis

Diagnosis is confirmed by radiograph and echocardiogram. Electrocardiography (ECG) can also be performed if arrhythmias are suspected or heard.

In cases of DCM, radiographs will show if there is evidence of presence of pleural effusions, tracheal elevation, an enlarged cardiac silhouette, pulmonary oedema, and allow a mVHS to be performed. The mediastinum should be examined for potential presence of lymphoma (Figure 10.14).

If the cardiac silhouette is enlarged, then echocardiogram should be used to rule out pericardial effusion and assess the chambers of the heart to differentiate between DCM and hypertrophic cardiomyopathy (HCM). In cases of DCM the left ventricle inner diameter will be enlarged (reference for inner diameter should be less than 1.3 cm). The heart will also be generally enlarged with thin-walled chambers and contractility is often generally poor.

Treatment

Treatment includes oxygen therapy and minimising stress. Furosemide 1–4 mg/kg q8–12hrs IV, SC, or PO. Another diuretic which can be used is spironolactone 1–2 mg/kg q12hrs. I usually start ferrets on pimobendan 0.5–1.25 mg/kg q12hrs. Digoxin 0.005–0.01 mg/kg q12–24hrs may be used if the ferret is tachycardic, monitoring is important (clinical signs of toxicity include anorexia, nausea, vomiting, and lethargy, especially in ferrets with renal insufficiency) and the serum therapeutic range is 1–2 ng/ml 6–12h following oral administration.

Ferrets are thought to be sensitive to the hypotensive effects of ace inhibitors and can quickly become weak and lethargic. Careful monitoring and starting at the lower dose are advisable. Benazepril 0.25–0.5 mg/kg q24hrs or enalapril 0.25–0.5 mg/kg q24–48hrs can be added to treatment is required.

If DCM is diagnosed early in the condition, then prognosis is reasonable with many living up to 2 years on medication. If the heart disease is advanced at time of diagnosis the prognosis is more guarded. Owners should be counselled on the risk of sudden death due to arrhythmias.

Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is rarely seen in ferrets and the cause is unknown. It is characterised by concentric hypertrophy of the left ventricular wall and interventricular septum.

Clinical signs

Clinical signs vary from asymptomatic through to lethargy, weakness, anorexia, weight loss, exercises intolerance, respiratory distress, weak pulses, pulse deficits, tachycardia (>280 bpm), heart murmur (systolic), gallop rhythm/arrhythmia, increased respiratory effort, increased respiratory sounds (moist rales or crackles), and hind end paresis/ataxia.

Diagnosis

Diagnosis is via radiography and echocardiography. Radiographs may reveal a normal to increased cardiac silhouette and if left sided heart failure is present, signs of pulmonary oedema or pleural effusion. Echocardiography may reveal (generalised or local) gross thickening of the interventricular septum and/or left ventricular free wall and decreased left ventricular dimensions.

Treatment of HCM includes beta-blockers such as atenolol 3.1–6.2 mg/kg q24hrs or propranolol 0.2–1.0 mg/kg q8–12hrs PO. The calcium channel blocker diltiazem 3.7–7.5 mg/kg q12hrs PO may also be given if the tachycardia is not fully controlled. Combined, the drugs help to reduce myocardial oxygen consumption and increase cardiac output, but owners should be advised that careful monitoring is required, as overdosing may lead to significant bradycardia, hypotension, lethargy, and/or inappetence. If heart failure is present diuretics and ACEI should be considered. Positive inotropic drugs (e.g. pimobendan or digoxin) should be avoided.

Valvular disease/endocardiosis

Valvular disease or endocardiosis is recognised with increasing frequency in middle aged to older ferrets with the mitral and aortic valves being the most commonly affected. Secondary dilatation of the left atrium and left ventricle may occur.

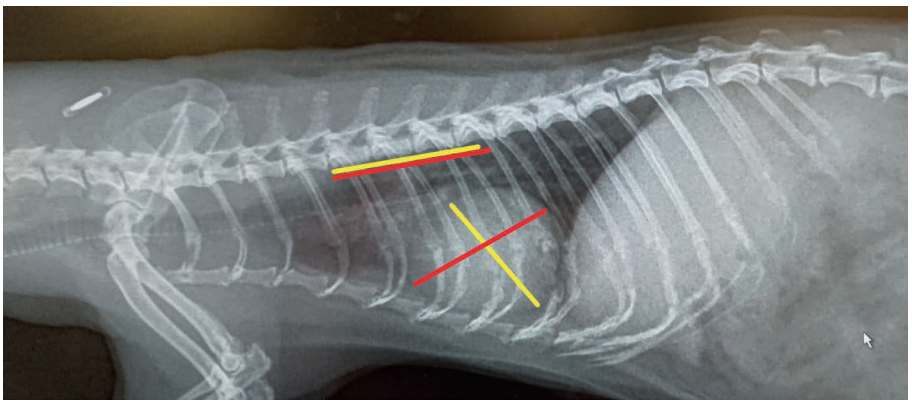


Figure 10.14 Lateral radiograph of a DCM case performing a mVHS.

Clinical signs

Clinically a murmur may be auscultated. In cases involving the mitral valve a systolic murmur is heard best over the left apical region, if the tricuspid valve is involved a systolic murmur is best heard in the right parasternal area, whereas aortic insufficiency, a diastolic murmur, is rarely heard on auscultation. There may be clinical signs of congestive heart failure which can include a weak pulse, abdominal distension resulting from hepatosplenomegaly and/or ascites, dyspnoea, tachypnoea, coughing, increased respiratory sounds, moist rales or crackles, or muffled heart and lung sounds.

Diagnosis

Diagnosis is achieved by thoracic radiography to assess cardiac size and check for pulmonary oedema or pleural effusion. Echocardiography is also advisable.

Treatment

Treatment is not required for asymptomatic heart murmurs, but regular check-ups are advisable. If congestive heart failure is present then treatment options are the same as in more familiar species: furosemide, pimobendan, ACEI, and digoxin.

Pericardial effusion

Primary causes of pericardial effusion are not reported in the ferret. Secondary causes include infection, neoplasia, uraemia, autoimmune disease, coagulopathies, hypoalbuminaemia, cardiomyopathy, or right sided heart failure.

Clinical signs and diagnosis

Clinical signs are similar as those reported in dogs and range from being absent to significant disease depending on the volume of fluid present.

If the fluid builds slowly then signs tend to be associated with right sided heart failure: dyspnoea, tachypnoea, cough due to pleural effusion, hepatomegaly, and ascites.

If the fluid builds rapidly it will result in both left and right outflow compromise (tamponade), leading to severe shock, syncope, and potentially death.

Diagnosis is via echocardiography.

Treatment

Treatment is dependent on volume of fluid and clinical signs.

If the ferret is asymptomatic and there is only a small volume of fluid, then no treatment is necessary. If the effusion is secondary to another condition treatment should be aimed at the underlying disease, and if there is a moderate to large volume of fluid then pericardiocentesis will help alleviate clinical signs and the fluid can be sent for cytology and bacteriology.

If the effusion recurs and is idiopathic a pericardiectomy can be considered.

Heartworm

Heartworm is uncommon but any ferrets in endemic areas should be tested for heartworm if they display cardiac signs. Ferrets most at risk are those living outdoors in endemic areas. Ferrets are susceptible to *Dirofilaria immitis*, which is spread by mosquitoes.

The adult worms reside in the right ventricle, cranial vena cava, or the main pulmonary artery. *Owing to the small size of ferrets' hearts, even one adult worm can result in severe heart disease, and two worms may be enough to result in a fatal right sided heart failure due to mechanical obstruction.*

Clinical signs and diagnosis

Clinical signs can vary from asymptomatic, anorexia, lethargy, weakness, depression, hypothermia, dyspnoea, tachypnoea, cyanosis, coughing, pale mucous membranes, abdominal distention, occasionally tachycardia and heart murmur, through to sudden death due to pulmonary artery obstruction.

Diagnosis can be achieved by echocardiography visualising the worms in the pulmonary artery, right ventricle, and/or right atrium as parallel linear echodensities. It is rare to get eosinophilia associated with heartworm.

Serology is also an option when looking for adult *D. immitis* antigen but false negatives are possible if there are less than five worms.

Circulating microfilariae are only present in around half of affected animals which makes the modified Knott's test of limited use in ferrets.

Treatment

Ferrets with symptomatic cardiac disease due to heartworm may require stabilisation before an adulticide is given. Stabilisation may include the administration of oxygen, frusemide, or enalapril. If significant pleural effusion is present, thoracocentesis can be performed.

Treatment requires an adulticide which may result in complications from worm emboli and prednisolone 0.5–1 mg/kg q12–24hrs should be administered concurrently with an adulticide and continued for approximately four months.

Ivermectin 0.05–0.1 mg/kg q30days SC is the recommended adulticide drug of choice and needs to be administered until resolution of the clinical signs and microfilaria is absent. Strict cage rest is advisable for 4–6 weeks after treatment with an adulticide.

It is advisable to perform serology 3 months after starting therapy and testing repeated at monthly intervals until the results are negative. If antigen test results remain positive, further diagnostic tests (radiographs, echocardiography) may be necessary to determine whether

heartworm infection persists. Most ferrets become seronegative within 4 months after successful adulticide therapy.

Preventative therapy is recommended in all heartworm endemic areas and should be started 1 month before the start of the expected heartworm season and continued on until 1 month after the end of the heart worm season. Options for prevention include ivermectin 0.05–0.1 mg/kg q30days SC or PO, selamectin 18 mg/kg q30days topically, or milbemycin oxime 1.15–2.33 mg/kg q30days PO. Housing ferrets indoors during mosquito season may help limit exposure.

Arrhythmias

Cardiac arrhythmias can be physiologic or pathologic in origin. *Sinus arrhythmia is the most common physiologic arrhythmia* and can be caused by respiration or an increased vagal tone. Sinus tachycardia and second-degree atrioventricular (AV) block have also been reported to occur physiologically in healthy ferrets. Most ECG abnormalities are incidental and do not clinically affect the ferret. Sinus bradycardia is commonly found in ferrets with insulinoma.

Pathologic arrhythmias may occur following stress, pain, hyperthermia, hypoxia, shock, electrolyte or metabolic disturbances, sepsis, intoxications (e.g. digoxin), anaesthesia, endocrine disease, anaemia, and cardiac abnormalities (e.g. DCM).

Clinical signs

Clinically arrhythmias may remain undetected until picked up on a routine auscultation or ECG. Second- and third-degree AV blocks may result in life-threatening bradycardia (<120 bpm), lethargy, weakness, exercise intolerance, pulse deficits, and syncope. If heart rate drops consistently below 80 bpm, congestive heart failure and hypoperfusion may develop. Ferrets with severe tachycardia (>300 bpm) may have similar signs, and ventricular tachycardias can quickly result in death if not promptly treated.

Diagnosis

Diagnosis requires ECG to determine the type of arrhythmia. If bradycardia is present the use of the atropine response test will determine if it is vagally mediated. Biochemistry, haematology, radiographs, and echocardiography may be used to rule out underlying conditions.

ATROPINE RESPONSE TEST

Atropine is given at a dose of 0.02–0.05 mg/kg IV, IM, SC. *If the bradycardia is vagally mediated there should be an increase in heart rate 15–30 minutes post-administration*. The dose can be repeated if necessary. A positive response is diagnostic of sinus bradyarrhythmia but as atropine is short acting, propantheline 0.25–0.5 mg/kg q8–12hrs PO is used to treat sinus bradyarrhythmia.

Treatment

Treatment of tachycardia or tachyarrhythmia includes antiarrhythmic drugs, such as lidocaine, digoxin, or beta-blockers (e.g. propranolol 0.2–1.0 mg/kg q8–12hrs PO, atenolol 3.125–6.25 mg/kg q24hrs), to lower the heart rate.

Ferrets with clinical signs resulting from high-grade second- and third-degree AV block may benefit from anticholinergics (e.g. propantheline 0.25–0.5 mg/kg q8–12hrs PO), beta adrenergics (e.g. terbutaline 2.5–5 mg/kg q12–24hrs, isoproterenol 20–25 µg/animal IM, q4–6hrs SC, 40–50 µg/animal q4–6hrs PO), and phosphodiesterase inhibitors (e.g. aminophylline 4–6.6 mg/kg q12hrs, theophylline 4.25 mg/kg q8–12hrs PO), especially if heart rate increased following atropine administration.

Any underlying conditions should be treated.

Ophthalmology

The approach to ophthalmology in ferrets is similar to our approach in dogs and cats, they are just eyes. Most *ferrets have a diminished or absent menace reflex*. There is a well-developed retrobulbar venous sinus, which can lead to significant blood loss during enucleation. Should bleeding occur during enucleation it can be controlled by direct pressure or placement of a collagen sponge, such as Collaclot. There are two lacrimal glands, one dorsolateral and one within the nictitating membrane. Tear production measured with standard Shirmer tear tests is documented as 5.3 +/– 1.3 mm/minute.

Ferrets have a round pupil when mydriatic but it is *horizontally ovoid when miotic*.

Staphylococcal spp. and *Corynebacterium* spp. have been isolated from the eyelid margins and conjunctival sacs of healthy ferrets and may be considered normal eye flora.

The most common ophthalmic conditions seen in ferrets are corneal ulcers and cataracts.

Entropion

Primary (and secondary to conjunctival foreign bodies) entropion has been documented in ferrets. (Figures 10.15a & b) Corneal ulceration may occur as a result of the secondary trichiasis. Surgical correction is the same as performed in more familiar species. I use a sterile cotton bud to tent the eyelid for the surgical incision.

Conjunctivitis

Unilateral conjunctivitis may be associated with a foreign body whereas bilateral conjunctivitis may be associated with dusty bedding causing corneal irritation and epiphora. Primary bacterial conjunctivitis is also seen in ferrets.

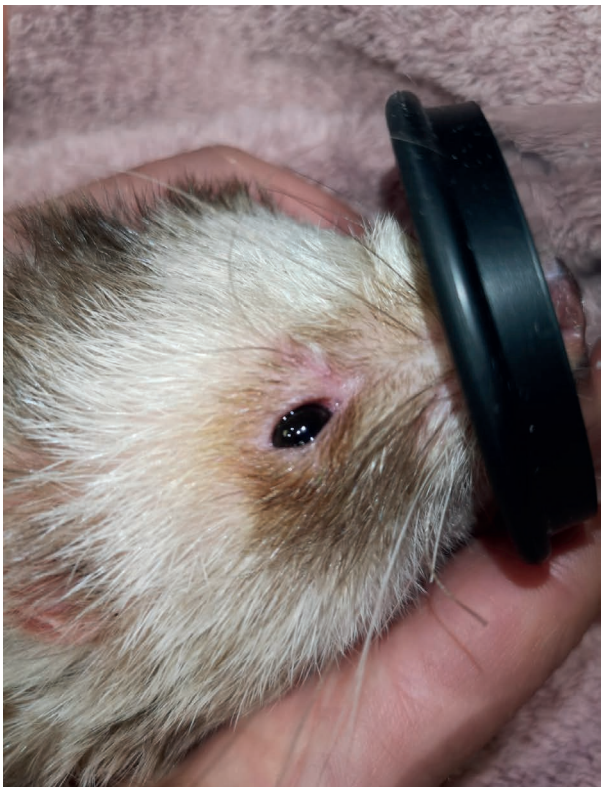


Figure 10.15a Entropion before manually correcting.

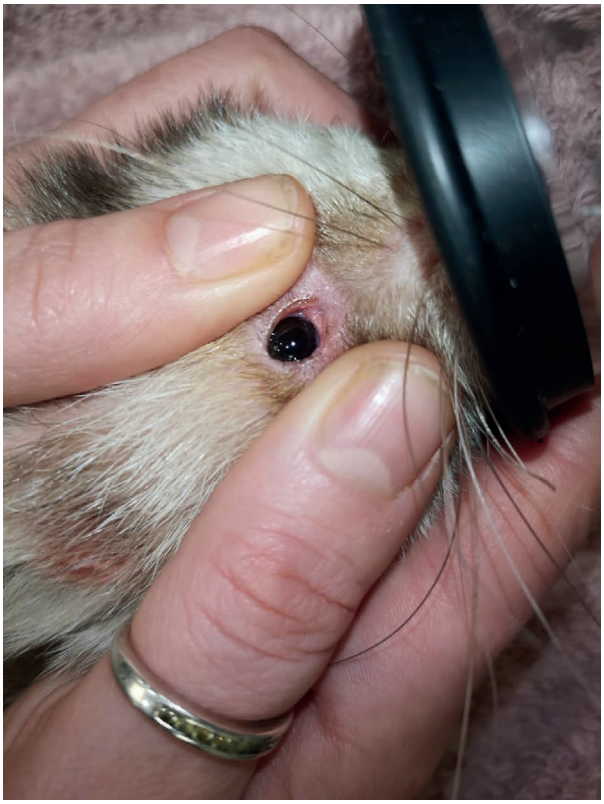


Figure 10.15b Entropion after manually correcting.

Bilateral conjunctivitis may also be associated with systemic conditions such as: canine distemper virus, influenza, mycobacterial infections, and *Salmonella*. Concurrent systemic clinical signs are used to definitely diagnose the underlying condition.

Treatment of conjunctivitis is similar to more familiar species. Conjunctivitis associated with systemic disease will require diagnosis and treatment of the underlying condition.

Corneal disease

Investigated in a similar manner to more familiar species using ophthalmic exam and fluorescein should be applied with any lesions documented.

Corneal erosions/ulcers

Straw bedding may lead to corneal ulceration due to 'poke' injuries. Treatment is the same as in more familiar species. Antibiotic eye drops should be used in cases where bacterial involvement is suspected. Topical lubrication is also advisable. Analgesia such as meloxicam should be provided until the cornea heals. Congenital corneal dermoids are sporadically seen and are similar to those seen in dogs.

Lens disorders

Nuclear sclerosis is noted in older ferrets.

Cataracts

Cataracts are very common in ferrets and the aetiology is not fully understood. They can be seen at any age but are most commonly seen in ferrets older than 5 years of age, congenital cataracts have been reported (Figure 10.16).



Figure 10.16 Cataract.

Successful surgical treatment of cataracts in ferrets using extracapsular or phacoemulsification techniques has been reported (van der Woerdt, 2003).

> In my experience, many owners do not notice their ferret has cataracts even when they are functionally blind. This is likely due to their small eyes, tendency to be in constant motion, and that they have very poor eyesight anyway. *If the ferret is happy and the eye is not painful then there is no need for further treatment or surgical intervention.*

Exophthalmos

Exophthalmos with secondary exposure keratitis may be seen with several conditions. The most common cause is retrobulbar lymphoma although other retrobulbar tumours are possible. Differential diagnosis includes trauma, zygomatic salivary gland mucocele and retrobulbar abscess due to a dental abscess.

Investigation is the same as for more familiar species.

Glaucoma

Primary and secondary glaucoma is considered rare in ferrets. The normal values of IOP in the ferret as measured with a Tono Pen XL is 12–17 mmHg (Montiani-Ferreira *et al.*, 2006) and 14.07 ± 0.35 as measured with a TonoVet (Di Girolamo *et al.*, 2013). Medical management may be attempted as per canine patients but from scant reports, medical management success is temporary at best.

Dermatology

Chronic feeding of an inappropriate diet may, among other things, result in a dull, dry hair coat.

> Owners should be advised that it is completely normal for any areas of hair regrowth (either in alopecic areas or from surgical clipping) to appear as bluish discolouration of the skin until the hair follicles erupt, to prevent them interpreting this as bruising.

Ferrets undergo a semi-annual moult that is non-pruritic and can result in bilateral, symmetrical alopecia of the dorsal lumbosacral region and will resolve spontaneously.

Bacterial skin disease

Bacterial pyodermas are uncommon in ferrets but may be seen secondary to mating (seen on the neck) or excoriation and can be treated as per dogs and cats.

Pododermatitis

Pododermatitis is seen in ferrets who are kept in wet, unsanitary condition. They may present with one or more feet swollen, erythematous, crusted, and painful to touch. The tail tip may also be affected.

Differential diagnosis should include distemper and the pedal form of *Sarcoptes*. *Staphylococcus aureus* is most commonly isolated. Treatment should include a full husbandry review, analgesia, and antibiotics.

Abscesses

Abscesses are seen secondary to fighting, self-trauma, play fighting, or rodent bites during hunting.

Management is similar to cat bite abscesses (Figures 10.17a–c), the ferret should be anaesthetised, the abscess lanced and flushed. Culture and sensitivity can be performed and most frequently *Staphylococcal aureus* or *Streptococcus* spp. are isolated. Analgesia and antibiosis should be given. My first line antibiotic choice in ferrets is clavulanate amoxicillin 25 mg/kg q8–12hrs PO.



Figure 10.17a Swelling in the neck.



Figure 10.17b Swelling in the neck once lanced.



Figure 10.17c Swelling in the neck once lanced and flushed.

Mycobacterium avium

Mycobacterium avium may present as: alopecia, bruising, scabs, and numerous skin nodules. Diagnosis is achieved by histopathology of skin biopsies. *Treatment is difficult as even prolonged courses of combination antibiotic therapy often fails to achieve a cure*. *M. avium* is zoonotic, and although owners generally would require to be immunocompromised to contract the disease, the *difficulty in treating coupled with the zoonotic risk means euthanasia should be recommended*.

Dermatophytosis

Dermatophytosis is rarely seen in ferrets and is generally non-pruritic. Most clinical cases have been found in young or immunocompromised individuals who have been exposed to infected cats. *It should be considered potentially zoonotic despite there never being a documented case of ferret to human transmission*. Trichophyton mentagrophytes and *Microsporum canis* are both reported in ferrets and a single report exists of *M. nanum*.

Clinical signs

Clinical signs include annular, non-pruritic, alopecia that spreads peripherally. Severe cases will present with generalised alopecia, diffuse scaling, erythema, and crusting.

Rarely, the fungus can spread into the dermis and SC adipose tissue. If this occurs, nodules are created which may ulcerate and produce a granular discharge. This form is referred to as *dermatophytic pseudomycetoma* and can be difficult to treat, there is a high risk of progression of disease and in most cases the animal will either be euthanised or die.

Diagnosis

Diagnosis is achieved by fungal culture and examination of the hair/scales under microscope. Investigation of the cause of immunosuppression should be performed.

Treatment

The treatment of choice is systemic antifungals. Options are griseofulvin 25 mg/kg q24hrs, itraconazole 5–10 mg/kg q24hrs, or fluconazole 10 mg/kg q12hrs for at least 2–4 weeks post-resolution of clinical signs but ideally until two negative cultures are returned. Ferrets treated with griseofulvin or itraconazole should be monitored for liver disease or bone marrow suppression.

Any in-contact animals should also be treated, and the environment should be disinfected to destroy spores. F10 disinfectant is my go to as it is effective against most pathogens and is sensitive enough for the ferret's delicate respiratory tract.

Ectoparasites

Mites

OTODECTES CYNOTIS

Ear mites are extremely common in ferrets and frequently missed during a clinical exam. This is due to the normal dark appearance of ferret ear wax and the fact even heavy infestations may be asymptomatic.

If present, clinical signs include mild to moderate pruritus, head shaking, hyperkeratosis of the pinnae, aural haematoma, erosions and crusting leading to otitis externa. The mites may spread to other parts of the body.

Microscopic examination of the ear wax (Figure 10.18) will readily demonstrate the mite.

Selamectin 15–45 mg/ferret as a single topical application is often enough to clear infestations although another treatment may be applied after 4 weeks.



Figure 10.18 Taking sample to check for ear mites.

As this is the same species of ear mite that infests dogs and cats, they may transfer between the species by direct contact. Some owners' ferrets play with their dogs and cats so it is important to check as all in-contact ferrets, dogs and cats must be treated at the same time.

Sarcoptes scabiei: although uncommon, two forms of disease are recognised in ferrets: localised, or generalised. Localised infestations of *S. scabiei affect the feet only*. There is severe pruritus of the feet, swelling of the toes and feet, paronychia, and onychauxis (overgrowth/thickening of the nail which is likely to become discoloured). The claws may slough. Dark brown scabs may form on the pads and owners may mistake this sign for distemper lesions.

Generalised infestations of *S. scabiei affect the body and head*. There is pruritus, cutaneous thickening, crusting, erythema, alopecia, and secondary bacterial infection. Severely affected ferrets can become lethargic and untreated they will eventually die.

Diagnosis is by microscopic examination of multiple deep skin scrapes (false negatives are common as relatively few mites can cause clinical signs) or histopathology. *Owners should be advised that this mite is zoonotic*.

Treatment options include SC injections of ivermectin 0.2–0.4 mg/kg q7–14 days alongside environmental cleaning q3 days until the mites are eradicated. Selamectin 45 mg/ferret applied topically q28 days for two treatments may also be effective. Trimming claws will help reduce excoriation from scratching. Warm water foot soaks are recommended for the pedal form of the condition. Analgesia is advisable and antibiotics should be given in cases of secondary bacterial infections.

Demodex spp. has been reported in the skin of the genital, perianal, and facial regions in healthy ferrets without any clinical signs. Clinically apparent cases are rare and when found are associated with conditions causing immunosuppression or following prolonged high dose glucocorticoid therapy.

Fleas

There is no ferret flea, so ferrets with fleas will generally have picked up *Ctenocephalides felis* or *C. canis*. Heavy infestations may result in anaemia, and intense pruritus which can cause alopecia of the head, neck, and thorax (Figure 10.19).

Diagnosis is direct visualisation of the flea or flea dirt. Treatment of the environment is required to help control the flea infestation as well as treatment of any other ferrets, dogs, or cats the owner has.

Ticks

Several species of ticks may be found on ferrets. Hunting ferrets or escaped pet ferrets are at a higher risk of picking up ticks (Figure 10.20). There is no louse species seen in ferrets.



Figure 10.19 Flea allergic dermatitis.



Figure 10.20 Ticks on the neck of a pet ferret who had escaped for a couple of weeks.

Neoplastic skin disease

Neoplasia is common in ferrets and approximately 20% of neoplasms affect the skin. *Most cutaneous neoplasms are benign.*

Sebaceous epithelioma

Sebaceous epithelioma are the most common benign skin tumours diagnosed in ferrets. They can present anywhere but are mainly seen on the head and the neck.

Sebaceous epithelioma can have a variety of presentations: localised, non-encapsulated, solid or cystic, plaque like or protruding, may be hyperpigmented, may be ulcerated, and can be solitary or multiple lesions may be present in the one individual. *The main differential diagnosis (especially for plaque like lesions) is mast cell tumours.*

Excision is curative. The cut surface of the tumour can be pale yellow through to white and is usually arranged in irregular lobules.

Mast cell tumours

Mast cell tumours (MCT) are very common skin tumours in ferrets and *are considered benign in this species*. The average age of the affected ferret is 4.5 years old but may be seen in younger animals.

MCT can have a variety of presentations. The mass may be discrete, flat and plaque-like or be nodular. Some MCT are pigmented, they may also be pruritic, erythematous, with ulceration and crusting forming if excoriated (Figures 10.21a & b). Reportedly, approximately 30% of ferrets affected will have more than one MCT.

Excision is curative. It is advisable to administer famotidine 2.5 mg/ferret q24hrs PO and an antihistamine pre-operatively. Chlorpheniramine 1–2 mg/kg q8–12hrs PO or diphenhydramine 0.5–2 mg/kg q8–12hrs PO, IV, IM can be used.

Chordomas

Chordomas are moderately common in ferrets despite being rare in most other mammals. Affected ferrets are generally 2 years or older and most commonly present with a smooth, firm, poorly haired, slow growing mass of the tail tip (Figures 10.22a & b). The mass may have superficial ulceration. Chordomas are locally invasive and can distort adjacent vertebrae.

Chordomas may be diagnosed using fine needle aspiration or excisional biopsy and histopathology. If associated with the tail tip, partial amputation of the tail above the affected vertebrae may be curative.

Although it is most commonly the tail tip that is affected, chordomas have been reported in association with cervical and thoracic vertebral bodies along with invasion of the spinal canal and impingement of the spinal cord leading to ataxia, paresis, or ventral deviation of the trachea depending on the location and size of the chordoma. These presentations of chordoma are difficult to manage due to being locally invasive, with a high rate of recurrence as full resection in these sites is impossible. Progression of disease may result in pathological fracture of the affected vertebrae necessitating euthanasia.

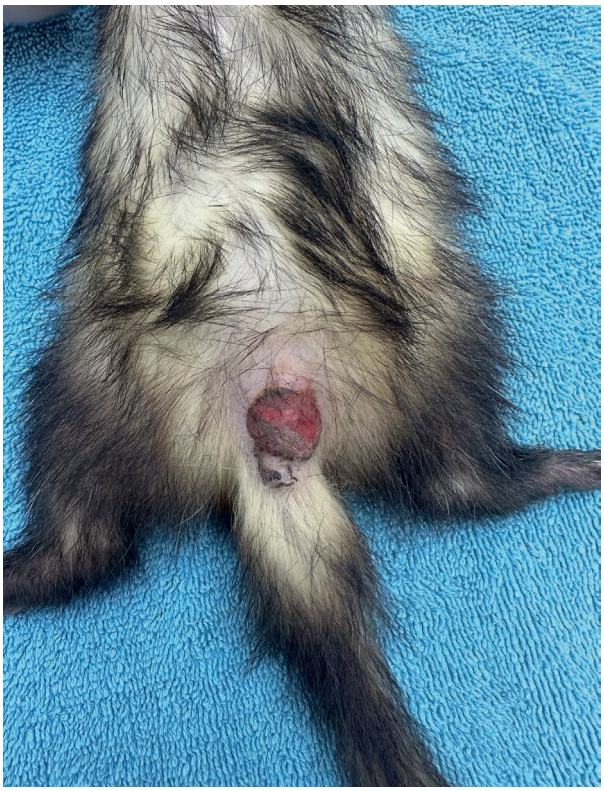


Figure 10.21a Mast cell tumour in a ferret.



Figure 10.21b Same mast cell tumour once scrubbed clean.



Figure 10.22a Chordoma of the tail. The lump was firm, note the loss of fur.

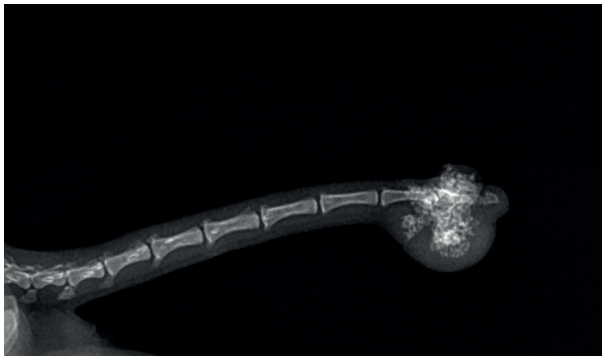


Figure 10.22b This radiograph is of the same ferret pictured in 10.22a. Note the bone involvement.

Squamous sell carcinoma

Squamous cell carcinoma (SCC) is malignant, can present anywhere but are more frequently seen on the head and face where they often extend to involve the oral cavity. They may be focal or multifocal cutaneous plaques with or without ulceration and can present as subcutaneous masses.

They are locally aggressive, and prognosis depends on the site and completeness of excision. For example, SCC of the face has a poor prognosis due to the invasion of soft tissues, mouth, nasal cavity, and bone.

Cutaneous lymphoma

Cases of cutaneous lymphoma are rarely reported and in one study (Kanfer & Reavill, 2013) accounted for 1% of the cutaneous neoplasia seen in ferrets. Lesions seem to be mainly on a limb or involve the feet although lesions over the lumbar region are occasionally seen. Foot lesions are often pruritic, with diffuse generalised alopecia, erythema, erosions, crusting, and ulcerated plaques. Multiple or single individual papules may be seen when located in the lumbar region. Visually epitheliotropic cutaneous lymphoma often is indistinguishable from non-epitheliotropic cutaneous lymphoma, so surgical biopsy is always required for diagnosis. From the few reports, all cutaneous cases are T-cell lymphomas.

Cutaneous lymphoma is relatively non-responsive to corticosteroids, although some cases will show an initial improvement. (Kanfer & Reavill, 2013) Supportive care, such as antibiotics if secondary bacterial infections are present and medicated shampoos, may keep pets comfortable. Treatment with isotretinoin 2 mg/kg q24hrs PO has been beneficial in some cases (Rosenbaum *et al.*, 1996).

Multiple chemotherapeutic treatment protocols have been described in the literature for lymphoma and *in my experience if there are such diverse treatment protocols available it is generally because none of them work particularly well.* Prognosis is variable as cutaneous lymphoma in ferrets uncommonly progresses into systemic disease. If the skin lesions are surgically resectable and are rapidly excised, prolonged survival times (possibly up to 3–4 years) may be seen.

With cutaneous lymphoma, further research is needed to determine an efficient and safe treatment plan for chemotherapy in ferrets, as information on treatment and outcome is scarce in the literature.

Apocrine gland masses

Apocrine glands are found in the highest concentrations around the head, neck, vulva, prepuce (see reproductive disorders), and perianal areas. Cystic dilatations of apocrine glands are common, and, in these cases, excision is curative (Figure 10.23). *Visually these cannot be differentiated from adenocarcinomas so either fine needle aspirate (FNA) or excisional biopsy is recommended to allow for surgical planning rather than performing drastic surgery such as preputial and penile amputation with urethrotomy needlessly* (Figure 10.24).

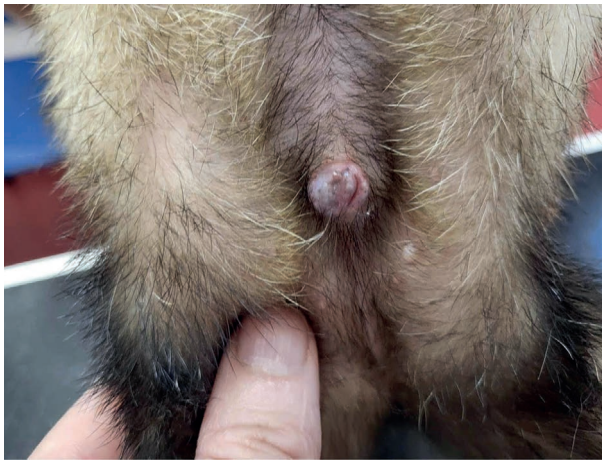


Figure 10.23 Preputial mass that was benign on histopathology.

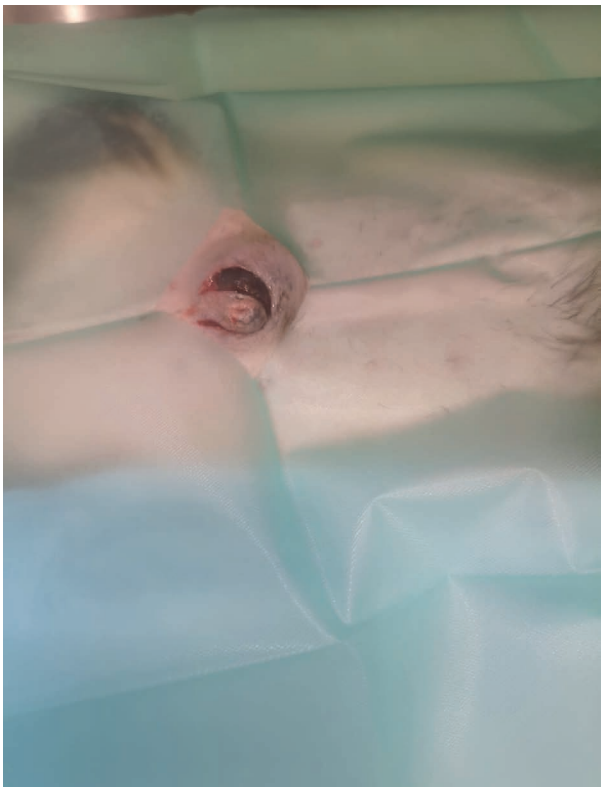


Figure 10.24 Preputial mass that was malignant on histopathology but local surgical resection was curative.

Mammary tumours

Mammary neoplasia is rare in ferrets.

Ear diseases

Ear mites, see *Otodectes cynotis* above.

Splenomegaly

The spleen is often readily palpated in the healthy ferret and the normal spleen has been reported to measure 5.1 cm in length, 1.8 cm in width, and 0.8 cm thick, however these measurements were taken during post mortems and given that we know the spleen reduces in size as part of post mortem change, these values should be considered low.

Suran *et al*. (2017) demonstrated the spleen was easily detected on abdominal radiography in the healthy ferret and normal spleens are frequently seen along the ventral border of the abdomen.

Splenomegaly is a common finding in the aged ferret, and it is often an incidental finding. *The vast majority of cases of an enlarged, smooth spleen in an asymptomatic ferret will be most often due to extra medullary haemopoiesis*. Occasionally the sheer size the organ can get to will cause discomfort (Figure 10.25). *Care should be taken when palpating a massive spleen in case of friability*.

However, if the spleen is nodular or the ferret is suffering from nonspecific clinical signs then investigation is

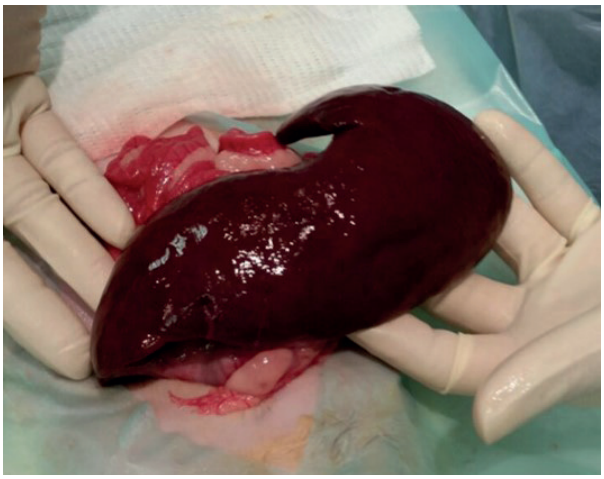


Figure 10.25 Splenomegaly where the sheer size of the organ was causing discomfort.

warranted. Babinska *et al*. (2017) demonstrated that several conditions including lymphoma (Figure 10.26) and mycobacterial infection could cause splenomegaly. Other conditions that may result in splenomegaly include DCM and Aleutian disease. *In cases of lymphoma ultrasound of the*

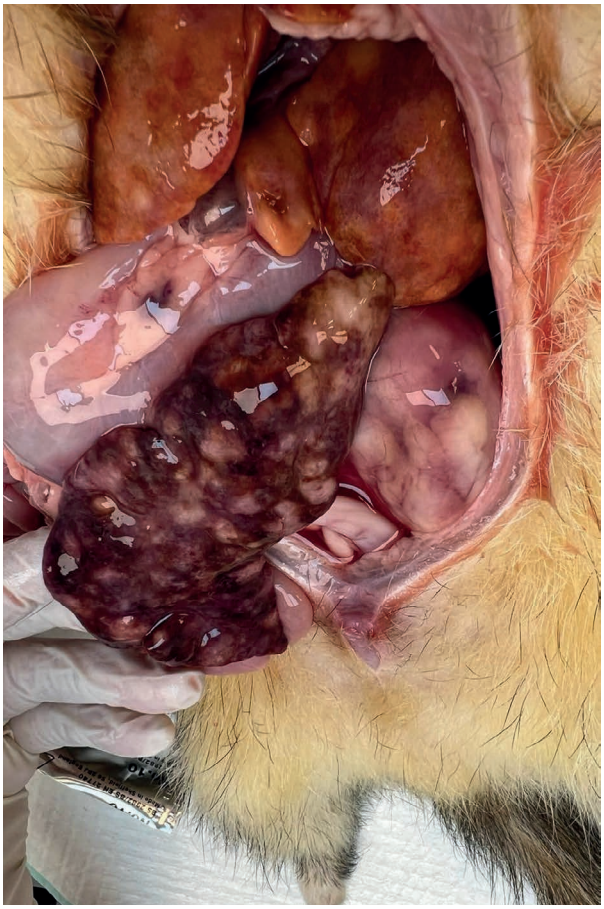


Figure 10.26 An example of splenomegaly due to lymphoma.

spleen will often show irregular and hypoechoic areas. Ultrasound guided needle biopsy prior to surgery is advised.

> The only indications for a splenectomy are a definitive diagnosis warranting surgery or if the size of the organ is causing discomfort.

Endocrine disorders

The three most commonly reported endocrine diseases reported in ferrets are insulinoma, hyperestrogenism, and adrenal gland disease. Hyperestrogenism and adrenal gland disease are covered in the section on reproductive disorders. We should remember that just because an endocrine disease has not been reported in ferrets does not mean that it cannot happen, so if the clinical signs correspond with an endocrine disease seen in more familiar species, then it is worthwhile investigating it.

Insulinoma

Insulinoma is a common endocrine condition accounting for 25% of all neoplasms seen in ferrets (Chen, 2010). The mean age of affected ferrets is 5 years old but it can be seen in ferrets as young as 2.

Tumours of the pancreatic beta cells produce excess insulin and the clinical signs are associated with that. The resulting hypoglycaemia is the most common cause for signs resembling neurological disease in ferrets. Onset of disease may be slow and insidious. Feeding a diet too high in carbohydrates has been suggested as a cause of insulinoma but no link has ever been proven. Other, rarer causes of hypoglycaemia in ferrets include liver disease, sepsis, neoplasia, starvation, or heatstroke.

Clinical signs

Clinical signs can include slight weakness or incoordination (appearing drunk), tremors, weight loss, lethargy, glazed expression, star gazing, sinus bradycardia, nausea (drool and paw at their mouths) (see Video 10.1a & b), melena, complete collapse, and coma. *Unlike dogs, seizures are not commonly seen in ferrets associated with insulinoma*. Clinical signs usually spontaneously resolve when fed (apart from coma).

Diagnosis

Whipple's triad says a presumptive diagnosis is based on the following: hypoglycaemia (<3.4 mmol/l (≤60 mg/dl)), neurologic signs, and resolution of clinical signs with glucose administration.

A blood glucose level of less than 3.4 mmol/l (60 mg/dl) if the ferret has been fasted for 4 h and has clinical signs can be considered diagnostic. If the glucose level is between 3.4–4.2 mmol/l (60–75 mg/dl) then fast for another 2 h before repeating the test. *The ferret should be monitored carefully for clinical signs during this time*.

In my experience ferrets exhibiting clinical signs have had blood glucose levels too low to read on POC (point of care) glucometers. Lab analysers should be considered gold standard for diagnosis and monitoring as even veterinary POC glucometers can over- or underestimate glucose. However as long as we are aware of the limitations POC glucometers can be useful and *Petritz (2013) found that the canine setting had the best correlation with bench top analysers. The toe paw pads can be used to gain a drop of blood via lancet needle*.

Duhamelle *et al.* (2018) found that fructosamine measurements did not significantly differ between the healthy and hypoglycaemic ferrets, which highly suggests that fructosamine measurement is not likely to be useful in determining insulinoma associated chronic hypoglycaemia in ferrets.

Insulin levels can be measured (but are of debatable value) and should be interpreted alongside blood glucose levels. *Insulin within reference range alongside a low blood glucose should be considered abnormal as when faced with a low blood glucose, the body's insulin level should be low not within reference range*.

Unlike in dogs, insulinomas in ferrets are generally benign (hyperplasia or adenomas) and as such rarely metastasise. The incidence of multiple insulinomas (approximately 61%) is higher than that of a single insulinoma (39%) (Weiss *et al.*, 1998). Although this condition cannot be cured it can be managed. Surgery and or medical management has been used.

Emergency treatment of hypoglycaemia

If the ferret is conscious, oral administration of a high dextrose gel such as those commonly used in hypoglycaemic diabetic patients can be given, followed by EmerAid Carnivore or the ferret's own food.

If the ferret is unconscious, injectable 50% glucose diluted 1:1 with saline can be given by IV bolus, 0.25–2 ml over 1–3 minutes to effect and once the ferret is conscious it is important to feed the animal as soon as possible. Some ferrets may require continued IV fluids spiked with glucose (as per dog and cat patients).

Surgery

Although several texts say the left lobe of the pancreas is most commonly affected (Caplan *et al.*, 1996; Weiss *et al.*, 1998) a study performed by Wu *et al.* (2017) on the 'Ultrasonographic features of insulinoma in six ferrets' found that the distribution of the pancreatic nodules in their cases was 46.4% in the left lobe, 50% in the right lobe, and 3.6% in the body of the pancreas and the size of the nodules varied from 1.5 mm × 1.5 mm to 4.1 mm × 5.6 mm. On ultrasound the pancreatic nodules appeared round to oval and hypoechoic or anechoic to hypoechoic. Therefore, ultrasonography of the pancreas may be a

useful part of investigation, diagnosis, and potentially preoperative planning.

Surgery can include nodulectomies or partial pancreatectomy but it should be noted that not all of the diseased tissue may be macroscopic. Macroscopic disease is usually seen as discrete red raised nodules varying in size from a few millimetres to over 1 cm. Medical management may need to be started when the ferret has recovered if clinical signs occur. The clinician should warn the owner that diabetes mellitus may be a complication of surgery. Pancreatitis may also occur but this seems to be uncommon (Fox & Marini, 2014).

Vascular anatomy of the pancreas should be studied prior to surgery. This is to prevent damage to the blood supply to the spleen (left lobe pancreas) or the duodenum (right lobe pancreas) (Fox & Marini, 2014).

Caplan *et al*. (1996) and Weiss *et al*. (1998) both showed that hypoglycaemic episodes recurred in 86–100% of surgical cases. Once clinical signs recurred medical treatment was started. They demonstrated a survival time of 456–668 days with a combination of surgery and medical management giving the longest survival. However, Fox and Marini (2014) state medical management alone can result in survival 60–510 days.

Medical management

In my experience, many owners opt for medical management and most ferrets do well (1–2 years). I do discuss surgical options and that there is the potential for several months of longer life using the combination of surgery and medical management.

Prednisone can be started at 0.25–1 mg/kg q12hrs orally. *The aim is not to restore euglycaemia but to alleviate clinical signs*. The dose can be titrated up to 2 mg/kg q12 hrs if more control is required. *Ferrets appear relatively resistant to the usual side effects of polyuria and polydipsia*. Weight gain and impaired hair growth may be seen with prolonged prednisolone use but is rarely an issue. If prednisone is not controlling clinical signs adequately then diazoxide can be introduced at 5–30 mg/kg q12hrs orally.

Some clinicians advocate starting with diazoxide first, however it is more expensive than prednisone, can potentially have more side effects (vomiting, anorexia, diarrhoea, lethargy), should be used with care if also has cardiomyopathy, and ferrets find it unpalatable. If vomiting or lethargy occurs, then famotidine can be given 2.5 mg/ferret. *However, there is an argument that famotidine should be given routinely at the start of treatment as prolonged periods of hypoglycaemia result in a prolonged release of gastric acid contributing to gastric ulceration*.

Along with medical management, dietary management plays a role. Owners should be advised to feed a high-quality low carbohydrate food, small amounts frequently. Treats should not contain sugar unless they are having a hypoglycaemic episode. Oxbow Carnivore Care or EmerAid Carnivore can be given to alleviate episodes or as a supplement to the daily rations.

Diabetes mellitus

Spontaneous diabetes mellitus is very uncommon in ferrets. Transient hyperglycaemia may be seen with partial pancreatectomy surgery for insulinoma, but it usually will resolve without therapy in 1–2 weeks. *If the hyperglycaemia post-surgery lasts for over 2 weeks treatment will be required*.

Clinical signs

Clinical signs are identical to those seen in dogs and cats: polyuria/polydipsia, lethargy, weight loss despite good appetite. In advanced stages the ferret may become anorexic, depressed, vomit, ataxic, and develop hepatomegaly due to secondary hepatic lipidosis and ketoacidosis.

Diagnosis

Diagnosis is based on repeated blood glucose measurement, fructosamine measurement and urinalysis. *Handheld glucometers should be on the dog setting and the toe paw pads can be used to gain a drop of blood via lancet needle*. Blood glucose over 21 mmol/l (400 mg/dl) is suggestive but is not diagnostic unless several measurements have been performed. Fructosamine reference range in ferrets has been established as 101–202 μmol/l. Glycosuria is not diagnostic on its own as stress-induced hyperglycaemia may possibly exceed renal threshold. *However, glucosuria, and the presence of a secondary bacterial cystitis even without ketonuria is highly suggestive*.

Treatment

Spontaneous diabetes mellitus ferrets may be ketoacidotic at time of diagnosis necessitating hospitalisation, IV fluids, and insulin. Diets offered should be low carbohydrate and high in animal protein with moderate fat content (which is the normal dietary requirement for ferrets) and should be fed in over four portions daily, one portion with each injection and another portion 4–6 h later. Insulin therapy may be started at 0.5–1 IU/kg q12–24hrs (Meredith, 2015) and blood glucose should be monitored. Owners should be advised of signs of hypoglycaemia and how to manage an episode. Reports are scant and prognosis may be guarded.

Hypothyroidism

Thyroiditis is seen in Aleutian disease but it is not associated with hypothyroidism. Hypothyroidism is rare in ferrets but several cases are described in the literature.

Clinical signs

Clinical signs seen include lethargy, obesity, and hind limb weakness. Fur loss and hypercholesterolemia, although

common in dogs with hypothyroidism, have not been seen in affected ferrets.

Diagnosis

Diagnosis is achieved by testing T4 and finding of low (<10 ng/ml (13 nmol/l)) and lack of response to a thyroid-stimulating hormone (TSH) stimulation test, which should be performed to eliminate cases of sick euthyroid syndrome.

To perform a TSH stimulation test, measure T3 and T4, administer injection of 100 μg of human recombinant TSH IM, and remeasure T3 and T4 4 h later. In cases of euthyroid post-stimulation levels will increase to 1.4 times the pre-stimulatory levels, but ferrets with hypothyroidism will show a maximum increase of 1.2 times baseline levels.

Treatment

Treatment with levothyroxine 50–100 μg PO q12hrs was found to be effective in resolving clinical signs and normalising serum T4 concentrations (Wagner, 2012).

Urinary tract disease

The bladder wall is thin and usually contains a maximum of 10 ml of urine. Clinical signs associated with and investigation of urinary tract disease are similar to more familiar species.

Urinalysis

Urinary specific gravity measurements in clinically healthy, young ferrets varies between the sexes and has been established as 1.034 to 1.070 in entire males and 1.026 to 1.060 in entire females.

Dipstick analysis showed that 80% of the ferrets had a trace of protein (<0.3 g/l) in their urine, with males having a higher incidence (93% compared to 73%), 41% of the ferrets had a trace of blood in their urine, and 16% of ferrets had bilirubin (+1) in their urine (Eshar *et al.*, 2012).

Urinary pH will vary with diet but ideally should be 6–6.5. Higher urinary pH may be seen if the ferret is fed on a diet that is too high in carbohydrates or plant-based proteins, and may predispose the ferret to calculi formation.

Urinary sediment examination is performed in the same manner as for dogs and cats. Normal ferret urine should not contain white blood cells, casts, crystals, or bacteria. Renal casts may indicate pathology of the kidneys. Leucocytes, especially neutrophils indicate a urinary tract infection, white blood cell renal casts suggest pyelonephritis.

Table 10.9 Ferret urinalysis normal.

Protein	Trace of protein (<0.3g/l)
pH	6–6.5
Specific gravity	1.034 to 1.070 in entire males and 1.026 to 1.060 in entire females
Sediment examination	No white blood cells, casts, crystals, or bacteria

Cystitis

Cystitis is less common in ferrets compared to other small mammals. Primary cystitis occurs due to an ascending bacterial infection. Cystitis may also occur secondary to pyelonephritis, cystic calculi, or in association with prostatitis (adrenal gland disease). As the underlying cause of the cystitis may be complicated, cases of recurrent cystitis warrant full investigation. Jills may be more prone to bacteria cystitis due to having a shorter urethra. Bacteria isolated from ferret urine include: *E. coli*, *Staphylococcus aureus*, *Pseudomonas* spp., and *Proteus* spp.

Clinical signs

Clinical signs are the same as in more familiar species: pollakiuria, dysuria (often seen as genital overgrooming), haematuria, and there may be pain on palpation of bladder.

Diagnosis and treatment

Diagnosis is based on clinical signs, clinical examination, and urinalysis. Ideally urinary bacterial culture and sensitivity should be performed. Empirical antimicrobial therapy should be started pending results, suitable choices include: clavulanate amoxicillin 25 mg/kg q12hrs, trimethoprim sulfadiazine 30 mg/kg q12hrs. Antibiotics should be continued for a minimum of 2 weeks and just as in more familiar species, it is important to administer an anti-inflammatory or an analgesic.

If the ferret is clinically unwell, or if clinical exam increases the suspicion that the cystitis is secondary, further investigation in addition to urinalysis with sediment examination may include biochemistry, haematology, radiographs, and/or ultrasonography.

Pyelonephritis

Although it can be challenging differentiating cystitis from pyelonephritis, ferrets suffering from pyelonephritis are usually clinically unwell.

Clinical signs and diagnosis

Clinical signs are usually acute in onset and include anorexia, lethargy, fever, and pain on palpation of the kidneys. Haemolytic *E. coli* and *Staphylococcus aureus* are the most common isolates. Left untreated pyelonephritis may progress to end stage chronic renal failure.

Advisable investigation includes haematology, biochemistry, urinalysis, culture and sensitivity of urine, and ultrasound examination of bladder and kidneys. Blood work may show a leucocytosis with either a left shift or a mature neutrophilia, depending on the severity of the infection the urea and creatinine may (or may not) be

elevated. Urinary sediment examination will usually have: a high white blood cell count (especially neutrophils), bacteria, red blood cells, and there may be renal tubular casts or white blood cell casts.

Treatment

Treatment includes fluid therapy, nutritional support, analgesia, and antibiotics for 4–6 weeks based on culture. Any underlying conditions such as urolithiasis or adrenal gland disease should be identified and treated to prevent recurrence.

Renal disease

Chronic interstitial nephritis is commonly found on post mortem in geriatric ferrets.

Clinical signs and diagnosis

Clinical signs associated with renal disease are similar to other species: depression, lethargy, anorexia, weight loss, melena, dehydration, weakness, vomiting, polydipsia (or reduced water intake), and polyuria. As in more familiar species, ferrets may present acutely.

Diagnostic work up of suspected renal disease is similar to that performed in dogs and cats.

Ferrets have considerably lower serum creatinine (crea) levels and have a narrower range than is seen in other mammals and concentrations of BUN associated with renal disease are not always accompanied by an increase in crea. Due to this, even small increases in crea levels should be viewed as significant. Increases in serum phosphate levels are commonly seen with renal disease. A non-regenerative anaemia may be seen in chronic cases.

Eshar *et al*. (2013) established radiographic measurement of the length and width of North American neutered pet ferret kidneys against the second lumbar vertebrae. Using a ventrodorsal radiograph, the kidney length:L2 was 2.21–2.31 for the right kidney and 2.15–2.25 for the left kidney, while the kidney width:L2 was 1.09–1.14 for the right kidney and 1.07–1.12 for the left kidney. *This may help veterinary professionals unfamiliar with ferrets establish if there is renomegaly*.

Treatment

Specific treatments will depend on the underlying cause of the renal disease (e.g. pyelonephritis versus chronic renal disease versus urethral obstruction), so ideally a diagnosis would be reached to allow targeted therapy.

General recommendations include stopping any nephrotoxic drugs, enteric phosphate binders can be given if there is hyperphosphatemia, IV fluids should be given if there is azotaemia (think kidney cat!), and metoclopramide or maropitant may be given if there is gastrointestinal discomfort.

Ferrets with chronic renal disease and proteinuria (with an inactive urinary sediment) may benefit from benazepril 0.25–0.5 mg/kg q24hrs. *Ferrets are thought to be sensitive to the hypotensive effects of ace inhibitors and may become weak and lethargic. In my experience, starting at the lower dose and increasing if required reduces this risk*. Renal diets are generally too low in protein for ferrets so should not be used.

Owing to the ubiquitous nature of Helicobacter in ferrets, if there are signs of gastric discomfort, treatment of Helicobacter should be started.

Omega 3 supplements may be given as there is evidence they can be of benefit in cases of renal disease in other species. Multivitamin supplements are advisable as the polyuria may result in loss of water-soluble vitamins.

Renal cysts

Renal cysts are common and are often incidental findings, although *if they are identified, renal function should be checked*. Depending on study read, incidence varies between 10–33% of ferrets are affected. In humans, renal cysts may be associated with haematuria and pain. If a large renal cyst is painful, and the biochemistry shows no sign of azotaemia, nephrectomy can be considered.

Polycystic kidney disease

Polycystic kidney disease is uncommon in ferrets but may progress to renal failure and present with anorexia, weight loss, vomiting, diarrhoea, severe lethargy, and may have two palpably enlarged kidneys. Investigations include biochemistry, haematology, and ultrasonography. No specific treatment exists other than the previous recommendations for chronic renal disease, and if the ferret is already in renal failure the prognosis is poor.

Renal neoplasia

Primary renal neoplasia is rare but renal lymphosarcoma is not uncommon.

Renal neoplasms have the potential to haemorrhage leading to hemoabdomen and abdominal distention. On ultrasound, renal neoplasms often present as cystic areas and so may be mistaken for renal cysts (renal cysts are far more common and are benign). In humans, renal tumours are often resistant to chemotherapy and in ferrets too little is known, so the current recommendation for unilateral primary renal tumours is nephrectomy.

Urolithiasis

Uroliths are seen sporadically but are more commonly seen in adults who are chronically fed on poor-quality diets. Poor-quality diets are more likely to be high in protein of vegetable origin leading to alkaline urine and an increased risk of struvite crystal formation. Calcium oxalate and cystine calculi are both also reported although the etiopathology is unclear. Over the last decade, cystine

has become the most prevalent ferret calculi in the USA accounting for 89% of the calculi submitted for analysis. For comparison, over the same time frame, cystine accounted for 27% of the calculi submitted for analysis in Europe.

Single or multiple uroliths of variable size can occur anywhere along the urogenital tract, although stones in the ureter are less common. Urethral obstruction may result in bladder rupture. Secondary bacterial infection is commonly associated with urolithiasis.

Clinical signs

Clinical signs of urolithiasis are similar to those seen in more familiar species: vocalisation during urination, stranguria, dribbling urine, urine scalding, genital overgrooming, pain on abdominal palpation, and haematuria. *Owners may misinterpret the ferret straining to pass urine and present the ferret for constipation*, it is not uncommon for ferrets to pass a small amount of faeces when straining to pass urine.

Diagnosis

Uroliths are radiopaque in ferrets making radiography perfect to achieve diagnosis (Figure 10.27). Good positioning is vital and the urethra should be checked carefully, especially in males as calculi lodged at the os penis may be easy to miss.

Blocked bladders

Blocked bladders present similarly to more familiar species.

Clinical signs may include dehydration, stranguria, dysuria, dribbling urine, urine scalding, genital overgrooming, abdominal pain, haematuria, anorexia, lethargy, and intermittent straining or may be collapsed. An enlarged, turgid bladder may be palpable unless rupture has occurred. Acute renal failure will occur secondary to obstruction unless the obstruction is relieved.

Urethral obstruction is more common in males and calculi lodged at the os penis may be easy to miss. Female ferrets straining with urethral obstruction may present with rectal or vaginal prolapse.

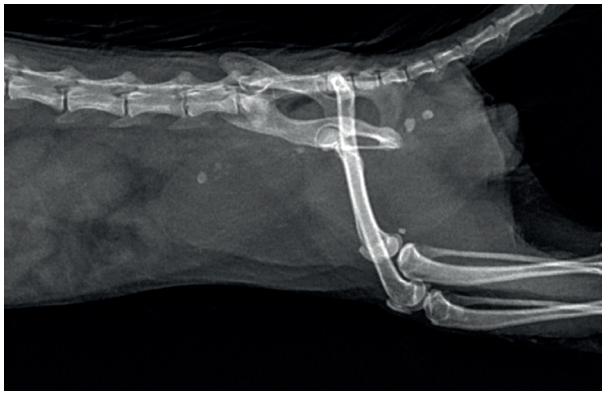


Figure 10.27 Lateral radiograph showing urolithiasis, including stones in the urethra.

Differential diagnosis for urethral obstruction includes uroliths, pyuria, prostatomegaly, periurethral cysts, and neoplasia (rare). Investigation is the same as performed in dogs and cats.

Diagnosis

Biochemistry may show azotaemia, hyperkalaemia, and hyperphosphatemia. It is recommended to correct hyperkalaemia prior to induction due to the cardiotoxic effects of potassium. Insulin and dextrose may be administered which should decrease the potassium within five minutes, however the ferret should be monitored for hypoglycaemia.

Treatment

Owing to the thin wall of the bladder, it is advisable to relieve the obstruction and avoid cystocentesis due to the high risk of bladder rupture.

If the obstruction cannot be relieved, cystocentesis may be performed using a 25g needle. Unless obstructed by prostatomegaly, male ferrets with unrelieved obstruction can have a urethrostomy performed immediately caudal to the base of the os penis.

Tube cystotomy

If a urinary catheter is unable to be passed in ferrets with adrenal gland disease (female ferrets with obstructions due to periurethral swellings or in male ferrets with obstruction due prostatomegaly, prostatic cysts, or periurethral swellings), a tube cystotomy may be performed as a temporary measure to allow time for the treatment of the adrenal gland disease.

During an exploratory laparotomy (allows assessment of prostate and adrenal glands and surgical treatment as needed) pre-place a small purse string suture in a relatively avascular area of the ventral bladder, a 5Fr or 8Fr Foley catheter is then passed into the bladder lumen through the centre of the pre-placed purse string, inflate the balloon of the Foley, and secure the purse string suture. Rakestraw *et al.* (1995) determined that balloon patency and failure were attributed to the process of resterilising the Foley catheters for repeated use, so single use is recommended.

The bladder should be sutured to the body wall using tacking sutures. The Foley catheter is then passed through a separate paramedian incision in the ventral abdomen and secured outside of the abdomen using a finger trap suture. The Foley catheter is capped with an injection cap and aspirated every 2 to 3 h. An abdominal bandage or stockinette bandage should be applied as it is very difficult to maintain an Elizabethan collar on a ferret due to their neck being wider than their heads!

Owners can be taught to aspirate the catheter allowing maintenance at home. It may take several days for

the ferret to pass urine normally and ideally the catheter should be removed at day ten and the bladder surgically repaired. This will allow time for the medical or surgical management of adrenal gland disease to have had an effect and shrink the prostatic tissue. Owners should be made aware of the risks of the ferrets removing the catheters and the potential for uroabdomen or peritonitis resulting. *Although perioperative antibiotics are controversial, if there is an active urinary sediment (which there often is in these cases), treatment with antibiotics, ideally guided by culture and sensitivity, are recommended*.

Urinary catheterisation

Tom cat catheters are not long enough to sit in the bladder of most ferrets. Ideally a 3–3.5Fr 11–14 cm open-ended urinary catheter should be used, another option is a 20–22g jugular catheter, which is approximately 20 cm long. The ferret should be sedated or fully anaesthetised and placed in dorsal recumbency.

The urethral papillae in the jill is found on the ventral vestibule of the vagina. Catheterisation in the jill may be achieved by having an assistant pull both hind legs cranially while maintaining the jill in dorsal recumbency (this position will cause the urethral papillae to open), and passing the urinary catheter into the vagina aiming ventrally. Alternatively, the catheter may be placed using a sterile speculum on an otoscope for direct visualisation.

In males, the urethral opening is a slit like opening on the ventral surface of the glans approximately 2–4 mm proximal to the tip of the penis.

Urethral stones

Urethral catheterisation and retropulsion of the stone into the bladder can be attempted. The *stone may be embedded* into the urethral mucosa which may prevent movement. Diluted lidocaine may be instilled via the catheter to alleviate urethra spasm, which may facilitate retropulsion. Administering diazepam or midazolam may be considered to reduce urethra spasm.

Unless obstructed by prostatomegaly, male ferrets with unrelieved obstruction can have a urethrostomy performed immediately caudal to the base of the os penis.

Cystotomy

Struvite diets are unpalatable to ferrets and too low in protein therefore should not be used in this species. Cystic calculi require surgical removal.

Toxic nephropathies

Ibuprofen toxicosis

Can occur 4–8 h after ingestion (65% will have clinical signs within 8 h of ingestion) and *ferrets appear to be more sensitive to the toxic effects of ibuprofen than dogs*. The lowest dose resulting in death was 220 mg/kg, which means one tablet could be fatal.

The target organs of ibuprofen toxicity are the CNS, gastrointestinal tract, and the kidneys. Renal papillary necrosis and gastrointestinal haemorrhage are commonly seen (see Toxicosis section for more details).

Zinc toxicity

Ferrets appear to be especially sensitive to zinc toxicity and fatalities can occur with ingestion of zinc due to toxic nephropathy (see Toxicosis section for more details).

Reproductive disorders

Ferrets have a very different reproductive strategy compared to more familiar species.

Reproduction

They are seasonal breeders with increasing photoperiod being the stimulus. They become sexually mature in the spring following their birth. The hobs' breeding season starts earlier than the jill, and in the northern hemisphere their testosterone levels start to rise in January. *There is a nice correlation between testicle size and testosterone levels, the peak of both occurring in February*. Jills typically come into season at the beginning of March although there is some individual variation and this year, I was presented with a jill in season on the 15 January.

Mating

Mating in ferrets is aggressive and lengthy. The hob will grab the neck of the jill and will drag her around the enclosure. The mating is often vocal with the jill making a screaming noise. Once the jill accepts the mating she will become limp and the hob will mount her.

Mating can last up to 3 h and some owners will find this distressing. The jill can experience damage to the skin at her scruff and during particularly aggressive matings there can be excoriation of the vulva.

Jills are induced ovulators and require both the neck bite and the stimulation of the cervix by the hob's j-shaped os penis to ovulate. The vulva will start to decrease in size 3–4 days post-mating if ovulation has occurred, failure to do so indicates ovulation has not occurred and the jill should be mated again (Lagerkvist, 1992).

Gestation

Pregnancy can be diagnosed via ultrasound at 12 days after fertilisation and will appear as 3–5 mm anechoic structures in the uterus (Peter *et al.*, 1990) or by palpation 14 days after fertilisation where the foetus can be palpated as walnut sized structures. Rubbing some food or salmon oil on the abdomen distracts the jill during ultrasound (Figure 10.28).

Diet is vitally important to support pregnancy and prevents conditions such as eclampsia, pregnancy toxaemia,



Figure 10.28 Rubbing some food or salmon oil on the abdomen distracts the jill during the ultrasound.

agalactia, and low litter size, for example. Ensuring a high-quality, meat source protein diet with protein 35% and at least 20% fat being readily available for the jill and placed in close proximity to the nest will prevent any issues due to diet. Fresh water should be provided daily.

Gestation in jills is 41–43 days. With 41 days being most common in primiparous jills whereas the average gestation is 42 days.

Parturition

Ferrets do not show overt signs of impending birth the way bitches do. This year, the author's own jills passed a mucous plug 48 h prior to kittening but showed no other signs. The actual birthing process is normally rapid. On average it will take 2–3 hours in total. In pet ferrets, handling of the kits does not appear to cause rejection of the litter provided the jill is comfortable with it. My jills actively pull my hand into the nest from day 1 of birth.

Dystocia is rare in ferrets. In colonies of ferrets, it is said to affect 1% of ferrets and to be even rarer in pet ferrets. Causes of dystocia are similar as seen in more familiar species: too few kits (single kit syndrome), very large litters resulting in uterine inertia, over large kits due to overlong gestation (over 43 days) or abnormal kit presentation. Environmental temperatures exceeding 21°C (70°F) can also result in dystocia.

Kits

Kits weigh approximately 6–10 g at birth and grow rapidly on their mothers' rich milk gaining at least 4 g per day during week 1. They are altricial and rely on the jill for all of their care during the first 3 weeks. Hand rearing of kits before 10 days of age is extremely difficult as they are nutritionally fastidious and require ferret milk only up to this age (Manning & Bell, 1990).

Kits start to sample mashed up wet food at 3 weeks of age while their eyes are still shut. Their eyes do not open until approximately 4.5 to 5 weeks of age. *In my opinion this makes complete sense if you have evolved to be born underground in a tunnel*, therefore you do not need to open your eyes until you are mature enough to leave the tunnels.

By 14 days their deciduous teeth have erupted and are fully through by 18 days of age. Their adult canines start to erupt between 42 and 52 days while they retain their deciduous canines until 56 days of age (Berkovitz and Silverstone, 1969). Their full adult dentition is through by 10 weeks old. Kits can hear sounds by 32 days, for comparison cats gain ability to hear sounds by 6 days (Moore, 1982).

Ferrets imprint on food at a young age by smell so kits should be offered several brands and also dry versus wet. This is to ensure that they will continue to eat well with their new owners and also if there is ever a disruption in the supply of the usual brand. *Some ferrets will point blank refuse to eat unfamiliar food and will starve themselves*.

The kits are ready to be independent and rehomed at 8 weeks.

Hyperestrogenism

Similarly, to rabbits and cats, jills are induced ovulators. *Unlike rabbits and cats, jills will continue to stay in oestrus until they either ovulate, the day length shortens, or they die*.

Ferrets appear to be particularly sensitive to the toxic effects of oestrogen (Sherrill & Gorham, 1985). The risk of hyperestrogenism is high especially if the jill has been in season for 4 weeks or longer. Sherrill and Gorham (1985) demonstrated an incidence of 50% of unmated jills developing hyperestrogenism with a *mortality rate of 40%*. The high, persistent oestrogen levels cause bone marrow suppression resulting in *hypoplasia of all the cell lines and decreases splenic extramedullary haematopoiesis*. The most common cause of mortality is haemorrhagic anaemia due to thrombocytopenia. The neutropenia increases the risk of bronchopneumonia and pyometra.

Clinical signs

Clinical signs include enlarged vulva, weakness, anorexia, progressive weight loss, dehydration, pale mucous membranes, symmetrical flank alopecia, and petechiae or ecchymosis. If bronchopneumonia or pyometra are present (secondary to the leukopenia), clinical signs attributable to those conditions will also be present (Figures 10.29a–d).



Figure 10.29a White appearance to vulva.



Figure 10.29c White appearance to foot pads.

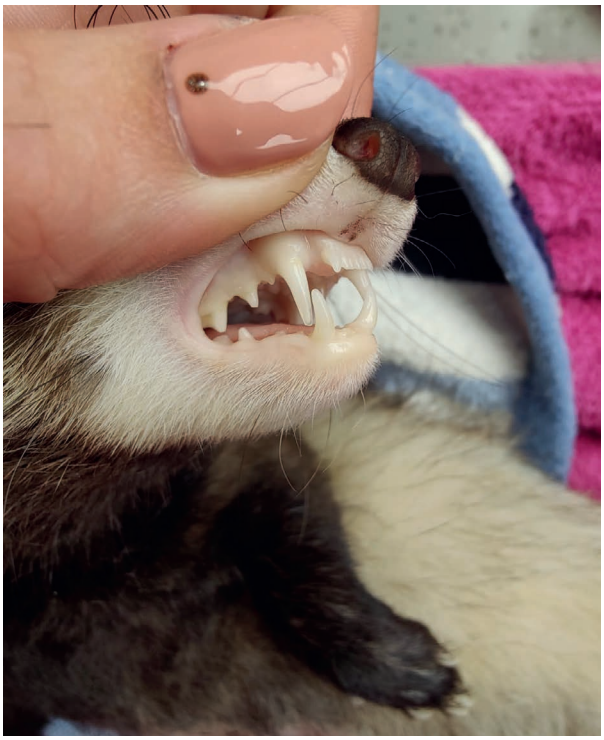


Figure 10.29b White appearance to oral mucous membranes.



Figure 10.29d Associated fur loss due to hyperestrogenism.

Table 10.10 Packed cell volume prognostic indicator.

Packed cell volume (%)	Prognosis
>25	Good
15–25	Fair
<15	Guarded
<10	Fatal

Diagnosis

Diagnosis is based on signalment and haematology shows a non-regenerative anaemia, thrombocytopenia, and leukopenia. *The normal haematocrit in ferrets ranges between 46% to 61%, which is much higher compared to dogs and cats*. Haematocrit can be used as a prognostic indicator (see Table 10.10).

Treatment

Treatment: With a packed cell volume (PCV) of at least 25% then administration of hCG (Chorulon, MSD) or GnRH (Receptal, MSD) should bring the jill out of season within 1 week. No further treatment will be required but the use of a deslorelin (Suprelorin, Virbac) implant in the autumn should be recommended to prevent further seasons.

> Deslorelin should not be administered to bring the jill out of season if she is already showing clinical signs of hyperestrogenism due to the initial hormonal surge resulting in another 2 weeks of oestrus.

For lower PCV between 15% and 25% more intensive treatment will be required. hCG (Chorulon, MSD) should be administered to bring the jill out of season (*the high volume required using GnRH (Receptal, MSD) should not be given as in my opinion putting 5 ml IM into a potentially thrombocytic patient is inappropriate even if given in multiple sites*) but it is highly likely with any PCV <20% a blood transfusion will be required.

Conveniently, ferrets do not have blood groups. A large male makes a good blood donor and severely affected jills may require multiple transfusions. Jekl and Hauptman (2017) recommend that in a healthy ferret 0.8% of the body weight as a volume of blood can be taken, obtaining blood from the cranial vena cava (under anaesthesia) and immediately mixing the blood with sodium citrate (ratio 6:1) then slowly administering to the recipient. Personally, I use the jugular for blood draws this size.

Where severe neutropenia is present, prophylactic antibiotics, for example clavulanate amoxicillin 25 mg/kg q8–12hrs, should be considered. *For jills with PCV less than 15 % the prognosis is grave despite intensive care*.

Pregnancy toxaemia

Pregnancy toxaemia is a life-threatening metabolic disease, also seen in several other species including guinea pigs and sheep. It occurs sporadically, with late gestation in young primiparous jills and those with large litters being overrepresented.

The condition is caused by a negative energy balance which can be caused by owners changing to an unfamiliar diet (resulting in anorexia), or in the case of very large litter, inadequate space for stomach distention post-feeding leading to less food being taken (Batchelder *et al*., 1999). Negative energy balance causes abnormal energy metabolism resulting in hyperlipidaemia, hypoglycaemia, ketosis, and hepatic lipidosis.

Clinical signs and diagnosis

Clinical signs include acute onset, severe lethargy, anorexia, weight loss, dehydration, excess coat shedding, +/– diarrhoea, melena, distended abdomen, and sudden death.

Diagnosis is based on signalment, biochemistry will show hypoproteinaemia, azotaemia, hypoglycaemia, raised bilirubin, raised liver enzymes and ketones. Anaemia will also be present. The more severe the results the poorer the prognosis.

Treatment

If the jill is to survive, aggressive therapy must be given. Intravenous fluids (maintenance is 60–100 ml/kg/day) and supportive feeding such as EmerAid Carnivore Care or Oxbow Carnivore Care should be given.

Gastric ulceration is common so gastro protectants such as administering famotidine (2.5 mg per ferret once daily), bismuth subsalicylate (Pepto-Bismol® 17.5 mg/kg twice daily) and sucralfate (75–125 mg/kg two to three times daily) administered 30 minutes before food or famotidine as needs acidic pH to work. In the UK sucralfate can be easily sourced from compounding companies. I obtain sucralfate from Summit.

Once stable a caesarean section should be performed to remove the energy drain on the jill. Kits under 39 days gestation are unlikely to survive. Post-operatively agalactia is common and cross fostering of jills should be considered.

Prognosis is guarded to grave. *Prevention is better than cure and owners should be counselled to provide a familiar good quality nutrition throughout gestation, minimise stress and ensure constant access to water (jills will not eat enough if they cannot drink)*.

Agalactia

Although not common in the pet ferret, recognised causes include pregnancy toxaemia, any concurrent infectious diseases (e.g. acute mastitis or pneumonia), a cold environment, poor-quality nutrition, or interruption to water supply.

Providing the kits are warm they can be successfully fostered onto another nursing jill (N.B. see Mastitis section). *Jills will not nurse cold kits*. Hand rearing under 10 days of age is extremely difficult in ferrets due to their reliance on ferret milk before this age. *After 10 days then kitten milk with added animal fat to increase the fat content can be used*. The kits must be stimulated to pass urine and faeces as per pups and kittens.

Mastitis

Mastitis can present peracutely with septicaemia and gangrene can set in within a few hours. Acute mastitis and chronic infection can also occur. Jills should be checked regularly for the typical signs of mastitis as per more familiar species: firm, painful swellings involving one or more mammary glands, associated colour changes to the overlying skin, and if expressed these glands may produce pus or blood contaminated discharge.

Antibiotic choice should be guided by bacteriology culture and sensitivity, but first line treatment with amoxicillin-clavulanate (25 mg/kg q8–12 hrs and metronidazole (15–20 mg/kg q12hrs) can be started while waiting for the results. Analgesia, fluid therapy and nutritional support should all be provided. If evidence of gangrene, then surgical excision of the affected gland/s should be performed. Famotidine (2.5 mg per jill once daily) should be administered during the time of healing as ferrets are extremely prone to stress-induced gastric ulceration.

Fostering of the kits onto another jill should ideally be avoided, due to the high risk of the kits transferring infection to the foster jill. If the kits are over ten days old, they can be hand reared, if they are younger, if possible, leave with the maternal jill and supplement feed with enriched kitten milk while still allowing them to suckle from the unaffected glands. *Affected jills should not be bred from again*.

Pyometra

Pyometras are uncommonly seen in pet ferrets but may be associated with hyperestrogenism. *Clinical signs are similar to those seen in bitches*. Adrenal gland disease or retained ovarian tissue can result in stump pyometras.

Diagnosis

Diagnosis can be confirmed using ultrasound. Blood should be taken for biochemistry and haematology to check for any evidence of bone marrow suppression and concurrent organ disease. *Owing to the effect that isoflurane or sevoflurane has on the redistribution of red blood cells, blood should be at least attempted to be drawn without anaesthesia and if unable to obtain, the effect needs to be taken into account when interpreting*.

Treatment

Surgical intervention is indicated. Intravenous fluids should be started, maintenance fluid requirement is considered to be 60–100 ml/kg/day and any deficits should be factored in. Systemic antibiotics, such as amoxicillin-clavulanate 25 mg/kg q8–12hrs, and analgesia meloxicam (0.2 mg/kg) can be administered peri operatively by subcutaneous injection followed 24 h later by 7 days oral administration. Famotidine (2.5 mg per ferret once daily) should be given alongside meloxicam as a gastric protectant.

Neoplasia in the jill reproductive tract

Ovarian and uterine neoplasia has been described in the jill but it is very uncommon. I have seen one case in 25 years of owning/working with ferrets. The ovarian mass was extremely mobile in this case and on presentation, was palpated beside the bladder!

Reproductive control in the jill

It is *vitally important that any jill that the owner does not want to have young is brought out of season*. Hyperestrogenism will result in death in up to 40% of unmated jills. Several options are available, and each has their pros and cons.

Vasectomised hob

Mating to a vasectomised hob can be used to bring a jill out of season. This will result in a false pregnancy which will last for 42 days. If this 'sham mating' has occurred early in the season, it is possible the jill may come back into season and will require another sham mating.

Jills during breeding season may be more irritable than normal. This can disrupt the human–animal bond. *Owners should be counselled on the aggressive nature of ferret mating as many novice owners will not be familiar with it*. The increase of owners keeping pet ferrets indoors is also likely to increase owners' concerns regarding the mating behaviour of ferrets.

Vasectomy

In my opinion, the easiest time to perform this procedure is at the start of the hobs breeding season. The testicular size increases therefore the associated structures increase in size with the increased circulating level of testosterone. The vast majority of cases are easy to perform as long as we keep calm and recognise that the vas deferens must be *inside* the vaginal tunic.

Pre-emptive analgesia should be administered. I use meloxicam 0.2 mg/kg SC.

Technique

A pre-scrotal incision is made, either a single one midline or two parallel incisions depending on the surgeon's preference. The vaginal tunic is identified and carefully opened. Inside the tunic there is a large vascular bundle and the vas



Figure 10.30 The vaginal tunic has been opened and the vas deferens is isolated.

deferens. Care should be taken not to damage the vascular bundle as the testicle still needs to function. The vas deferens can be identified as a white line approx. 2 mm wide with a blood vessel running parallel bilaterally (Figure 10.30). Remove 1 cm and ligate either end of the stumps using Polyglycolic acid (Vicryl) to prevent recanalisation.

The removed sections can be submitted for histopathology to confirm. Closure can be performed using intradermal 1.5m (4/0) Polyglycolic acid (Vicryl). Ferrets can bruise significantly postoperatively which can be alarming to owners (Figure 10.10). The hoblet should not be used for 7 weeks postoperatively to ensure degeneration of any viable sperm.

Ovariohysterectomy

Previously it was believed that the age of surgical neutering in ferrets was a predisposing factor to the development of adrenal gland disease (AGD). It has now been shown that should a ferret be surgically neutered it will develop AGD should it live long enough (Wagner *et al.*, 2009).

The incidence of AGD is much higher in the USA, this is likely to be due to a combination of factors, such as early age of neutering (they are spayed at 5 weeks of age), genetics (many ferrets in the USA come from the same genetic stock sourced from Marshall Farms), and higher number of owners keeping their ferrets indoors.

AGD is a multifactorial condition including surgical neutering, genetics, and increased exposure to long day length.

In time there may be other factors that become apparent. Given the fact we have no impact on the genetics (our clients already have their ferret), and that many more people keep their ferrets indoors therefore are likely to be exposed to artificially increased daylength, the one factor we do have control over is advice regarding surgical neutering.

Currently I advise against surgical neutering unless there is a medical reason to perform it such as a uterine mass. If owners insist on surgical neutering despite being informed of the risks I insist on a disclaimer form being signed. This states that the owner is aware of the increased risk of AGD, they are aware that surgical neutering is against medical advice and that they accept all financial and legal responsibility should their ferret develop AGD. Many rescues may be reluctant to implant due to a combination of the implant not being permanent (potentially allowing owners to breed) and the costs. Currently many rescues neuter but advise new owners to implant the ferrets to prevent AGD.

Human chorionic gonadotropin

It should be noted that human chorionic gonadotropin (hCG) (Chorulon, MSD) treatment is not licensed in the UK. An intramuscular injection of 100 IU given on day 10 after vulvar enlargement, which should induce ovulation approximately 35 h later (Mead *et al.*, 1988). The injection should be repeated 1 week later if vulvar turgidity fails to recede. hCG induces antibodies which may decrease the effectiveness over time. The owner should be aware that repeated doses of hCG increases the risk of anaphylaxis and has also been linked with an increased risk of ovarian neoplasia (Fox & Marini, 2014).

hCG is my drug of choice in jills who need to be brought out of season. Instead of using the full amount of diluent to dissolve the vial of drug I only use 1 ml. *The drug still dissolves fully but I only need to inject 0.07 ml IM instead of 0.33 ml.*

GnRH injection

It should be noted that this treatment is not licensed in the UK. An IM injection of 20 μg on day 10 after vulvar enlargement, which should induce ovulation approximately 35 h later in 95% of cases, followed by a false pregnancy (Schoemaker, 2003). The injection should be repeated 10 days later should vulvar turgidity fail to recede. Owners should be advised of the risk of anaphylaxis although premedication with an antihistamine has been shown to reduce this (Schoemaker, 2003). *The volume of GnRH required is much greater than hCG, which necessitates administration in multiple sites*.

Deslorelin (Suprelorin, Virbac) implant

In the UK neither the 4.7 mg or the 9.4 mg implant is licensed for use in the jill. It is only the 9.4 mg implant that is licensed for use in hobs.

I use isoflurane to sedate the ferret prior to implant. Wrapping in a towel burrito, allowing four breaths of 100% oxygen to saturate the blood then turning to 5% isoflurane results in the ferret being asleep in approximately 30–60 seconds with minimal distress. Ferret skin is thicker than expected for such a small mammal and the needle is quite large. Wound glue is then used to close the skin to prevent migration of the implant back out which I have anecdotally heard of on several occasions.

If the jill is implanted in anoestrus for example November in the northern hemisphere, the implant should not result in the jill coming into season and, in my experience, can result in the 4.7 mg implant lasting for up to 4 years. It is common for the first season after the implant wears off to be infertile however subsequent seasons will show normal fertility.

If the jill is implanted during breeding season, the owners should be advised that there is often an initial hormonal surge resulting in the jill coming into a fertile oestrus. The jill must be kept separate from hobs at this time as if mated she can carry the pregnancy to term, but she is likely to mismother the litter. Left unmated the season will normally only last two weeks and no further action need be taken.

The implant will decrease the musky odour ferrets are famous for.

Reproductive control in the hob

Suprelorin implant

This is a GnRH agonist that suppresses testosterone concentrations leading to reductions in testis volume and spermatogenesis. Neal *et al*. (1977) and Schoemaker *et al*. (2008) demonstrated a positive correlation between testicular size and testosterone concentration. The 4.7 mg implant can last on average for three years in my experience. Therefore, *owners can monitor testicle size as an indicator as when they should repeat the implant*. The implant resulted in: a decrease in the musky odour, decrease in aggression between males, and an increase in play behaviour compared to surgically neutered ferrets (Vinke *et al*., 2008). *Given that play behaviour is used as an indicator of good welfare, potentially an argument could be made that the implant is good for ferret welfare*!

Castration

Surgical castration is only advised if there is a medical reason such as testicular tumours or cryptorchidism. The rationale is the same as for surgical neutering in the jill. The technique is the same as in dogs.

Cryptorchidism

The testis descends into the scrotum at 10 weeks of age. Bodri (2000) reported an incidence of 0.75%, therefore it would be fair to conclude it is a rare finding. Owing to the fact that the retained testicle commonly becomes neoplastic in older ferrets, then surgical castration or at least removal of the retained testicle is recommended together with deslorelin implantation.

Preputial tumour

These commonly present as a blue/grey mass of varying size around the entrance of the prepuce and subcutaneously in the region of the prepuce, which are often tumours of the apocrine glands of the prepuce. Approximately 75% of these masses are malignant and they can metastasise to local lymph nodes. Pulmonary metastasis may occur.

Cystic dilatations of apocrine glands are also seen, and, in these cases, excision is curative (see Figure 10.23). *Visually these cannot be differentiated from adenocarcinomas so either FNA or excisional biopsy is recommended to allow for surgical planning* (see Figure 10.24).

Surgery can be difficult depending on the size. Further surgery may be required depending on histopathology including a salvage procedure such as surgical amputation of the prepuce, penis, and a urethrotomy.

Return of sexual behaviour in the neutered ferret

Sprite

OVARIAN REMNANT SYNDROME

The ovary is encapsulated in a fatty bursa, which completely encloses the ovary in the ferret. During surgical neutering it is *important not to enter the bursa* to prevent leaving part of the ovary behind. In the UK should surgical neutering occur it usually occurs at around 6 months of age. Compare this to the USA where surgical neutering is commonly performed at 6 weeks or younger. Spaying at this young age increases the risk of leaving part of the ovary behind (Fox & Marini, 2014).

Clinical signs include vulval swelling in neutered jills, and should the oestrogen levels be high enough then bone marrow suppression may result.

Treatment includes uses of Suprelorin implant, hCG (Chorulon, MSD), GnRH (Receptal, MSD), or exploratory surgery during breeding season to identify and remove the remnants.

A differential diagnosis is AGD. *As an adrenal panel measures sex hormone production, it will not differentiate between AGD and ovarian remnant*. Ultrasound of the adrenal glands may help differentiate.

Adrenal gland disease: sprites and hobbles

The production of the sex hormones leads to the clinical signs associated with AGD: symmetrical ascending alopecia (Figure 10.31), vulva swelling in neutered jills, recurrence of sexual behaviour in neutered hobs, pruritus, prostatic enlargement (can lead to urinary obstruction), anaemia seems to be rare in jills, stump pyometras, and infected prostatic cysts may also be present. Polyuria/polydipsia is reported with AGD but it is advisable to check for concurrent renal disease. It is seen mainly in ferrets four years and older. Differential diagnosis include ovarian remnants, food allergy (rare) in cases of severe alopecia and pruritus, and seasonal tail alopecia.



Figure 10.31 Symmetrical ascending alopecia seen in a case of AGD.

Diagnosis

Diagnosis is mainly based on the clinical signs, however, ultrasound of the adrenal glands can be used to confirm. If AGD is present then Kuijten *et al.* (2007) demonstrated the *gland will have an increase in thickness, generally measuring over 3.9 mm*. The gland will also be more rounded in appearance, heterogeneous structure, and an increase in echogenicity.

Interestingly, there is no atrophy seen in the non-neoplastic contralateral adrenal gland, and the condition may be (or become) bilateral. Chen (2010) demonstrated that at least two of oestradiol, androsterone and 17a-hydroxyprogesterone will be elevated in affected ferrets, however *they will also be elevated in jills with ovarian remnants*.

Treatment

Surgery used to be the treatment of choice which could pose a challenge if the right adrenal gland was affected due to its close position to the caudal vena cava (Figure 10.32).

Schoemaker (2017) reported that no indication of prognosis can be given based on the histopathology. However, Lennox and Wagner (2012) found that the relapse times for ferrets' treatment medically with deslorelin (Suprelorin) was longer than those treated surgically. Therefore, we can consider Suprelorin as a first line treatment for AGD in sprites. The study did show the implant may have little effect on the actual growth of the tumour but did increase quality of life for longer.

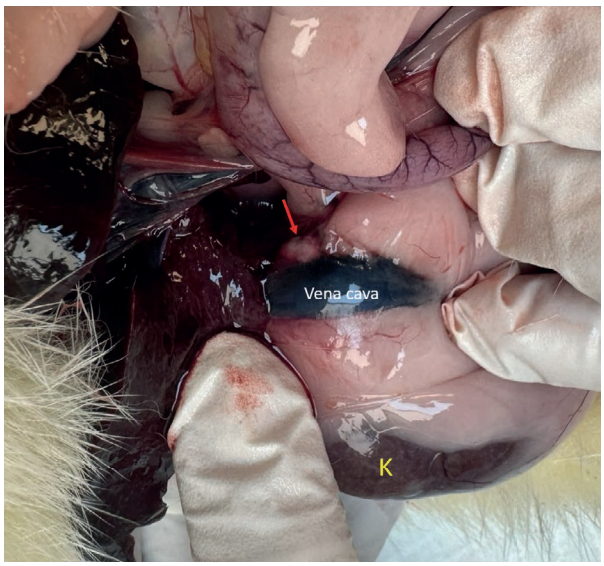


Figure 10.32 The yellow K indicates the right kidney and the red arrow highlights the right adrenal gland which is slightly larger than normal. Note how close the adrenal gland lays to the caudal vena cava. It is understandable why surgery is challenging when the right adrenal gland is affected.

Suprelorin down-regulates the GnRH receptors, which stops the release of gonadotropins. Most ferrets will show a complete to partial response within 1 month. Most will show improvement within 2 weeks. The length of time the implant lasts is dependent on the type implanted but the 4.7 mg should last at least 1 year and in my experience, lasting 2–3 years is common. *Some adrenal gland tumours become autonomous, no longer relying on stimulation from the brain to produce sex hormones, meaning the implant will no longer be effective*.

A non-surgical alternative to Suprelorin is monthly IM injections of the GnRH agonist leuprolide acetate. Results seem to be more consistent with the 30-day depo formulation of leuprolide. Ferrets weighing less than 1 kg should be given 100 μg, and ferrets more than 1 kg should receive 200 μg every 4–6 weeks. Over time a higher dose may be required to adequately control clinical signs. The injection can be painful and cause local irritation. The owner should be counselled that it is unlikely that this therapy will reduce the size of, or prevent the growth of, the adrenal gland tumour. Some ferrets have achieved adequate control of clinical signs for over two years.

Prostate disease

The prostate is the only accessory sexual gland in the hob. It surrounds the urethra at the base of the urinary bladder.

In the USA prostatic disease is the leading cause of urethral obstruction in middle aged or older hobbles and is most frequently seen in association with AGD, along with the other relevant clinical signs of AGD.

Prostatic disease includes prostatomegaly, prostatic cysts and prostatic abscesses. Owing to the position of the prostate, enlargement can result in occlusion of the urethra and can present as a blocked bladder. *Owners may misinterpret straining to pass urine as straining to pass faeces and the ferret may vocalise during the process*. An enlarged prostate may be palpated as a fluctuant swelling caudal to the bladder but not all prostatic issues will present as prostatomegaly. Differential diagnosis of obstruction of the urethra includes uroliths or pyuria.

The prostate communicates with the bladder and purulent material can induce a bacterial cystitis. Diagnosis can be achieved by ultrasound and *catheterisation can be difficult to achieve due to prostatic compression of the pelvic urethra*.

Definitive diagnosis and treatment involve exploratory laparotomy. *Owing to the potential of an initial hormonal flare up, deslorelin implant should be used with care in cases of urethral obstruction* (Quesenberry *et al*., 2021). Adrenalectomy or debulk is considered the surgical treatment of choice in cases of AGD induced prostatomegaly. This may be used in conjunction with deslorelin implant.

Alternatively, medical treatment with osaterone acetate (Ypozane, Virbac) may be given to shrink the prostate, as there is no established dose in ferrets I use the licensed canine dose. Once the prostate has reduced in size a deslorelin implant may be placed.

Orcutt (2003) describes good surgical success treating prostatic abscessation using the technique of marsupialisation to the ventral abdominal wall. This allows drainage of the septic content, flushing, reduction in risk of peritonitis, and the stoma will heal by secondary intention. Ideally the prostate should be biopsied, and tissues send for culture and sensitivity. Antibiosis should be started while waiting on the lab results and examples of suitable antibiotics include enrofloxacin 10 mg/kg q12hrs, trimethoprim sulfadiazine 30 mg/kg q12hrs, chloramphenicol 50 mg/kg q12hrs, clindamycin 6–10 mg/kg q12hrs (Orcutt, 2003). Analgesia should be provided.

Neoplasia

Ferrets are extremely prone to neoplasia and are more commonly diagnosed in ferrets over three years of age. *It is very common (up to 20% of ferrets) to have multiple concurrent types of tumours*, up to four different types of concurrent tumours in a single ferret have been documented. *Interestingly, the clinical behaviours and prognosis of neoplasms in ferrets are often different from the same neoplasms seen in dogs and cats*.

Endocrine neoplasia

Endocrine neoplasia includes insulinoma (see endocrine disorder) and AGD (see Reproductive disorders section).

Intestinal neoplasia

Intestinal lymphoma will often blend macroscopically imperceptibly with surrounding tissue, whereas adenocarcinomas tend to form discrete masses (Johnson-Delany, 2010) (see Intestinal neoplasia in Gastrointestinal section).

Lymphoma/sarcoma

Lymphoma is the most common malignant neoplasm in ferrets and can be seen at any age. Outbreaks of lymphoma in colonies of ferrets has led to the suggestion that a retrovirus may be responsible for some cases but as of yet a viral cause has not been identified. Although B-cell lymphoma is seen, T-cell lymphoma is the predominant type of lymphoma seen in ferrets.

Presentation and organ distribution of lymphoma has been associated with the age of onset, mediastinal lymphoma is more prevalent in young ferrets and is sometimes termed juvenile lymphoma whereas ferrets 3 years old (or older) have a variable presentation with multicentric disease being more prevalent and is sometimes termed adult/chronic lymphoma.

Juvenile lymphoma, which affects ferrets less than 2 years of age, presents acutely and is rapidly progressive and fatal; and adult/chronic lymphoma affects animals over three years old and is slowly progressive with clinical signs often waxing and waning.

Clinical signs

Clinical signs are extremely variable depending on the body systems involved. Anorexia, chronic weight loss, internal and/or peripheral lymphadenopathy, splenomegaly (see splenomegaly), reduced thoracic compliance (mediastinal/thymic), dyspnoea (pleural effusion, mediastinal/thymic), cough (mediastinal, thoracic lymphadenopathy), diarrhoea (intestinal), cutaneous lesions (see dermatology), large kidneys, peritoneal effusion, and exophthalmos (retrobulbar lymphoma, see ophthalmology).

Investigation

Haematology commonly shows a non-regenerative anaemia, whereas marked lymphocytosis and thrombocytopenia are uncommon. Biochemistry results are inconsistent in lymphoma cases, but hypercalcaemia may occasionally be seen.

Thoracic radiographs may show masses in the cranial mediastinum (Figure 10.33), or bone changes. Ultrasound guided FNA of mediastinal masses may help with diagnosis.

Ultrasound examination can be used to visualise the spleen and liver, and pleural and abdominal effusions. Areas of concern showing as mottling and hypoechoic lesions within the spleen or viscera. It should be noted that ultrasound examination of the spleen cannot differentiate between hypoechoic areas due to extramedullary hematopoiesis and hypoechoic areas due to lymphoma infiltration.

Enlarged lymph nodes are frequently seen during the abdominal ultrasonography examination but they are frequently enlarged due to chronic inflammation either due to *Helicobacter mustelae* or inflammatory bowel disease, *therefore presence of enlarged abdominal lymph nodes should never be considered as evidence of lymphoma*. Unfortunately, *chronic gastrointestinal inflammation results in changes in abdominal lymph nodes that are almost indistinguishable from lymphoma therefore there is no point in performing a biopsy of abdominal lymph nodes*. Peripheral lymph nodes are less likely to be affected by local inflammation and so are suitable for biopsy.

Diagnosis

Diagnosis requires histopathology of affected tissue and immunophenotyping (B- or T-cell) is advised. *Biopsy is preferred over FNA*. Immunophenotyping is important as B-cell lymphoma when treated with chemotherapy in general has a longer survival time compared to T-cell lymphoma (8.8 months versus 4.3 months (Ammersbach *et al.*, 2008)).

A standardised classification scheme was proposed (Mayer & Burgess, 2012) with three descriptors being recommended for each case: clinical stage, histological evaluation (grade), and immunophenotyping.

Staging lymphoma as defined by:



Figure 10.33 Dorsoventral radiograph showing a widened mediastinal space, confirmed as lymphoma on FNA.

Stage 1: single anatomic lesion (nodal or extranodal).
Stage 2: single lesion with regional lymph node involvement limited to one side of the diaphragm.
Stage 3: lesions on both sides of the diaphragm including intra-abdominal or gastrointestinal locations.
Stage 4: multiple sites on both sides of the diaphragm are affected with or without the visceral organs.
Stage 5: manifestation in the blood and involvement of bone marrow and/or other organ systems.
Subclassification: (a) without clinical signs; (b) with systemic signs.

Treatment

Treatment is generally aimed at improvement in their clinical signs and quality of life. *Treatment is likely to involve combinations of chemotherapy, surgical tumour removal, radiation therapy*. Owners of ferrets that have been on steroid therapy for concurrent illness, e.g. insulinoma, should

be counselled that the lymphoma may not be as sensitive to steroid therapy.

A formula for chemotherapeutic drug calculation based on ferret body surface area, 9.94 × (body weight) ⅔, was developed but it is so similar to the feline body surface area formula, 10 × (body weight) ⅔ that either formula was deemed acceptable (Jones *et al*., 2015).

There are many examples of chemotherapy protocols in the literature and there is no conclusive evidence that any one treatment is better than another. Ammersbach *et al*. (2008) found ferrets that received chemotherapy who had T-cell lymphoma survived on average 4.3 months (range 0.5–14.0), and those treated who had B-cell lymphoma survived on average 8.8 months (range 2.0–19.0). Ammersbach *et al*. (2008) also looked at mean survival if no treatment was given for ferrets with T-cell lymphoma which was 5 months (range 0.5–14.0) and for ferrets with B-cell lymphoma it was 8.4 months (range 2–19 months), which was very similar survival lengths to those ferrets treated with chemotherapy. *Schoemaker (2017) stated it was debatable whether chemotherapy provides any added value in the treatment of lymphoma in ferrets*.

Webb *et al*. (2019) reported that the 36 ferrets treated with multiagent therapy had a mean survival time of 4.13 months (range 0.36–40 months). The study looked at 44 cases, the 10 different therapies utilised and noted that only two modified chemotherapy protocols (cyclophosphamide, vincristine (known as Oncovin) and prednisolone (COP) and Tufts no-IV) had published chemotherapy schedules. They stated, 'with this wide variation, statistical comparison of chemotherapy treatment protocols would be inappropriate and is therefore not provided' (page 66).

Fifteen ferrets treated with modified COP (Table 10.11) with or without additional chemotherapeutics had a mean survival time of 14.3 days (range 1.2–40 months). *Ferrets will require sedation for IV placement before IV chemotherapeutic drugs are administered*.

The mean survival time for the seven ferrets that received the Tufts no-IV (see Table 10.12) protocol was 2.86 months (range 0.63–29.6 months). This protocol relies on the compounding of the oral chemotherapeutic drugs, if a tablet form is not available, liquid oral medications must be administered in the clinic and not by the owners.

Prednisolone may be used as a single agent for palliative chemotherapy 0.5–1 mg/kg q12hrs, Webb *et al*. (2019) reported one ferret with small cell lymphoma treated with prednisolone alone was immunophenotyped T-cell lymphoma and survived 24.8 months, although four other ferrets with large cell lymphoma treated with prednisolone alone had survival times of 3, 8, 16 and 179 days.

Just like more familiar species, adverse effects of chemotherapy in ferrets are gastrointestinal damage, myelosuppression, and loss of fur. Haematology should be checked as recommended in the relevant tables. Neutropenia often manifests as diarrhoea in affected

Table 10.11 Modified chemotherapy protocol.[a]

Drug	Dose	Route	Frequency
L-asparaginase	400 IU/kg	IM	Once[b]
Prednisolone	1 mg/kg	PO	q12–48h[c]
Vincristine	0.12 mg/kg	IV	13 weeks[d]
Cyclophosphamide	10 mg/kg	PO or SC	3 weeks

Notes: [a]CBC evaluated weekly; [b]administer 3 days prior to week of COP; [c]administer q12h weeks 12, q24h weeks 36, q48h weeks 752; [d]administer weekly for weeks 13 then every 3 weeks.

Table 10.12 Tufts no-IV chemo ferret.[a,b]

Drug	Dose	Route	Weeks administered
Prednisolone	2 mg/kg	PO	All
L-asparaginase	10,000 IU/m^2	SC	1–3
Cyclophosphamide	250 mg/m^2	PO	1, 5, 9, 13, 18, 23
Cytarabine	300 mg/m^2 [c,d]	SC	3, 11, 20
Methotrexate	0.8 mg/kg	IM	7
Chlorambucil	1 tab[d]	PO	11, 20
Procarbazine	50 mg/m^2	IM	15, 26

Notes: [a]CBC evaluated weeks 2, 4, 7, 8, 12, 16, 17, and 27; [b]repeat weeks 20–26 for three cycles if not in remission; [c]administer 2 days in a row on week 3; [d]administer 2 days in a row on weeks 11 and 20.

animals; antimicrobials and gastro protectants may be required for severely neutropenic animals alongside a change in chemotherapeutic drug. Nausea can be treated with metoclopramide or maropitant citrate.

Good nutrition is vitally important to help mitigate cachexia, many ferrets enjoy EmerAid Carnivore and will readily take it from a bowl.

More work is required before recommendations can be made for one protocol over another. Ultimately the decision made will depend on the nature of the ferret (whether treatment is a feasible option or does the animal find handling for medicating incredibly stressful), financial resources, and the owner's previous experiences or personal beliefs about chemotherapy.

Neurology

It should be remembered that in ferrets virtually any condition can result in hind end weakness *which may mimic neurological disease*.

Primary neurological disease is rare in ferrets. Neurological clinical examination of ferrets is very similar to more familiar species with the exception that most ferrets have a diminished or absent menace reflex.

Clostridium botulinum

Hunting ferrets, or ferrets fed raw food are at an increased risk of botulism and reportedly ferrets are less resistant to the endotoxin than dogs.

Clinical signs occur 12–96 h after eating contaminated food: dysphagia, drooling, ataxia, protrusion of the third eyelid, and paresis. An ascending flaccid paralysis starting from the hindlegs occurs, which leads to shallow respiration due to partial paralysis of the intercostal muscles and diaphragm. Death occurs due to respiratory failure.

Diagnosis is presumptive based on history and clinical signs although identification of the botulinum toxin in food, blood, serum, or gastrointestinal contents allows definitive diagnosis.

Treatment may be attempted by administering type C antitoxin (antitoxin will not be effective once paralysis has occurred) and giving supportive care. With supportive care, recovery from paralysis is possible but may take 2 weeks or more. Ferrets may require urinary catheterisation and intensive nursing depending on the severity.

Gastrointestinal disease

Ferrets are obligate carnivores with a short digestive tract and no caecum. Their intestinal length is only five times their body length (compared to the cat's intestinal length of approximately 8–10 times their body length); this means that *their digestion is relatively inefficient and relies on small, frequent meals of high protein and fat content*.

Their gut transit time is approximately 2.5–3.5 h; as such, they should not be starved for as long as dogs and cats before surgery, as this holds a real risk of inducing hypoglycaemia, especially in older ferrets where subclinical insulinoma is common. The recommended time for starving before surgery is a maximum of four hours. As they are *prone to developing stress-induced gastric ulceration*, preemptive treatment with famotidine (2.5 mg) once daily to reduce gastric acid secretion should be considered several days before any elective surgery. A ferret's short gut transit time also means that any gastrointestinal (GI) disturbance quickly becomes apparent. It also means that *they can dehydrate and deteriorate quickly*.

What are normal faeces like?

Owing to their inefficient digestion and rapid gut transit time, their normal faeces are soft, but formed (Figure 10.8). Ferrets with anorexia will often have scant green tinged faeces due to the fact that the whole intestinal tract can secrete cholecystokinin, the hormone responsible for bile secretion.

Diarrhoea

Approach

The initial approach to a ferret with diarrhoea will be the same regardless of the inciting cause and is very similar to more familiar species.

A full clinical history should be taken, including:

- age (this may help shorten differential list) (see Table 10.13)
- diet (all treats, scraps, and any changes)
- when and where the ferret was obtained
- how many ferrets do they have and whether any others are affected
- has there been exposure to new ferrets
- types of toys given
- vaccine status
- exposure to toxins or drugs
- any illness in the in-contact humans (e.g. influenza, SARS CoV-2, diarrhoea)
- any vomiting or pawing at the mouth, its timing (e.g. after stressful incident) and appearance of vomitus, the consistency, colour and appearance of any diarrhoea, including any tenesmus or vocalisation (note that diarrhoea is difficult to classify as large intestinal or small intestinal unlike in dogs)
- has there been any progression of the condition
- has there been any weight loss (*this can be complicated by the fact that it is physiologically normal for ferrets to gain up to 40% of their body weight during the autumn and winter and lose weight during spring and summer*).

Table 10.13 Differential list by age.

Age of ferret	Conditions seen
<1 year old	Foreign body (common, occasionally seen in older ferrets) Proliferative bowel disease (rare) Rotavirus (rare)
<1.5 years old	Ferret systemic corona virus
2–4 years old	Aleutian disease
>4 years old	Inflammatory bowel disease (common) *Helicobacter mustelae* associated gastritis (common) Dental disease (common) Trichobezoars Gastro-intestinal lymphoma Gastro-intestinal adenocarcinoma
Any age	Dietary Indiscretion (common) Stress ulcers (common) Bacterial food poisoning Ferret enteric corona virus (epizootic catarrhal enteritis) Influenza Distemper

Depending on history and clinical exam findings, either further diagnostics or symptomatic treatment may be elected at this stage.

Symptomatic treatment

Mild dehydration can be corrected orally using a rehydration formula; however, many ferrets may not find these formulas palatable. Alternatively, a mix of highly palatable soft food such as Hill's Prescription Diet™ a/d Canine/Feline with warm water will often be readily consumed by ferrets.

Moderate or severe dehydration will require intravenous fluid therapy. Maintenance fluid requirement is considered to be 60–100 ml/kg/day. In hobs, a 22G IV catheter can easily be placed in the cephalic vein, while jills may require a smaller gauge (Figure 10.34). The catheter gauge should be judged on vein size rather than the size of the animal. Application of a topical EMLA 5% cream (2.5% lidocaine, 2.5% prilocaine, Aspen) may make placement easier. As *ferrets' necks are wider than their head it is not feasible to maintain a Buster collar in place*, but most ferrets will leave their bandages alone.

Intraosseous catheters can also be utilised for fluid therapy and can be placed as for puppies and kittens. It is the author's opinion that subcutaneous fluids should not be given to moderately or severely dehydrated ferrets due to their prolonged absorption times and pain associated with administration.



Figure 10.34 Placing a catheter in the cephalic vein.

Anorexic ferrets should be syringe fed using EmerAid Carnivore or Oxbow Carnivore Care. Supportive feeding should be calculated at a minimum of 400 kcal/kg body weight and split into four to six meals daily.

Vomiting can be controlled with maropitant 1 mg/kg SC q24hrs or metoclopramide 0.5mg/kg SC or orally q8hrs if a foreign body has been ruled out.

Ferrets suffering from chronic diarrhoea will benefit from vitamin B_{12} supplementation. For oral supplementation of vitamin B_{12} I use Cobalaplex, Protexin (half a capsule daily).

The cause of the diarrhoea may become apparent from the history as ferrets often develop diarrhoea due to dietary indiscretion as dogs and cats. In these cases, the diarrhoea should resolve within a couple of days with symptomatic treatment such as a specific anti-diarrheal probiotic paste. I use Pro-Kolin Advanced for cats 15 ml, Protexin. Any diarrhoea lasting longer than three days warrants further investigation.

Diagnostic options

Blood should be taken for a full biochemistry and haematology. *Gastrointestinal disease will frequently cause an increase of lipase, Alanine transaminase, Alkaline phosphatase, Gamma-glutamyl transferase and globulins. Therefore, liver disease should not be definitively diagnosed based on a single blood sample.*

Radiography and ultrasound can also be utilised to achieve a diagnosis. Faecal samples may be sent for

bacteriology, culture, and sensitivity. Faecal flotations for parasitology should also be performed.

Common conditions of the oesophagus

Megaoesophagus is recognised in the ferret but is rare and the cause is still unknown. Several cases have been described in older ferrets suggesting that this may be an acquired condition. Treatment of these cases is often unsuccessful, but the same principles apply as for managing the condition in dogs. *Metoclopramide and cisapride are not recommended, because these drugs work on smooth muscle and the entire oesophagus of the ferret is striated muscle*. As these drugs also work to tighten the distal oesophageal sphincter, they may actually slow the emptying of the oesophagus, potentially worsening the situation.

Foreign bodies may be seen in the oesophagus, albeit with less frequency compared to other parts of the digestive tract. These may be removed either endoscopically or surgically.

Common conditions of the stomach

Gastric ulcers

Gastric ulceration is commonly seen in ferrets and may be the result of stress or secondary to medication or concurrent disease (e.g. hypoglycaemia induced by insulinoma results in sustained stimulation of gastric acid secretion).

Possible stressors include dietary change, veterinary visits, overcrowding and addition of new animals. Gastric ulcers are rarely seen with corticosteroid use even at high doses (Fox & Marini, 2014). In contrast, non-steroidal anti-inflammatory drugs are known to result in ulcers if given at high or prolonged dosing, although these still remain valuable analgesics for ferrets when used appropriately (van Oostrom *et al.*, 2011).

Ulceration may be suspected based on blood results, but endoscopic examination of the gastric mucosa will provide more information. Endoscopy allows visualisation, mucosal biopsy and may allow retrieval of foreign bodies. The short gastrointestinal tract of ferrets means that endoscopy is relatively easy in this species. The small lumen size of the intestine limits the size of the endoscope that can be used. For ferrets weighing over 1 kg, an 8.6 mm gastrofiber endoscope can be used, whereas a 3 mm bronchofiber endoscope will be required in smaller ferrets. Full thickness biopsies cannot be obtained without laparotomy.

Ferrets are hardy creatures and *are good at masking pain. Gastric pain may show as tooth grinding*. Nausea can result in frantic pawing/clawing at the mouth as well as profuse salivation. Unlike dogs and cats, *ferrets spontaneously secrete stomach acid, so pain may not only be obvious at feeding time but can present at any time*. Owners may perceive the ferret as having oral pain if they demonstrate tooth grinding during eating. Clinical signs can also include weight loss, hypoproteinaemia, depression, diarrhoea and coughing or gagging due to gastric reflux. Bleeding from gastric ulcers may result in melaena and anaemia (Figure 10.35).

Treatment of gastric ulcers involve administering famotidine 2.5 mg per ferret q24hrs, bismuth subsalicylate 17.5 mg/kg q12hrs (Pepto-Bismol®), and sucralfate 75–125 mg/kg q8–12hrs. *Sucralfate should be administered thirty minutes before food or famotidine as an acidic pH is required for this to work effectively*. In the UK sucralfate can be easily sourced from compounding companies. I obtain sucralfate from Summit Veterinary Pharmaceuticals.

Helicobacter spp. infection may also play a role, although while *H. mustelae* is considered ubiquitous in the ferret population, there is *debate as to whether it is a causative agent or opportunistic* in the development of gastric ulceration. *H. mustelae* infection is lifelong and causes hypergastrinemia (Fox & Marini, 2014) and lymphoproliferation of the gastric mucosa which increases in severity as the ferret ages (Fox *et al.*, 1990). This results in an increased risk of developing gastric lymphoma (Fox & Marini, 2001; Lennox, 2001). Definitive diagnosis requires gastric biopsy showing both chronic damage and the causative organism, but given that infection is considered ubiquitous and that even if *H. mustelae* is not the causative agent, it will exacerbate opportunistically, then an argument can be made for presumptive treatment based on clinical signs.



Figure 10.35 Bleeding from gastric ulcers may result in melaena.

Treatment can be curative, but reinfection is common unless all in-contact ferrets are treated. Triple combination therapy is recommended, consisting of amoxicillin 30 mg/kg q12 hrs, metronidazole 15 mg/kg q12hrs, and bismuth subsalicylate 17.5 mg/kg q12hrs for a minimum of 21 days (Johnson-Delaney, 2010; Morrisey, 2013). Given the ubiquitous nature of *H. mustelae* and the fact it can act as an opportunistic organism, due consideration should be given to treating ferrets against *H. mustelae* if they are receiving immunosuppressive therapy.

Gastric bloat

Bloat occurs occasionally in ferrets and is mainly due to gastric outflow obstruction. It is rare to occur without obstruction.

Clinical signs usually have an acute onset, with the ferret presenting very weak to moribund with a dilated stomach. Radiography will confirm diagnosis. The *cause of the obstruction will impact prognosis*. A foreign body for example will have a better prognosis than a pyloric adenocarcinoma. Spontaneous gastric adenocarcinoma has been reported in ferrets (Sleeman *et al.*, 1995).

Surgical intervention is usually necessary, but ideally the ferret should be stabilised prior to surgery. Intravenous fluids should be administered at twice maintenance to counter shock. Gastric pressure can be relieved by passing an orogastric tube. Mask induction with isoflurane or sevoflurane will facilitate this quick procedure.

Foreign bodies

Gastric foreign bodies are more commonly seen in ferrets under 1 year old, but can be seen in older ferrets too. Trichobezoars are more commonly seen in older ferrets (>4 years) and can act as a gastric foreign body. Linear foreign bodies are rarely encountered in ferrets.

CLINICAL SIGNS

Strangely, *vomiting is not commonly seen with foreign bodies unless gastric outflow is obstructed*. Chronic weight loss, anorexia and intermittent diarrhoea are common clinical signs. Clinical signs often wax and wane. Similar to dogs, small gastric foreign bodies can remain in the stomach for a long time and cause intermittent gastric issues.

DIAGNOSIS

It may be possible to palpate the foreign body during the clinical exam. Restraining the ferret with your non-dominant hand across the thoracic girdle and allowing the rest of the animal to dangle will result in the abdominal contents becoming more accessible to palpation with your dominant hand.

Radiography with or without contrast may be required to definitely diagnose if not palpable. If the ferret is painful, then palpation under sedation may be required. Whole body radiographs should be obtained to allow evaluation of the oesophagus.



Figure 10.36 Peritonitis due to a foreign body causing rupture of the bowel. Photo: Dr Conor Chrichton.

TREATMENT

Foreign bodies should always be considered a surgical emergency in ferrets as they seem to be more prone to perforation and it is very rare for them to pass naturally (Figure 10.36). Careful exploration of the other organs should be performed at time of laparotomy due to the high occurrence of concurrent diseases (e.g. lymphoma, insulinoma, AGD) especially in older ferrets.

Biopsies should also be taken from the stomach, lymph nodes and intestine at time of surgery to rule out lymphoma or inflammatory bowel disease. This is especially important in cases of trichobezoars as chronic slowing of gastric emptying (due to chronic inflammation) has been implicated as a reason for these developing in the first place.

It should be remembered that *cervical manipulation during intubating the ferret can result in stimulation of the vagal nerve leading to an increase in the intestinal motility at the beginning of surgery (Johnson-Delaney, 2006),* which could increase the risk of perforation.

The most common sites for foreign bodies in descending order are stomach, jejunum, duodenum, and oesophagus. However, it is advisable to *check the entire digestive tract as it is not uncommon to have more than one foreign body* (Figure 10.37).

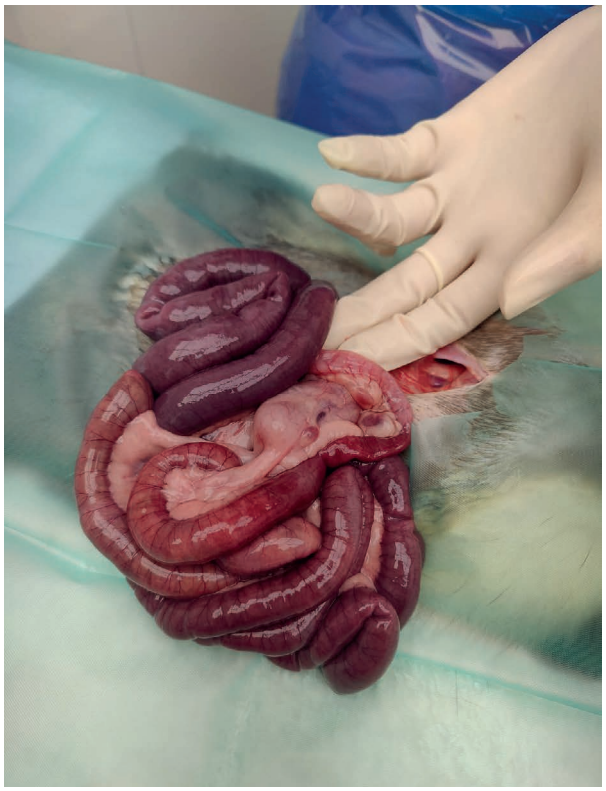


Figure 10.37 Ferret with two foreign bodies, both of which were foam ear plugs.

Post-operatively, moist food should be offered for 24–48 h. Most ferrets will eat within 12 h of surgery and I offer food within 1 h of full recovery from general anaesthesia. Some ferrets may require syringe feeding. Palatable options include: Hill's™ Prescription Diet™ a/d™ Canine/Feline, Lafeber's EmerAid Nutritional care systems Carnivore diet, Oxbow Animal Health Carnivore Care or soaking the ferret's own kibble to make a gruel.

Analgesics are an important component of post-operative care. Ferrets can appear stoical animals, but they do feel pain. Tramadol 5–10 mg/kg q12–24hrs (Lichtenberger & Ko, 2007; Jepson, 2015) can work well but is bitter. Buprenorphine 0.04–0.06 mg/kg q4–6hrs can be given by oral transmucosal route, may have less of a sedative effect. Buprenorphine should be administered for 3 days. Meloxicam 0.2 mg/kg q24hrs can be administered postoperatively by subcutaneous injection followed 24 h later by 7 days oral administration. Famotidine 2.5 mg per ferret q24hrs should be given alongside meloxicam as a gastric protectant (Keeble & Meredith, 2009). Sucralfate should be administered twice daily at least 30 minutes before food or an acid suppressor. Sucralfate has the added benefit of treating any reflux oesophagitis that may have developed.

PREVENTION

Prevention of further foreign bodies should be discussed with the owners. Regular use of cat hairball laxatives during shedding season may reduce incidence of trichobezoars, especially in Angora ferrets. Increased brushing by the owner may also help.

Access to any potential foreign bodies should be limited, for example: rubber handled hair brushes, hair bobbles, rubber gloves, remote controls, sponges. Owners should be educated on careful toy selection and to avoid rubber toys. Ferrets are incredibly inquisitive and seem determined to get into trouble at times, therefore close supervision of free roam playtimes is necessary.

Toxicosis

Owing to their curious nature, their ability to get into almost anything and their strong jaws, ferrets are prone to opening sealed containers. Their small size means even a small ingestion can mean a large dosage relative to their mass.

Initial approach will be the same as with dogs and cats by treating the clinical signs that the patient is exhibiting. Emesis should only be attempted if it would be appropriate in more familiar species. Apomorphine 0.04 mg/kg SC can be used to produce emesis.

Activated charcoal 1–3 g/kg may be appropriate depending on the toxin but ferrets should be closely monitored for secondary hypernatraemia which can occur due the shift of fluid into the gut.

Clinical signs include ataxia, tremors and seizures and can occur within 4 h of administration of activated charcoal. Treatment for hypernatraemia can include low sodium intravenous fluids such as 5% dextrose in water or plain water enemas. Warm water should be used to prevent hypothermia and 5–10 ml/kg is flushed into the rectum; water is absorbed quickly by the colon.

Sorbitol 3 ml/kg may be safely given as an osmotic cathartic with each repeated charcoal administration to facilitate the removal of the activated charcoal bound substance. *Sorbitol should not be given to dehydrated ferrets or if the ferret has diarrhoea.*

Ibuprofen toxicosis

Can occur as soon as 4–8 h after ingestion (65% will have clinical signs within 8 h of ingestion) and ferrets appear to be more sensitive to the toxic effects of ibuprofen than dogs. *The lowest dose resulting in death was 220 mg/kg which means one tablet could be fatal.*

Clinical signs and diagnosis

The target organs of ibuprofen toxicity are the CNS, gastrointestinal tract, and the kidneys. Renal papillary necrosis and gastrointestinal haemorrhage are commonly seen.

Clinical signs include weakness, ataxia, tremors, anorexia, vomiting, gagging, diarrhoea, melaena, polyuria/polydipsia, dysuria, and recumbency.

Diagnosis is presumptive based on history of exposure to ibuprofen (tablets, liquids, or topical gel) and clinical signs.

Treatment

Treatment options include the following.

- Emesis if asymptomatic and within 1–2 h of ingestion. Apomorphine 0.04 mg/kg SC can be used to produce emesis.
- Activated charcoal should be given 1–3 g/kg as there is enterohepatic recirculation of ibuprofen. This can be repeated but, the ferret should be monitored closely for secondary hypernatraemia (see above). Sorbitol 3 ml/kg may be safely given as an osmotic cathartic with each repeated charcoal administration to facilitate the removal of the activated charcoal bound ibuprofen. Sorbitol should not be given to dehydrated ferrets or if the ferret has diarrhoea.
- Gastrointestinal protectants should be given. Misoprostol 1–5 µg/kg PO q8hrs or sucralfate 125 mg PO q6hrs and famotidine 2.5 mg/ferret q24hrs. As sucralfate requires an acidic environment to work it should always be given at least 30 minutes before famotidine.
- Oxygen can be given to treat cases of brain hypoxia. Diazepam 1–2 mg/kg may be administered to controlled seizures. Fluid therapy and thermal support may be required. Nutritional support is vital to avoid hepatic lipidosis.

Paracetamol (acetaminophen) toxicosis

Paracetamol toxicosis is seen anecdotally resulting in hepatic necrosis and death. Ferrets are thought to have a similarly slow glucuronidation of paracetamol compared to cats.

Benzocaine induced methemoglobinemia

Benzocaine induced methemoglobinemia occurred in ferrets that had their larynx sprayed with benzocaine to facilitate endotracheal intubation. If the methemoglobinemia is of a high enough concentration, there will be decreased oxygen carrying capacity of the erythrocytes. This did not occur when lidocaine was used.

Zinc toxicity

Ferrets appear to be especially sensitive to zinc toxicity and fatalities can occur with ingestion of zinc due to toxic nephropathy.

Sources of zinc

Owing to this risk, ferrets should not be housed in galvanised cages and should not be exposed to galvanised metals. Other common sources of zinc found in domestic houses include batteries, wood preservatives, paints, zinc-oxide creams, zippers on clothing and luggage, board game pieces, herbal supplements, the coating on some galvanised cookware, and screws or nuts.

Ingestion of zinc, including the metal forms, causes formation of highly toxic and corrosive soluble zinc salts in the acidic environment of the stomach which are also absorbed via the duodenum (and rapidly distributed to tissues throughout the body) as well as causing local damage. Zinc also interferes with iron and copper metabolism therefore affecting haematopoiesis.

Clinical signs and diagnosis

Clinical signs vary depending on exposure duration and amount but may include vomiting, anorexia, polyuria, polydipsia, anaemia, melena, icterus, and diarrhoea.

Diagnosis is based on history (especially if housed in galvanised cages), survey radiographs may show metal radiopacities, and blood can be taken to check zinc levels.

Treatment

Treatment options include removal of zinc containing materials in the stomach by inducing emesis, gastric lavage, and endoscopic or surgical extraction. Fluid diuresis is recommended. Administration of H2 blockers is advisable in cases of gastric zinc foreign bodies as *reduction of the gastric acid will reduce the zinc salts produced*.

Helicobacter is likely to be an opportunistic complicating pathogen and presumptive treatment should be instigated. Iron administration and nutritional support is advised. The efficacy of heavy metal chelation is reportedly ambiguous but calcium ethylenediaminetetraacetic acid (EDTA) 30 mg/kg SC q12hrs should be considered. *Activated charcoal does not bind zinc in appreciable amounts to justify its use, especially as activated charcoal can cause problems in ferrets* (see above).

Common conditions of the intestine

Inflammatory bowel disease

Inflammatory bowel disease is *very common in older ferrets and is underdiagnosed as it can mimic other conditions*. The exact cause and mechanism are unknown, but diet is implicated, and some cases have improved when put on a novel protein source or prescription hypoallergenic cat food. I suggest Virbac Veterinary VHM Feline Digestive support as it is high in animal sourced protein and low in carbohydrates.

CLINICAL SIGNS

Diarrhoea will often be described as having a 'bird seed' appearance due to the presence of undigested food (Figure 10.38), although the appearance of the diarrhoea is not pathognomonic. Chronic weight loss is a common clinical sign and vomiting may also occur. Owners may not notice slow weight loss or a gradual change in the softness of the faeces

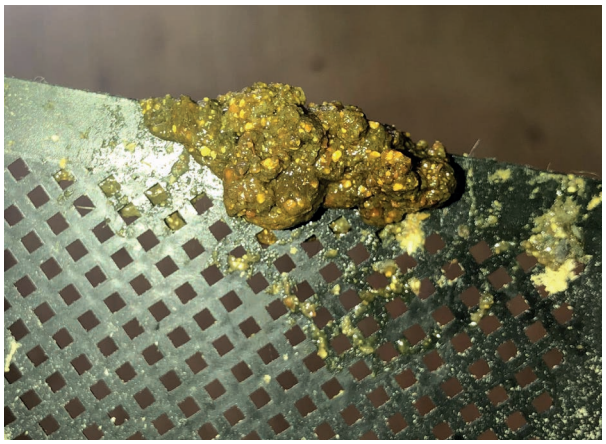


Figure 10.38 Bird seed appearance diarrhoea.

until the condition is advanced, further complicated by the fact the clinical signs can be intermittent.

DIAGNOSIS

Differential diagnoses include dietary indiscretion, *H. mustelae*, intestinal lymphoma, ferret enteric coronavirus, and eosinophilic gastroenteritis (high levels of eosinophils on haematology are highly suggestive of the condition and may be as high as 35%).

Definitive diagnosis is achieved by multiple gastric and intestinal biopsies. *It can be a challenge to differentiate lymphoma and inflammatory bowel disease due to the lymphoplasmacytic inflammation of inflammatory bowel disease* (Hoefer, 2020). Multiple biopsies assist with the correct histopathological diagnosis but may increase the risk of complications such as stricture formation.

TREATMENT

Treatment is based on reducing the inflammation. Prednisolone can be started 1 mg/kg orally q12–24hrs and/or azathioprine 0.9 mg/kg orally every 1 to 3 days. A course of metronidazole 50 mg/kg q24hrs could be considered (Lennox, 2005). Metronidazole has the added benefit of immunomodulatory effects on the bowel. Owing to the chronicity of the condition, oral supplementation of vitamin B_{12} is recommended. I use Cobalaplex, Protexin (half a capsule daily). Owners should be counselled that chronic inflammation can result in lymphoma (Brown & Ramsell, 2012).

Intestinal neoplasia

Lymphoma is the third most common tumour seen in ferrets. Adenocarcinomas of the gastrointestinal tract are also seen. *Intestinal lymphoma will often blend macroscopically imperceptibly with surrounding tissue, whereas adenocarcinomas tend to form discrete masses* (Johnson-Delany, 2010). The masses formed by adenocarcinomas can lead to intestinal obstruction and present as a foreign body.

Clinical signs and diagnosis

Lymphoma can have a wide range of clinical presentations. Minimal clinical databases should include biochemistry, haematology, urinalysis, radiography, and ultrasound. Bone marrow aspirate may be required in some cases.

Diagnosis requires histopathology of affected tissue and immunophenotyping (B- or T-cell). It is common to get false positives in intestinal biopsies due to the similarity seen in cases of inflammatory bowel disease. *Surgical biopsies are preferred to FNAs as it has been shown that there is poor correlation between classifications of lymphoma via fine needle aspirate and surgical biopsy.*

Treatment is likely to involve both surgical and medical management. There are many examples of chemotherapy protocols in the literature and there is no conclusive evidence that any one treatment is better than another. Owners of ferrets that have been on steroid therapy for concurrent illness e.g. Insulinoma, should be counselled that the lymphoma may not be as sensitive to steroid therapy.

Ferrets with stage one will likely have a better prognosis. Variable survival times are in the literature. See section on lymphoma/sarcoma.

Infectious causes of diarrhoea associated with raw feeding

A history of raw feeding should raise the index of suspicion of an infectious cause of the diarrhoea with possible pathogens including *Salmonella*, *Campylobacter*, *E. coli*, and even mycobacterial infections.

Salmonella will result in fever, lethargy, conjunctivitis, and bloody diarrhoea. Diagnosis is via bacteriology but it should be remembered that *Salmonella* can be intermittently shed so a negative result does not necessarily mean that *Salmonella* is not involved (Powers, 2009). Pooling faecal samples over a period of 3 days prior to submission will increase the likelihood of detection. Treatment should be based on culture and sensitivity. Owners should be counselled that the *infection could be zoonotic* and even with treatment could result in a carrier status animal. Owners who are immunosuppressed may consider euthanasia rather than treatment.

Campylobacter, also zoonotic, can cause diarrhoea with mucous and blood and the treatment of choice is erythromycin 10 mg/kg q6hrs. *E. coli* has been cultured from ferrets with diarrhoea but is of unknown clinical significance (Lennox, 2005).

Mycobacterial infection is frequently seen in ferrets and often associated with raw feeding, unpasteurised dairy or feeding carrion. Clinical signs include conjunctivitis, vomiting, diarrhoea, anorexia, weight loss, splenomegaly, lymph node enlargement, and sometimes pneumonia. *Mycobacterial infection should be included in the differential*

Table 10.14 Differential diagnosis for ocular and GI involvement.

Differential List for ferrets with both ocular and gastro-intestinal signs
Mycobacteriosis
Salmonella
Ferret Systemic Corona Virus
Distemper

diagnosis list for any ferrets with ocular, respiratory and gastrointestinal diseases (Table 10.14). Grossly mycobacterial granulomas can look like those seen with ferret systemic coronavirus. Diagnosis is via biopsy of bowel, spleen or lymph nodes sent for histopathology or PCR. Treatment can be unrewarding due to the difficulty in eliminating the infection despite long courses of treatment. The zoonotic potential, especially in immune suppressed owners, must also be discussed.

Uncommon/rare causes of diarrhoea in clinical practice

Rotavirus can cause diarrhoea in young kits 1–6 weeks of age. Older animals exhibit mild transient diarrhoea. Rotavirus is most likely to be seen in a large breeding facility.

Owing to the young age of most affected animals, rapid dehydration is a significant concern and immediate supportive care is required for survival. IV fluids and assisted feeding should be initiated. Langlois (2005) recommends antibiotics to decrease mortality rates as secondary bacterial infections play an important role in this condition. Antibiotics should be chosen based on culture and sensitivity of the diarrhoea.

Proliferative bowel disease causes diarrhoea in ferrets between 4 and 6 months of age due to infection with *Lawsonia intracellularis*. Straining and vocalisation is a clinical feature and rectal prolapse can occur. A rectal swab can be sent for PCR to diagnose this condition and the recommended treatment is chloramphenicol 50 mg/kg twice daily for 10 days.

Endoparasites are not common in pet ferrets but a parasite screen is advised in cases of diarrhoea, especially in shelter outbreaks. *Coccidia* and *Giardia* have both been documented in ferrets.

Coccidia

Three species of coccidia are reported to occur in ferrets: *Eimeria furonis*, *Eimeria ictidea*, and *Isospora laidlawi*.

CLINICAL SIGNS

Young, healthy ferrets may show no clinical signs and stress can be a predisposing factor for clinical disease. Clinical signs include diarrhoea (which can be watery, mucoid, or haemorrhagic), lethargy, dehydration, rectal prolapse, mesenteric lymphadenopathy, and palpably thickened intestines.

DIAGNOSIS AND TREATMENT

Diagnosis is achieved by performing a faecal flotation or a faecal wet mount. Treatment options are sulfadimethoxine 50 mg/kg PO on day 1, then 25 mg/kg PO q24hrs for 9 days, trimethoprim sulfadiazine 30 mg/kg q12hrs, amprolium 19 mg/kg PO for at least 2 weeks, and decoquinate 0.5 mg/kg PO q24hrs for 2 weeks.

Maintaining good hygiene and rapid removal of faeces will help prevent ingestion of oocysts during outbreaks.

Giardia

Clinical reports are rare but there has been suggestion that infection rates are increasing in pet ferrets. Owners should be warned of the potential of zoonotic infection. Clinicals signs are soft, voluminous, or grainy stools (due to malabsorption caused by the parasite), weight loss, and a poor-quality coat.

Diagnosis is achieved by examining wet mounted fresh faeces and identifying trophozoites and cysts. Adding iodine stain can help with identification.

The treatment of choice is metronidazole 15–20 mg/kg q12hrs recommended for a minimum of 5 days up to 2 weeks.

Viral causes of diarrhoea

Any viral disease that does not primarily affect the gastrointestinal tract may still result in diarrhoea secondary to stress.

Ferret enteric coronavirus

Classically, the history with ferret enteric coronavirus infection (FRECV) is that a new apparently healthy ferret has recently been introduced into the group. Incubation period is 48–72 h with clinical signs in one or more of the established ferrets initially being lethargy, inappetence and vomiting. They then develop malodorous green mucoid diarrhoea which can develop into a 'bird seed' diarrhoea.

Table 10.15 Viral causes of diarrhoea.

Viral causes of diarrhoea (in order of most commonly occurring)
Ferret enteric corona virus aka epizootic catarrhal enteritis or green slime disease
Ferret systemic corona virus
Aleutian disease
Influenza
Canine distemper
COVID-19



Figure 10.39 Green mucoid diarrhoea, which can be seen in cases of ferret enteric coronavirus infection.

Resulting dehydration can be rapid and severe (Figures 10.38 & 10.39).

The morbidity is high (up to 100%) but with supportive care, mortality is low (<5%) (Murray *et al.*, 2010). FRECV can be seen in ferrets of any age, but older ferrets that recover can develop chronic diarrhoea and weight loss due to blunting and atrophy of the intestinal villi.

DIAGNOSIS AND TREATMENT

Diagnosis is often presumptive based on clinical signs and history of a new ferret being introduced recently. Definitive diagnosis is achieved by PCR of faeces or intestinal biopsy. Treatment is symptomatic and supportive (see above). If chronic malabsorption develops, prednisolone may need to be given orally along with an elemental diet such as Lafeber's EmerAid Nutritional care systems Carnivore diet, Oxbow Animal Health Carnivore Care to supplement the diet.

Ferret systemic coronavirus

Ferret systemic coronavirus (FRSCV) is usually seen in ferrets under 18 months of age. The condition is very similar to the dry form of feline infectious peritonitis. There are no pathognomonic clinical signs and diagnostic testing is required for diagnosis.

CLINICAL SIGNS AND DIAGNOSIS

Clinical signs commonly seen are diarrhoea, weight loss, lethargy, anorexia, vomiting, organomegaly and abdominal masses. Occasionally neurological signs such as: ataxia, tremors, seizures, hind limb paresis are seen. Fever may be present and less common signs are sneezing and coughing.

Biochemistry will show a polyclonal hypergammaglobulinemia, hypoalbuminemia and any other parameters changed will reflect any damage to other organs affected. Haematology will show a non-regenerative anaemia and thrombocytopaenia. *Green urine may also be a feature due to increased levels of biliverdin*. The current understanding is that the biliverdin is from micro haemorrhage into tissues and extravascular coagulation.

Typical clinical signs, blood work results, and a polyclonal gammopathy on serum protein electrophoresis are suggestive of FRSCV-associated disease but definitive diagnosis is by histopathology and immunohistochemical staining of granulomatous tissue.

TREATMENT

There is no cure for FRSCV although palliative treatment may provide several months of good quality life for the ferret. Treatment is aimed at suppressing the inflammatory response. Prednisolone is started 1–2 mg/kg q12hrs and then tapered to the lowest controlling dose.

Vasculitis is a common feature, so treatment with pentoxifylline 20–25 mg/kg q12hrs can help reduce fibrosis. Broad-spectrum antibiosis is recommended during immunosuppressive therapy to prevent secondary bacterial infection and doxycycline is a good choice 10 mg/kg q12hrs. *Doxycycline has the added benefits of having anti-inflammatory properties, decreasing fibrosis, inhibiting tumour necrosis factor alpha production and also inhibiting leukocyte adhesion to endothelial cells* (Murray *et al.*, 2010).

Concurrent stress-induced gastric ulceration is likely so treatment with sucralfate 75–125 mg/kg q8–12hrs should be administered at least 30 minutes before feeding or administering prednisolone. Although omeprazole and famotidine have the advantage of being once daily administration, *cimetidine is the H2 receptor antagonist of choice despite requiring three times daily administration 10 mg/kg. This is due to cimetidine having the added benefits of antiviral activity, helping to stimulate cell mediated immunity and increasing T-lymphocyte levels* (Murray *et al.*, 2010). Other treatment is symptomatic as previously discussed.

Aleutian disease

Aleutian disease is a parvovirus that affects various mustelids. *It is not the virus itself that results in the disease but the host's immune response and as such this is considered an immune-mediated disease*. It is for this reason that a vaccine cannot be produced. Most animals affected are between two and four years of age. It should be noted that ferrets

can be asymptomatic carriers of the virus and also shed the virus in their saliva, urine, and faeces.

CLINICAL SIGNS

Clinical signs include weight loss, lethargy, inappetence, melena, pallor, organomegaly, ascending paresis/paralysis, seizures, muscle fasciculations, faecal and urine staining due to incontinence.

DIAGNOSIS

Biochemistry may show azotaemia, hypoalbuminemia, hyperglobulinemia, and liver enzymes may be increased. Serum protein electrophoresis will show hypergammaglobulinemia which if present in affected ferrets should constitute 20% or more of the total serum concentration. *Hypergammaglobulinemia is a consistent finding in affected mink but it is a variable finding in affected ferrets*. Antibody testing will confirm exposure but does not definitively diagnose. Definitive diagnosis in the live animal is challenging but a presumptive diagnosis can be made with expected clinical signs, biochemistry, and confirmed exposure (Table 10.16).

TREATMENT

There is *no specific treatment. Prognosis is grave for severely affected individuals. Mildly affected* individuals may recover. As it is the immune response that causes the disease, immuno-suppressive therapy using prednisolone 2 mg/kg q24hrs may be beneficial.

Influenza

Ferrets are susceptible to human influenza; it should be remembered that this and other zoonotic conditions can go both ways. *Any veterinary staff who are displaying flu-like symptoms, even mild ones, should either avoid working with ferrets or wear gloves and a mask*.

CLINICAL SIGNS

Clinical signs include transient diarrhoea, fever, anorexic, lethargy, and upper respiratory signs such as coughing and sneezing. In the early stages this could resemble distemper infection.

Table 10.16 Differential list for hypergammaglobulinemia in ferrets.

Chronic infection (e.g. *Helicobacter mustelae*)
Chronic inflammation (e.g. inflammatory bowel disease)
Lymphoma/lymphosarcoma
Ferret systemic corona virus
Aleutian disease (will be ≥ 20% total protein)
Multiple myeloma

TREATMENT

Treatment is symptomatic and antihistamines, for example, chlorpheniramine 1–2 mg/kg q8–12hrs may help. Meloxicam 0.2 mg/kg q24hrs can be given. Although pneumonia appears to be uncommon, the ferret should be monitored for any signs of secondary bacterial involvement such as green/yellow sputum and any worsening of respiratory signs.

Common conditions of the rectum/anus

Prolapse

Removal of the anal glands is prohibited in the UK unless it is due to disease. In other countries (e.g. the USA), the anal glands are routinely removed at a very young age. Rectal prolapse can occur due to poor surgical technique. *If the anal glands have been removed, this increases the risk of anal prolapse in any animal with severe or chronic diarrhoea*.

Prolapse can also occur due to severe straining. The age of the ferret may help with your diagnosis. Young ferrets are at higher risk of infectious conditions such as coccidiosis, proliferative bowel disease, and colitis. Diagnostic tests would include faecal exam (smears, flotation and staining for bacteria, and red blood cells); biochemistry and haematology as a minimum.

Older ferrets are more likely to present with rectal prolapse caused by more chronic conditions. Prostatic disease in male ferrets, urinary outflow obstruction, or enlarged sub lumbar lymph nodes can all result in severe straining. Minimum clinical database would also include radiographs and ultrasound in older ferrets.

TREATMENT

Treatment should be aimed at addressing the underlying condition as well as the prolapse. Mild cases can be treated with topical application of a haemorrhoid cream such as Preparation-H (GlaxoSmithKline) three to four times daily. If there is more than 0.2 cm tissue protruding or the tissue is necrotic then corrective surgery is indicated.

Anal gland infections

Anal gland infections occur uncommonly. Clinical signs are similar to dogs with infected anal glands. Ferrets may require sedation to empty painful glands and just like with dogs, they may require flushing of the glands. Antibiotic choice should be based on culture and sensitivity. Analgesia should also be provided.

Euthanasia

End of life conversations can be a difficult area to broach with some clients. Occasionally owners will wait for us to broach the topic as they are 'worried' if they do so we will think they are not dedicated enough. Reassuring clients

that sometimes we need to love our pets enough to let them go when the time is right can be invaluable.

Many owners will want to stay with their pet ferret. Pre-medication can be given SC or IM using a cocktail of medetomidine, butorphanol, and acepromazine. Phenobarbitone is then given IV using either the cephalic or lateral saphenous vein. I will offer clients a paw print and a lock of fur. If your clinic offers private cremations for dogs and cats, they should also be offered to your ferret clients. Personally, I will send a sympathy card if I know the client well.

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Appendix: History forms

RABBIT HISTORY FORM

Date: …….. Pet's Name: ……. Owner's Name: ……

Owner's Address: ……

Breed ……..……..…… Age ……..…… Sex ……

Insured? No ☐ Yes ☐ If yes, who with? ……

Background Information:

Length of time owned: ……

Where acquired? Breeder ☐ Pet Store ☐

Other ……

Wormed?/E cunic Treatment? No ☐ Yes ☐

If yes, with what/ when/length of course? ……

……

Ectoparasite treatment? No ☐ Yes ☐

If yes, with what/when/length of course? ……

Vaccinated? No ☐ Yes ☐ Date of last vaccination ……

How often is animal handled? Daily ☐ Occasionally ☐ Never ☐

Details ……

How often is the rabbit let out of the cage? Daily ☐ Occasionally ☐ Never ☐

Details ……

If daily: Percentage inside cage …… %

Percentage outside cage …… %

Animal ever taken outside of home (e.g. pet shows etc.)? No ☐ Yes ☐

Details .....

Any other pets? No ☐ Yes ☐

Details .....

Do they interact with rabbits? No ☐ Yes ☐

Details .....

Any other rabbits? No ☐ Yes ☐ If yes, specify .....

Are rabbits housed together or singly?

Details .....

Any new additions to the rabbit household? No ☐ Yes ☐ If yes, specify .....

Husbandry:

Housed indoors/outdoors? .....

Where is cage located? ..... Access to wildlife? No ☐ Yes ☐

If yes, specify .....

Type of cage .....

Cage materials .....

Cage substrate ..... Size of enclosure .....

Does the rabbit free roam in the house? No ☐ Yes ☐ Details .....

Does the rabbit have an outside run? No ☐ Yes ☐ Details .....

Cage furniture .....

Frequency and routine of cage cleaning? Details .....

Is a litter tray used? No ☐ Yes ☐ Type of disinfectant used? .....

Sketch of Cage/Environment:

Nutrition:

Processed rabbit food offered? No ☐ Yes ☐ If yes: Course Mix ☐ Pellet food ☐

Brand …… Amount fed/frequency ……

Hay given? No ☐ Yes ☐ If yes what type ……

Amount fed/frequency …… Where is hay placed?

Fruit and vegetable? No ☐ Yes ☐ If yes what type ……

Amount fed/frequency ……

Water source? …… How often is it replaced? ……

Any additional vitamin or mineral supplements? No ☐ Yes ☐ If yes what type ……

Character of faeces/caecotrophs? Details ……

……

Past Medical History/Problems/Treatments:

……

……

……

……

Current Presenting Problem:

……

……

……

……

Duration of Complaint:

……

……

……

……

GUINEA PIG HISTORY FORM

Date: …….. Pet's Name: …… Owner's Name: ……

Owner's Address: ……

Colour morph/breed ……..……..…… Age ……..…… Sex ……..

Background Information:

Length of time owned: ……

Where acquired? Breeder ☐ Pet Store ☐ Other ……

Ectoparasite treatment? No ☐ Yes ☐

If yes, with what/ when/length of course? ……

How often is animal handled? Daily ☐ Occasionally ☐ Never ☐

Details ……

Animal ever taken outside of home (e.g. pet shows etc.)? No ☐ Yes ☐

Details ……

Any other pets? No ☐ Yes ☐

Details ……..…..

Do they interact with Guinea pig? No ☐ Yes ☐

Details ……

Any other Guinea pigs? No ☐ Yes ☐

If yes, specify ……

Are Guinea pigs housed together or singly?

Details ……

Any new additions to the Guinea pig household? No ☐ Yes ☐

If yes, specify ……

Husbandry:

Housed indoors/outdoors? .....

Where is cage located? .....

Access to wildlife? No ☐ Yes ☐

If yes, specify .....

Type of cage .....

Cage materials .....

Cage substrate .....

Size of enclosure .....

Does the Guinea-pig free roam in the house? No ☐ Yes ☐

Details .....

Does the Guinea-pig have an outdoor run? No ☐ Yes ☐

Details .....

Cage furniture .....

Frequency and routine of cage cleaning?

Details .....

Is a litter tray used? No ☐ Yes ☐

Substrate in litter tray? .....

Type of disinfectant used? .....

Sketch of Cage/Environment:

Nutrition:

Processed Guinea-pig food offered? No ☐ Yes ☐ If yes: Course Mix ☐ Pellet food ☐

Brand .....

Amount fed/frequency .....

Hay given? No ☐ Yes ☐ If yes what type .....

Amount fed/frequency .....

Where is hay placed? .....

Fruit and vegetable? No ☐ Yes ☐ If yes what type .....

Amount fed/frequency .....

Water source? .....

How often is it replaced? .....

Any additional vitamin or mineral supplements? No ☐ Yes ☐ If yes what type .....

Character of faeces? Details .....

.....

Past Medical History/Problems/Treatments:

.....

.....

.....

Current Presenting Problem:

.....

.....

.....

Duration of Complaint:

.....

.....

.....

SMALL FURRY HISTORY FORM

Date: …….. Pet's Name: ……. Owner's Name: ……

Owner's Address: ……

Species/Breed ……..……..…… Age ……..…… Sex …….

Insured? No ☐ Yes ☐ If yes, who with? .....

Background Information:

Length of time owned: …… Where acquired? Breeder ☐ Pet Store ☐

Other ……

Wormed/ *Hymenolepis* treatment? No ☐ Yes ☐

If yes, with what/ when/length of course? ……

Ectoparasite treatment? If yes, with what/ when/length of course? ……

How often is animal handled? Daily ☐ Occasionally ☐ Never ☐

Details ……

Animal ever taken outside of home (e.g. pet shows etc.)? No ☐ Yes ☐

Details ……

Any other pets? No ☐ Yes ☐

Details ……

Do they interact with small furry No ☐ Yes ☐

Details ……

Any other small furries? No ☐ Yes ☐

If yes, specify ……

Are small furries housed together or singly?

Details ……

Any new additions to the small furry household? No ☐ Yes ☐

If yes, specify ……

Husbandry:

Housed indoors/outdoors? .....

Where is cage located? .....

Access to wildlife? No ☐ Yes ☐

If yes, specify .....

Type of cage .....

Cage materials .....

Cage substrate .....

Size of enclosure .....

Does the small furry ever free roam in the house? No ☐ Yes ☐

Details .....

Does the small furry have access to outside? No ☐ Yes ☐

Details .....

Cage furniture .....

Frequency and routine of cage cleaning?

Details .....

Is a litter tray used? .....

Substrate in litter tray .....

Type of disinfectant used .....

Sketch of Cage/Environment:

Nutrition:

Species specific processed food offered? No ☐ Yes ☐ If yes: Course Mix ☐ Pellet food ☐

Brand .....

Amount fed/frequency .....

Hay given? No ☐ Yes ☐ If yes what type .....

Amount fed/frequency .....

Where is hay placed? .....

Fruit and vegetable? No ☐ Yes ☐

If yes what type .....

Amount fed/frequency .....

Insects/live food? No ☐ Yes ☐

If yes what type .....

Amount fed/frequency .....

Water source .....

How often is it replaced? .....

Any additional vitamin or mineral supplements? No ☐ Yes ☐

If yes what type .....

Character of faeces? Details .....

.....

.....

Past Medical History/Problems/Treatments:

.....

.....

.....

.....

Current Presenting Problem:

Presenting problem symptom .....

Seen by another vet? No ☐ Yes ☐

Details .....

Has any medication been given (prescribed or otherwise)? No ☐ Yes ☐

Details .....

Has there been any response to treatment? No ☐ Yes ☐

Details .....

Problem Details .....

.....

.....

Duration of Complaint:

How long been unwell? .....

Sudden or gradual onset? .....

Details .....

FERRET HISTORY FORM

Date: …….. Pet's Name: ……. Owner's Name: ……

Owner's Address: ……

Breed ……..……..…… Age ……..…… Sex …….

Reproductive stasis? Neutered ☐ Vasectomized ☐ Entire ☐

If entire female how is this managed? Suprelorin implant ☐ hCG or GnRH injections ☐

Vasectomized hob ☐ Breeding female ☐

Details ……

Background Information:

Length of time owned: ……

Where acquired? Breeder ☐ Pet Store ☐

Other ……

Wormed (heartworm)? If yes, with what/ when/length of course? ……

Ectoparasite treatment? If yes, with what/ when/length of course? ……

Vaccinated? No ☐ Yes ☐ Distemper ☐ Rabies ☐

Date of last vaccination ……

How often is animal handled? Daily ☐ Occasionally ☐ Never ☐

Details ……

Animal ever taken outside of home (e.g. pet shows etc.)? No ☐ Yes ☐

Details ……

Is the animal worked? No ☐ Yes ☐

Details ……

Any other pets? No ☐ Yes ☐

Details ……..……..

Do they interact with ferrets? No ☐ Yes ☐

Details ……

Any other ferrets? No ☐ Yes ☐ If yes, specify ……

Are ferrets housed together or singly?

Details .....

Any new additions to the ferret household? No ☐ Yes ☐

If yes, specify .....

Husbandry:

Housed indoors/outdoors? .....

Where is cage located? .....

Access to wildlife? No ☐ Yes ☐

If yes, specify .....

Type of cage .....

Cage materials .....

Cage substrate .....

Size of enclosure .....

Does the ferret free roam in the house? No ☐ Yes ☐

Details .....

Does the ferret have an outside run? No ☐ Yes ☐

Details .....

Cage furniture .....

Toys .....

Frequency and routine of cage cleaning?

Details .....

Is a litter tray used? .....

Substrate in litter tray .....

Type of disinfectant used .....

Sketch of Cage/Environment:

Nutrition:

Cat food? No ☐ Yes ☐ If yes what type .....

Amount fed/frequency .....

Ferret food? No ☐ Yes ☐ If yes what type .....

Amount fed/frequency .....

Raw fed? No ☐ Yes ☐ If yes what type .....

Amount fed/frequency .....

Treats? No ☐ Yes ☐

Details .....

Water source .....

How often is it replaced? .....

Any additional vitamin or mineral supplements? No ☐ Yes ☐

If yes what type .....

Character of faeces?

Details .....

Past Medical History/Problems/Treatments:

.....

.....

.....

Current Presenting Problem:

.....

.....

.....

Duration of Complaint:

.....

.....

.....

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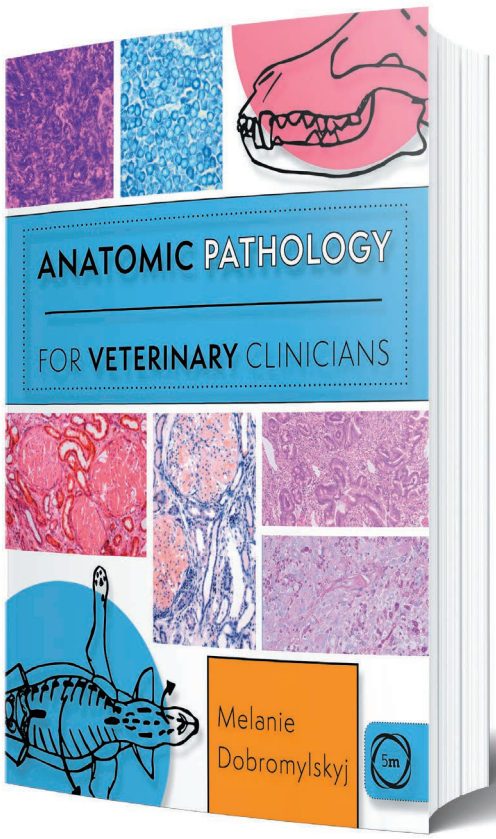
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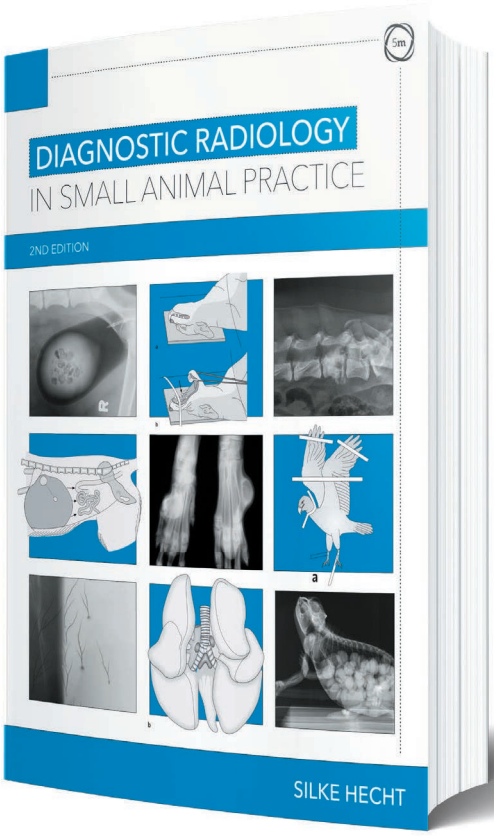
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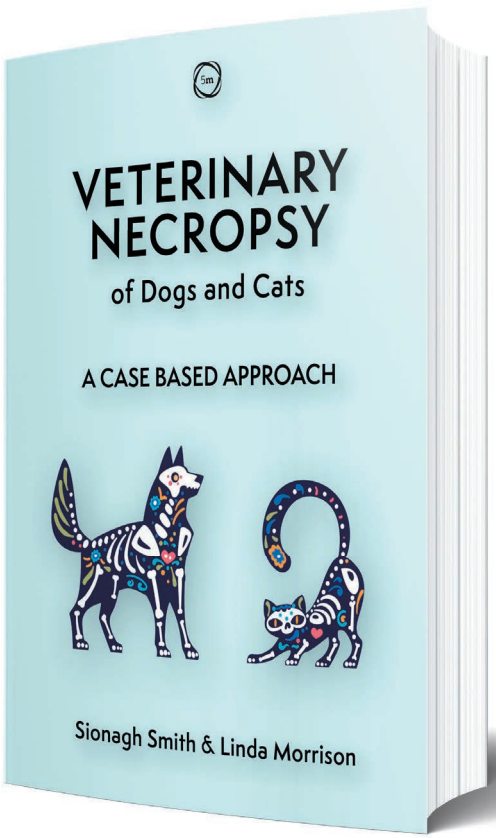
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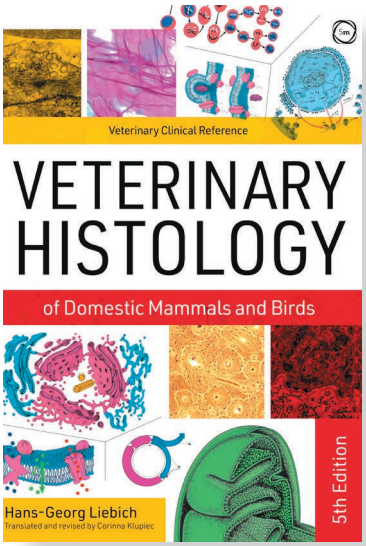
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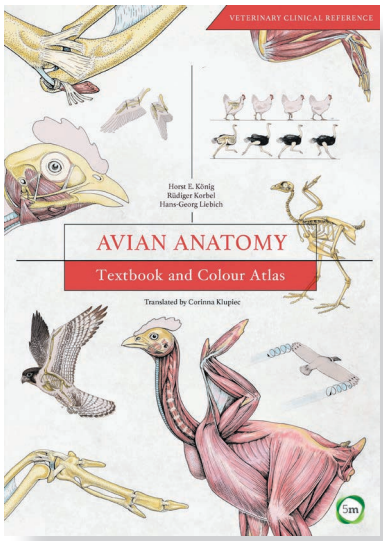
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